



I.M.A.G.S.B. NEWS BULLETIN

Estd. On 2-3-1945

GUJARAT MEDICAL JOURNAL

INDIAN MEDICAL ASSOCIATION, GUJARAT STATE BRANCH

Office : A.M.A. House, 2nd Floor, Opp. H. K. College, Ashram Road, Ahmedabad-380 009.

Fax / Phone : (079) 2658 73 70 E-mail : imagsb@gmail.com, gujaratmedicaljournal@gmail.com

G
M
J

EDITORIAL BOARD

President

Dr. Chetan N. Patel

Hon. State Secretary

Dr. Jitendra N. Patel

Hon. Treasurer

Dr. Devendra R. Patel

Hon. Editor

Dr. K. R. Sanghavi

Hon. Jt. Editor

Dr. Harshad C. Patel

Hon. Secretary

Dr. B. I. Patel

Associate Editors

Dr. Mahadev Desai

Dr. Atul H. Kansara

Dr. Amit P. Shah

Dr. Mukund M. Prabhakar

Dr. Tushar Patel

Dr. Haresh Doshi

Dr. Shivangi A. Patel

Dr. Jitesh A. Desai

Dr. Pravinaben M. Santwani

Dr. Pankaj Modi

Dr. Niyat Pandya

Dr. Manish G. Shah

Members

Dr. Bhavesh Patel

Dr. Kailashben Parikh

Dr. Bhavin Patel

Dr. Shailesh Raval

Dr. K. G. Kanani

Dr. L. R. Gohil

Ahmedabad

Vadodara

Surat

Nadiad

Rajkot

Bharuch

Ahmedabad Zone

Vadodara Zone

Surat Zone

Central Zone

West Zone

South Zone



**STATE PRESIDENT
AND
HON. STATE SECRETARY'S
MESSAGE**



Dear Friends,

Today our profession is undergoing tremendous upheaval and we are wading through deep waters due to the continuous interference of the Government. There are several challenges in front of us.

Majority of our members are not aware of the Draconian Clinical Establishment Act. This act was actively opposed by IMA at all levels.

The security of medical professionals providing healthcare to the public has become a major issue. If the doctors are always working under stress of being attacked by relatives if some untoward event happens with their patient how will be they able to perform their duty of providing a good healthcare. One should understand that the disease process takes its own course. At times media and public unnecessarily blame the doctors. Our state has Prevention of Violence and damage to property bill for Hospital and medical professionals' protection. FIR should be registered immediately against such persons involved in violence against doctors and hospitals.

We appeal to the members to constantly improve their communication skills and avoid situations leading to violence.

Another issue of utmost importance is concerning the Health Insurance. There is no clear policy and directives. This requires an urgent attention and should be amicably settled so that all the stake holders are satisfied.

In the PPS seminar, organized by IMA Ahmedabad branch, the issue of IPC 304 & 304 A was well discussed. All the members should know the difference between two. IMA Gujarat State has many times represent to the Government of Gujarat regarding our concern for this.

Medicos are often falsely blamed by the Government agencies and media for not going to villages. The lack of infrastructure in rural area, sometimes frustrates the medicos working in the rural areas when they are unable to do justice while treating patients.

Friends, there are lots many issues and we have to unite. This is the need of hour. To overcome all the challenges we have to work in unison, and it is possible only with your cooperation and support. Only then

we can make IMA a strong association. My job is to encourage you rather than instruct you because We know **" Instruction does much, but encouragement does everything."**

For the first time on 01st July every State Branch has organized a press conference at 12 noon. IMA Gujarat State Branch has also organized at Vadodara. Our problems and our social activities were highlighted to the media and they gave good coverage. Many Branches have observed the Doctor's Day. If they have not reported to the State Office, I request the local branch President / Hon. Secretary to do so.

IMA GSB office is in a process of forming a young Doctor's Wing. Very soon a draft will be put before you for your suggestions.

Indian Medical Association is the umbrella for all the specialities and is ready to take the lead for this. From our side, we have tried to collect the information of State President & Hon. Secretaries of the various speciality Associations. We request all the speciality Association office bearers to give their details to State office. We want to have a meeting for better implementation of various projects and utmost importance is to strengthen our unity. Prevention of Diabetic Blindness Programme is one such project where IMA and Indian Association of Ophthalmologists joined for a good cause.

Our another wish is to prepare roster of speaker / talents. These speakers may be called upon to speak by various IMA branches for updating themselves. Large branches have wealth of speakers / talents. Dear Presidents, Secretaries can we have their names, please. You know them. The other branches can take advantage of such talented persons.

We request all of you to register for next GIMACON 2015 to be held at Vadodara on 28th & 29th November. The registration form is available at IMA GSB website. We once again request to organize the programmes on the initiatives which we have planned at the beginning of the year.

Friends, we welcome valuable inputs from all IMA members and we assure you that all office bearers shall strive to make our organization on very strong foundations.

Let us pledge **together we will achieve.**

DR. CHETAN N. PATEL
President, IMA GSB.

DR. JITENDRA N. PATEL
Hon Secretary, IMA GSB.

Character is more important than intelligence for success.

FROM THE DESK OF EDITORS



Dear friends,

While putting the second issue of Gujarat Medical Journal in your hands in this year, we feel proud that for decades GMJ is published non-stop, with few exceptions, where situations were not in our hands or because of some office problems. For this credit and thanks goes to our all the president-secretaries and dedicated editors of yester years.

We are very well aware that conventional epidemic diseases like malaria, typhoid, bacterial diarrhea respiratory tract infections in children are decreasing. Of course newer virus diseases like swine flue, dengue are coming in endemic form. But now our profession is more worried about life threatening life style diseases like obesity, diabetes mellitus, hypertension and more. Our associations are preparing our doctors to combat these by creating awareness through CMEs and lectures.

In last five years of time many new medical collages have started in our state and that will create many new doctors to serve the country and the society. At the same time a demand for new academic minded medical teachers is also increasing. Our hospitals and expertise are world class and that pushes the medical tourism in Gujarat far ahead. Our hospitals and institutes are well equipped with world class equipments and infrastructures.

People from developed and underdeveloped countries come here for treatment and we provide them world best treatment at a cheaper rates then that is available in developed countries. Also we get large number of patients from our own domestic population and this provides ample of opportunities for our colleagues working in hospitals, medical collages and research institutes for research. GMJ provides them a platform.

You all know our GMJ is an INDEXED JOURNAL. For last few years, indeed, we get more research articles for publication. Without making any compromise in our laid down standards and policy, it has always remained our effort to make GMJ more informative, more interesting and more popular so that large number of our colleagues read it and utilize the knowledge and information provided in it. For this, we welcome your suggestions and comments also.

Our sincere thanks to GSB president Dr. Chetan Patel and hon. secretary Dr. Jitendra N. Patel for encouragement and suggestions. We are grateful to Dr. Kirtibhai Patel and Dr. Mahendrabhai Desai for their guidance and help. Our particular thanks to GMJ ex. editor Dr. Amitbhai Shah for all sorts of help and guidance that he has provided us time to time.

Promising you the best reading,

DR. K. R. SANGHAVI
Editor-IMA-GSB-GMJ

DR. B. I. PATEL
Hon. Secretary -IMA-GSB-GMJ

DR. HARSHAD C. PATEL
Jt. Editor-IMA-GSB-GMJ



**OFFICE BEARERS OF
GUJARAT STATE BRANCH, INDIAN MEDICAL ASSOCIATION**



2014-2015

PRESIDENT	DR. CHETAN N. PATEL MOB. 94263 78078	VADODARA
IMM. PAST PRESIDENT	DR. BIPIN M. PATEL MOB. 98250 62381	AHMEDABAD
VICE PRESIDENTS	DR. VINAY A. PATEL	AHMEDABAD
	DR. MANHAR A. SANTWANI	WEST ZONE
	DR. SUNIL L. ACHARYA	CENTRAL ZONE
	DR. PARESH P. GOLWALA	VADODARA
	DR. PARESH MUNSHI	SURAT
	DR. S. S. VAISHYA	SOUTH ZONE
HON. STATE SECRETARY	DR. JITENDRA N. PATEL MOB. 98253 25200	AHMEDABAD
HON. JOINT SECY.	DR. SHAILENDRA N. VORA	AHMEDABAD
HON. ASST. SECY.	DR. BHARAT I. PATEL	AHMEDABAD
TREASURER	DR. DEVENDRA R. PATEL	AHMEDABAD
STATE SCIENTIFIC SECY.	DR. BUPENDRA M. SHAH	HIMATNAGAR
HON. JOINT ZONAL SECRETARIES	DR. BHARAT R. PATEL	AHMEDABAD
	DR. RASHMI UPADHYAY	WEST ZONE
	DR. PRADIP BHAVSAR	CENTRAL ZONE
	DR. VINOD MEHTA	VADODARA
	DR. NAVIN D. PATEL	SURAT
	DR. BHASKAR MAHAJAN	SOUTH ZONE
GUJARAT MEDICAL JOURNAL (GMJ) HON. EDITOR	DR. K. R. SANGHAVI	AHMEDABAD
SOCIAL SECURITY SCHEME HON. SECRETARY	DR. JITENDRA B. PATEL	AHMEDABAD
HEALTH SCHEME, I.M.A., G.S.B. CHAIRMAN	DR. NAVNEET K. PATEL	AHMEDABAD
COLLEGE OF G.P. DIRECTOR	DR. KIRIT C. GADHAVI	AHMEDABAD
ACADEMY OF MEDICAL SPECIALITIES CHAIRMAN	DR. VIDYUT J. DESAI	AHMEDABAD
PROFESSIONAL PROTECTION SCHEME MANAGING DIRECTOR	DR. BIPIN M. PATEL	AHMEDABAD

I.M.A. G.S.B. NEWS BULLETIN (Gujarat Medical Journal)

Vol. : 10

JULY-2015

Issue : 7

CONTENTS

- * State President and Hon. Secretary's Message18
- * From the Desk of Editors20

REVIEW ARTICLE

- * Spontaneous hemoperitoneum
Dr. Nitin M. Parmar*, Dr. Mrinal D. Patel**, Dr. Shubham S. Negi***, Dr. Chetan M. Savani****,
Dr. Nirmal L. Desai*****, Dr. Adarsh P. Patel*****25

ORIGINAL ARTICLE

- * Prehypertension, Heart rate, Obesity and their co-relation; Survey on Medical Students
Dr. Lavina Sinha*33
- * Visual Outcome and Major Surgical Complications of Manual Small Incision Cataract Surgery versus
Extra capsular Cataract Extraction performed by resident doctors in a Regional Institute of
Ophthalmology.
Agrawal Garima*, Thakker Hansa**, Audich Kamini***37
- * Diagnostic Role of Bone Marrow Aspiration and Trepine Biopsy in Haematological Practice
Dr. Shilpa Patel*, Dr. Pooja Nathani**, Dr. Nailesh Shah***, Dr. C.K. Shah****43
- * Study of Acute Kidney injury in H1N1 Patients (Year2015)
Dr. Jagdish Patel *, Dr. Abhijit Khadtare **, Dr. Ishwar Parmar**, Dr. Arjun Kelaiya**, Dr. Chintan Prajapati***48
- * Grey hair turning to black following Mesenchymal Stem Cell Therapy in patients with neurological
disorders: A Report of 4 Cases
Rajni Vyas, Dr. Bhaskar Vyas**67

CASE REPORT

- * Anaesthetic Management of Bilateral Carotid Body Tumor Resection
Dr. Saeed Ahmed Vohra*, Dr. Rini Majumdar**, Dr. Vinay Solanki***, Dr. B. M. Shah****,
Dr. J. M. Thakkar*****, Dr. B. M. Patel*****71
- * Apseudoaneurysm of the External Jugular Vein - A case report
Dr. Nina M. Shah*, Dr. Vishal S. Shah**, Dr. Parth R. Dalal***, Dr. Prafull Mishra****74
- * Intra Uterine Contraceptive Device in Urinary Bladder ~ An Iatrogenic Complication
Dr Rajal Thaker*, Dr Ajit Rawal**, Dr Sapana Shah***77

I.M.A. G.S.B. NEWS BULLETIN (Gujarat Medical Journal)

Vol. : 10

JULY-2015

Issue : 7

* Posterior Reverse Encephalopathy Syndrome In a case of Eclampsia Rajani Parikh*, Kanaklata Nakum*, A V Gokhle**	79
* Unilateral gestational macromastia – a rare disorder Hafsa M Vohra*, Harita M Desai**, Nilesh A Shah***	82
* Multiple Endocrine Neoplasia Type 1 (Wermer's Syndrome) Dr. A. A. Ghasura, Dr. K. H. Patel, Dr. Bharat Prajapati, Dr. Hitesh Mahida, Dr. Vikramaditya Oza.....	84
* Pregnancy In Rudimentary Horn: A rare entity Hina V. Oza*, Hafsa M. Vohra**, Nisarg H. Patel**, Daisy Y. Shethna***	87
* Hemoptysis as The Presenting Symptom in Ards in A Patient of Plasmodium Vivax Malaria (A Case Report). Dr. Alpa Leuva, Dr. Jyoti Mannari, Dr. Chinmaye Sapre.....	89
* Mandatory Submission Form	91
* Instruction of Authors	92

COPYRIGHT

No part of this publication may be reprinted or published without prior approval of the editor. Submissions of all manuscripts to the journal are understood to imply that it is not being considered for publication elsewhere. Submission of multi-authored manuscript implies that the consent of each & all authors has been obtained. Every effort has been made NOT to publish any inaccurate or misleading information in the journal. However, editor, editorial team, advisory board, publisher and printer accept no liability in consequence of such statements / information.

Disclaimer:

All statements, opinions, views etc expressed in the manuscripts by the authors are their individual ones and do not necessarily reflect those of I.M.A. G.S.B. NEWS BULLETIN (Gujarat Medical Journal) or its' editorial team or publisher. The editor(s) and/ or publisher(s) do not accept any type/ form of accountability/liability for such material.

The editorial team and publisher neither guarantee nor endorse any product or service advertised in the journal. Any claim made by the manufacturer of such product or service is a matter of solicitation from manufacturer/ distributor of said product/ service.

Spontaneous hemoperitoneum**Dr. Nitin M. Parmar***, **Dr. Mrinal D. Patel****, **Dr. Shubham S. Negi*****, **Dr. Chetan M. Savani******,
Dr. Nirmal L. Desai*****, **Dr. Adarsh P. Patel********Associate Professor, **4th year Resident, ***Assistant Professor, ****3rd year Resident, *****2nd year Resident, 1st year Resident, B.J.Medical College, Civil Hospital, Ahmedabad, Gujarat, India.**KEY WORDS** : spontaneous hemoperitoneum, Warfarin, Acute abdomen**ABSTRACT**

Acute abdomen due to Spontaneous hemoperitoneum is a serious condition demanding careful consideration from the treating surgeon. Though uncommon, everyone sometimes faces such a surgical dilemma and there arises a need to contemplate the multifold and diverse etiology associated with it. A sound knowledge of the potential causes of acute abdomen and massive spontaneous hemoperitoneum along with a high index of suspicion are necessary for proper patient management.

We came across two patients of acute abdomen due to spontaneous hemoperitoneum. Here, we discuss the two cases and the review of literature related to it.

INTRODUCTION

Spontaneous hemoperitoneum is defined as the presence of intra-abdominal haemorrhage from a non-traumatic cause. It usually presents as acute abdomen with shock.^{1,2,3} Hence, it is pertinent to include spontaneous hemoperitoneum in the differential diagnosis of acute abdomen. Our aim is to discuss the cases and review the literature on the diverse etiology associated with spontaneous hemoperitoneum that presents as acute abdomen.

CASE REPORT 1

A 33-year-old man with a history of alcohol abuse was admitted in our hospital with acute right upper quadrant abdominal pain and signs of hypovolaemia. He had a history of high grade fever before 10 days. He had no history of wt loss. On examination, he was pale, dehydrated, with a tense and distended abdomen but no signs of external bleeding; blood pressure was 90/50 mmHg and heart rate 116 bpm. His haemoglobin was 7.2 g/dl and there was no history of trauma. He was admitted to the intensive care unit (ICU) and after stabilization, abdominal ultrasonography was done, which revealed free intraperitoneal fluid. Diagnostic paracentesis revealed blood in the right paracolic gutter. After transfusion of four units of red cell concentrate, hemodynamic stability was achieved. CECT of abdomen and pelvis revealed a 6*6 cm mass in segment VII and VIII of liver. Pt was initially managed conservatively, but on 7th

post admission day laparotomy was performed as patient again showed signs of internal bleeding. It revealed a 3-litre hemoperitoneum and a 6 cm diameter liver tumour in segment VII and VIII with breach in capsule and bleeding. Paraffin gauge and absorbable gelatin sponge packing in tumour site was done to stop bleeding. Abdomen was closed with packing & Abgel in situ, for second look laparotomy if needed. But the patient did not maintain hemodynamic stability and died on day 10

CASE REPORT 2

A 42 year female presented to the emergency department with acute abdominal pain and breathlessness for the last 6 hours. She was conscious but tachypnoeic, extremely pale with cold extremities and a weak and rapid pulse rate of 120 beats per minute with a systolic blood pressure of 90 mm Hg and generalised abdominal distension and tenderness. There was no evidence of any external bleeding or hematuria and there was no history of trauma.

Her medical history revealed that she had been previously diagnosed with cerebral venous thrombosis two years ago for which she was on oral warfarin and leviteracetam. Laboratory data showed haemoglobin of 3.2 with hematocrit 20, normal platelet count, total WBC count, urea, creatinine and liver function tests and serum lipase. Her PT was 35 with a control of 13 and INR 2.57.

The patient was resuscitated with i.v. crystalloids, packed cell and Fresh frozen plasma. A portable ultrasonography showed free fluid in peritoneal cavity with internal echoes.

Correspondence Address : **Dr. Shubham S. Negi**
49, Parmeshwar-2, B/h Satyamev Hospital, Chandkheda, Ahmedabad 382424
Email : drnegishubham@gmail.com

However, abdominal radiographs were normal. After sufficient hemodynamic stability, CECT of thorax abdomen and pelvis was performed to identify cause of intra peritoneal bleed. CECT shows approximately 12*8*6 cm sized non enhancing heterogeneously hypodense lesion in pelvis postero-superior to uterus, suggestive of hematoma with pockets of fluid within. No source of active bleeding was identified.

Based on history, laboratory and CT findings a diagnosis of warfarin induced spontaneous hemoperitoneum was deduced and warfarin was immediately stopped. The patient was managed conservatively in ICU for 7 days on bi-PAP mode for respiratory distress with further PCV and Fresh frozen plasma transfusions, vitamin K and prophylactic antibiotics. Starting from 5th post admission day, abdominal tenderness and abdominal girth begun to reduce and she showed signs of improvement. Her follow up MR venography brain showed no evidence of any thrombus. The patient was discharged on 10th day with omission of warfarin. At two month follow up, patient was asymptomatic.

DISCUSSION

Idiopathic spontaneous intraperitoneal hemorrhage (ISH) was first reported by Barber in 1909 and was later termed "Abdominal apoplexy" by Green and Powers in 1931. Its true incidence is unknown.⁴

The various causes of spontaneous hemoperitoneum are classified into following based on source of bleeding.^{1,5,6}

HEPATIC CAUSES

Spontaneous hepatic bleeding is mainly due to rupture of underlying hypervascular tumour. It may be due to rupture of benign hepatic adenoma which usually occurs in young women receiving long-term oral contraceptive therapy or in pregnancy or in males with exogenous androgenic steroids ingestion. Hemangioma and Focal nodular hyperplasia are also common benign liver lesions. However, their rupture is an infrequent event mostly related to increased intravascular volume that occurs during pregnancy.^{5,6} Rupture of a hepatocellular carcinoma or primary angiomyosarcoma of the liver are malignant lesions known to cause spontaneous hemoperitoneum. Rupture of a metastatic lesion from primary malignancy in colon, lung, kidney, testis and choriochoriocarcinoma has also been reported to cause SH.

Both primary and metastatic tumours can be highly vascular and necrotic and therefore prone to intraperitoneal rupture. Factors that contribute to bleeding may include increased intravascular pressure secondary to tumour embolus, causing intra-hepatic venous

obstruction with shunting of blood, and a hyperaemic liver circulation caused by proximity of vessels to metastatic nodules or primary tumours. However, direct pressure of the tumour against the capsular surface of the liver seems the most plausible explanation. Extensive replacement of liver tissue by tumour together with poor nutrition may reduce coagulation factors and promote haemorrhage. Systemic chemotherapy can also lead to considerable tumour necrosis as well as thrombocytopenia. A sudden increase in intra-abdominal pressure resulting from sneezing, coughing or vomiting may cause rupture of tumours that are necrotic or hyper-vascular. Minor trauma or iatrogenic damage by needle biopsy or liver palpation should be ruled out before the rupture and hemoperitoneum are classified as spontaneous.^{7,8,9,10}

There is some controversy regarding the treatment of choice in this situation. Bleeding is often difficult to control, and the mortality rate is high. The available treatment options are merely palliative unless resection is possible. Surgical treatment includes hepatic wedge resection or lobectomy, ligation of the bleeding source and hepatic artery ligation. Several authors advocate an aggressive surgical intervention with resection of the affected liver lobe whenever possible. Unfortunately, only a few patients are suited for this procedure, owing to the presence of cirrhosis or extensive replacement of liver tissue by tumour. Hepatic artery ligation may stop the bleeding, but it is associated with a high risk of death from liver failure.

Transarterial embolization (TAE), in which a mixture of gelfoam and mitomycin C is injected in the feeding artery of the tumour, can achieve good hemostasis. Intratumoural injection of absolute alcohol has also been used to treat haemoperitoneum secondary to non-traumatic liver rupture, based on the ability of this substance to destroy HCC under ultrasonographic control and to stop the bleeding in oesophageal varices and peptic ulcers. Bleeding stops owing to a process of tissue dehydration and fixation, followed by thrombosis of the vessel. It may be useful when resection or hepatic ligation is either not possible or ineffective, and when transcatheter embolisation is not available. Recent developments include percutaneous radiofrequency ablation (RFA) of the tumour.

The documented survival of patients with spontaneous rupture of hepatomas is extremely poor. The outcome is determined by the stage of both the neoplastic and the underlying liver disease, the rapidity of diagnosis, the degree of haemorrhage and the type of therapy.

Infiltrative disease of the liver like amyloidosis and

Table 1: Causes of spontaneous hemoperitoneum

<p>CAUSES OF SPONTANEOUS HEMOPERITONIUM</p> <p>1. Hepatic</p> <ul style="list-style-type: none"> • Rupture of hepatic adenoma/ hepatic adenomatosis/ hemangioma/ FNH • Rupture of Hepatocellular carcinoma/ primary angiosarcoma • Rupture of metastatic lesion(colon, lung, Renal Cell Carcinoma, testicular, wilms, choriocarcinoma) • Benign infiltrative disease of liver, Amyloidosis • Liver rupture (HELLP syndrome) • Cirrhosis with portal hypertension and intraperitoneal rupture of varices • Spontaneous bacterial peritonitis leading to hemoperitoneum 	<p>6. Gynecological</p> <ul style="list-style-type: none"> • Rupture of ovarian cyst • Rupture of ectopic pregnancy • Retrograde menstruation • Ectopic endometrial tissue • Metastatic disease like gestational trophoblastic tumour • Pregnancy/Post partum spontaneous hemorrhage(rupture of uterine vessel) • Hemorrhagic corpus leuteum cyst torsion • HELLP syndrome • Rupture of uterine leiomyoma
<p>2. Splenic</p> <ul style="list-style-type: none"> • Post trauma – delayed splenic rupture • Infectious mononucleosis Cytomegalo virus infection, AIDS, malaria, EBV, bartonella • Hematological malignancy associated splenomegaly (leukemia/ lymphoma) • Tumor – hemangiopericytoma, Primary/secondary angiosarcoma • Splenic cyst/ abscess • Splenic infarcts(infective endocarditis) • Torsion of wandering splenic pedicle • Torsion of spleen • PHT and splenomegaly with liver cirrhosis • Sickle cell anemia • Infiltrative diseases (Amyloidosis, Gaucher's disease) 	<p>7. Vascular</p> <p>Arterial</p> <ul style="list-style-type: none"> • Rupture of arteriosclerotic/ mycotic/ congenital aneurysm • Splanchnic arterial aneurysm/ mesenteric/ retroperitoneal vessel rupture • Erosion of vessel by neoplastic/ inflammatory disorder(pancreatitis/ cholecystitis / appendicitis / Meckel's diverticulitis) <p>Venous</p> <ul style="list-style-type: none"> • Spontaneous intra abdominal variceal rupture • Rupture of uterine veins • Rupture of rectus hematoma into peritoneum <p>A-V malformations</p>
<p>3. Renal</p> <ul style="list-style-type: none"> • Angiomyolipoma • Renal Cell Carcinoma • Coagulopathy • Vasculitis – PAN, Wegeners 	<p>8. Anticoagulation</p> <ul style="list-style-type: none"> • Hemophilia • Idiopathic Thrombocytopenic Purpura • Hepatic failure • Anticoagulant therapy(Warfarin/ Heparin) • SLE/PAN • Blood dyscrasias • Myeloproliferative disorder(leukemia/ lymphoma) • Dengue • Disseminated Intravascular Coagulation • Severe celiac disease with vit K malabsorption • Rodenticide poisoning Brodifacoum(superwarfarin) • Congenital factor X deficiency
<p>4. Adrenal</p> <ul style="list-style-type: none"> • Severe stress • Sepsis • Anticoagulation 	<p>9. Other</p> <ul style="list-style-type: none"> • CAPD (continuous ambulatory peritoneal dialysis) • Hemodialysis dependent
<p>5. Gastrointestinal</p> <ul style="list-style-type: none"> • Diverticulum of sigmoid colon • Colonic/ intestinal / peptic perforation • Angiodysplasia / A V malformation of the gut 	<p>10. Idiopathic</p>

gaucher's disease may also result in SH. There have also been singular case reports citing gall bladder necrosis and spontaneous bacterial peritonitis as the pathology behind SH.

Intraperitoneal rupture of ectopic varices is a rare complication of portal hypertension. Objective management included suspicion for diagnosis, aggressive resuscitation and correction of coagulopathy, reduction of portal hypertension and if required surgical intervention for direct control of the bleeding.¹¹ Spontaneous liver rupture with hemoperitoneum also occurs in cases of HELLP syndrome in pregnant females. It is discussed under gynaecological causes.

SPLenic CAUSES

Delayed splenic rupture may occur following blunt abdominal trauma. Spontaneous splenic rupture mainly occurs in cases of marked splenomegaly because of underlying hematologic malignancies (acute leukemia or lymphoma) or infectious causes such as mononucleosis or Cytomegalovirus, malaria, EBV, Bartonellahenselae, AIDS in young patients. Few cases are reported citing spontaneous rupture of hypervascular tumors like hemangiopericytoma leading to SH. Wandering spleen is an unusual condition characterized by increased splenic motility due to ligament laxity and its torsion can lead to spontaneous splenic rupture.¹²

Clinical presentation in such cases includes acute abdominal and shoulder pain due to diaphragmatic irritation. Diagnosis at CT is suggested by the identification of a grossly abnormal spleen with perisplenic hemorrhage or a clot in the organ. Treatment may be conservative, or surgical in the form of total or partial splenectomy, or transcatheter embolization, depending on the underlying disease.^{5,13}

GASTROINTESTINAL CAUSES

Peptic perforation or colonic perforation can lead to spontaneous hemoperitoneum and be associated with fecal/ biliary peritonitis. Angiodysplasia of the colon can be the cause of spontaneous intra peritoneal bleeding. Such cases frequently need urgent exploration to treat the cause.^{14,15}

Only 2 cases of hemoperitoneum resulting from colonic diverticulum have been reported, one due to hypertension and other due to spontaneous bleeding of serosal vessel of colonic diverticulum. Invagination and diverticulectomy are two ways to treat diverticula while preserving the blood supply of the bowel. Invagination of bowel minimizes bowel leakage and can be easily performed laparoscopically with suture technique.¹⁵

GYNECOLOGICAL CAUSES

Rupture of an ectopic pregnancy or rupture of an ovarian cyst are the most common causes of spontaneous hemoperitoneum in women of childbearing age.

Ectopic pregnancy is a potentially life-threatening condition that must be considered in every woman of reproductive age with abdominal or pelvic pain, usually starting with measuring the serum HCG and performing pelvic sonography. In the emergency setting, CT may be performed in these patients because of the presenting severe symptoms and a falsely negative urine pregnancy test. Ectopic pregnancy commonly occurs in the fallopian tube and presents as a ring-enhancing adnexal cystic mass surrounded by hemoperitoneum. Emergency laparotomy is required in this condition.^{1,5,6}

Rupture of an ovarian cyst should be suspected in young women presenting with pelvic pain and negative serum β -HCG. Ectopic endometrial tissue or metastatic diseases like gestational trophoblastic tumor can also lead to spontaneous hemorrhage. Retrograde menstruation is one condition where in USG/CT findings of hemoperitoneum may not reveal any associated pathology.

SH during pregnancy is rare but has high morbidity and mortality rate. The pre operative diagnosis is usually placental abruption which masquerades SH.¹⁶ The etiology of SH in pregnancy or post partum period is poorly understood. Apart from spontaneous rupture of tumors associated with pregnancy, as discussed previously, spontaneous rupture of utero- ovarian vessel remains a possibility.¹⁷

Utero –ovarian vessel dilatation due to increase physiological demand of pregnancy, in conjunction with a sudden rise in venous pressure resulting from muscle contraction associated with labour, coughing, defecation or coitus may increase tendency for vessel rupture. Other contributing factors include tortuous nature, lack of valves and repeated distention of this vessel during pregnancy. Volume replacement and timely Ceasarian Section is very essential in such cases to save both the mother and the baby.

Spontaneous abdominal hemorrhage in pregnancy associated with toxemia is usually a manifestation of the HELLP syndrome. HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome is a severe variant of preeclampsia that should always be considered in pregnant women with acute abdominal pain and accompanying laboratory abnormalities. This serious obstetric condition may be associated with hepatic necrosis and intrahepatic hemorrhagic infarction. In these

patients, CT is the study of choice to detect hepatic sub capsular hematomas, intrahepatic liver hemorrhage, and infarcts. Treatment consists of expeditious delivery of the neonate and emergency surgery or selective embolization of hepatic arteries in case of liver rupture for the mother.⁶

Hemorrhagic corpus luteum cyst torsion, leiomyosarcoma, and other uterine tumors have been reported to cause SH.

VASCULAR CAUSES

Vascular causes are divided into arterial and venous causes. Arterial causes of SH included aneurysm, pseudo aneurysm, mycotic aneurysm and arterial dissection.

CT is usually performed in patients with known abdominal aortic aneurysm (AAA) presenting with abdominal pain to exclude rupture or to identify other causes for the patient's symptoms. On unenhanced CT images, findings associated with increased risk of rupture include increasing diameter of the aneurysm (> 5 cm), focal discontinuity in circumferential wall calcifications, and presence of a crescent-shaped area of high attenuation in the mural thrombus or in the aneurysmal wall, known as the hyper attenuating crescent sign

Rupture is usually associated with a large retroperitoneal hematoma adjacent to the aneurysm. Very few patients with intraperitoneal rupture of AAA survive to make it to the hospital and rarely upto a CT scan. Management includes volume resuscitation followed by surgical correction in the form of resection of aneurysm, arterial reconstruction.

In patients with a concomitant coagulopathic condition, it is critical to determine the cause of the retroperitoneal hematoma because a ruptured AAA requires prompt treatment by surgery or endovascular intervention, whereas surgery is usually contraindicated in cases of coagulopathic hemorrhage.¹⁸

Less common vascular causes of spontaneous abdominal hemorrhage include rupture of a splanchnic artery aneurysm and erosion of a vessel by an adjacent neoplastic or inflammatory disorder (e.g., pancreatitis). Though splenic and renal arteries are most commonly involved, spontaneous rupture of left gastro epiploic artery, hepatic artery, gastric artery, mesenteric artery have been mentioned.

Hemoperitoneum resulting from venous cause is usually due to rupture of abdominal varices in cirrhotics with a mortality of 75%. It may also result from increase intra abdominal pressure associated with labor as discussed previously. A-V malformation of GIT may also result in SH.

ANTICOAGULATION

Abdominal hemorrhage due to anticoagulation or bleeding diatheses (e.g., hepatic failure, hemophilia, idiopathic thrombocytopenic purpura and systemic lupus erythematosus) commonly involves multiple sites, and especially the body wall muscle compartments, such as the rectus sheath or the iliopsoas muscle. Abdominal viscera are less commonly the sites of coagulopathic hemorrhage, but cases of ruptured liver, spleen and perirenal and intramural bowel hematomas have been cited. When contrast-enhanced CT detects coagulopathy-associated active extravasation, this is more frequently venous than arterial, usually not requiring surgery or embolization. Treatment is mainly conservative and based on withholding of anticoagulant medications.

Dengue fever associated anticoagulation has been reported to cause SH. Dengue fever can lead to bleeding manifestations like gum bleeding, bleeding into internal organs and bleeding into serosal cavities with altered or normal abdomen. Thus, regular monitoring of vital signs, hemoglobin, hematocrit, platelet counts, serology of dengue is important when hemoperitoneum is associated with fever.¹⁹

SH can occur in superwarfarin (Brodifacoum) rodenticide exposure (which is mostly accidental) that causes severe bleeding problems that may last for month.^{20,21}

There have been reports of hemoperitoneum resulting from GB rupture in patient on anticoagulation, spontaneous rupture of spleen in patient on heparin. The most common hematological disorder that may result in SH is hemophilia. Congenital factor X deficiency has also been reported to cause SH after rupture of luteal cyst.

Hemodialysis dependent population is at a particular risk for SH. These patients have poorly functioning platelets that predispose them to spontaneous bleeding. Also they receive significant quantities of heparin at each hemodialysis which increase susceptibility to bleed.²²

Here, we would like to include a specific mention about warfarin which is the commonest cause of drug induced spontaneous hemoperitoneum.^{23,24,25,26}

Warfarin is a life saving drug, extensively used in the treatment and the prophylaxis for the various clinical conditions. Bleeding in the gastrointestinal tract is by far the most common complication of the warfarin therapy

The unmasking of previously unidentified bowel tumors has been described after spontaneous warfarin-associated bleeding. However, spontaneous mesenteric hemorrhage is extremely rare.

Prescribing the dose that both avoids hemorrhagic complications and achieves sufficient suppression of thrombosis requires a thorough understanding of the drug's unique pharmacology. Warfarin has a complex dose-response relationship that makes safe and effective use a challenge. Warfarin exerts its action by inhibiting Vitamin K dependent coagulation factors (II, VII, IX and X). It also inhibits the synthesis of natural anticoagulants in the blood, protein C and S. Due to the difference in the half life of "coagulation factors" and "anticoagulants", the coagulation system may be transiently biased towards

The maintenance dose of warfarin can fluctuate significantly depending on the amount of vitamin K in the diet.

The two most important determinants of the warfarin induced bleeding is the intensity of therapy and the maximal time in therapeutic range. Bleeding is a major complication in the early phase of the warfarin therapy according to the most studies. Bleeding is more likely to occur in the patients with the more intense therapeutic range (INR between 2.5 and 3.5) than in the less intense therapeutic range of warfarin (INR between 2 and 3). However cases of SH are reported even with less intense range of INR. Dedicated monitoring of the coagulation profile in patients taking long-term warfarin is mandatory to prevent this complication.



Fig 1.CECT abdomen showing HCC mass

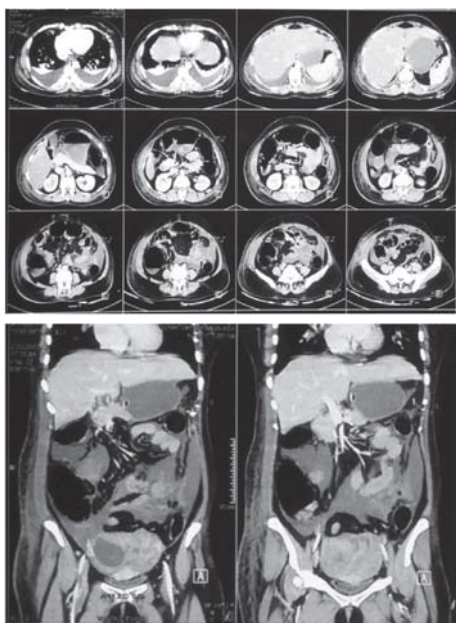


Fig 2.CECT images showing hemoperitoneum without any active extravasation of contrast in the patient with warfarin induced spontaneous hemoperitoneum

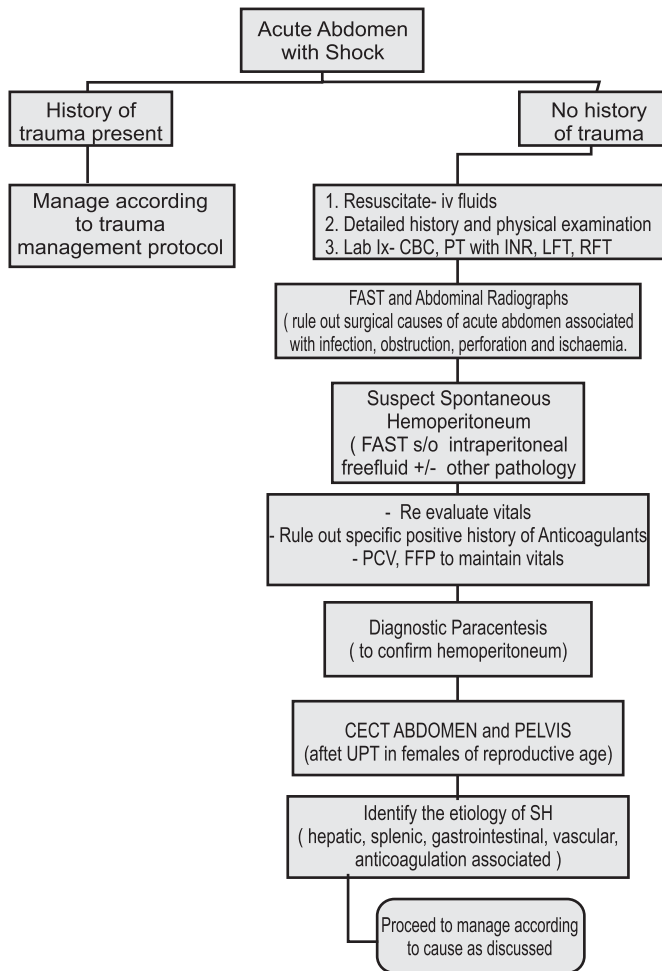
clotting after starting warfarin. The target International normalized ratio (INR) is maintained around 2–3

A high INR predisposes to a high risk of bleeding, while an INR below the therapeutic target indicates that the dose of warfarin is insufficient to protect against thromboembolic events.

MANAGEMENT

Treatment of spontaneous intraperitoneal bleeding revolves around resuscitation and restoration of circulating volume. This has traditionally been followed by surgical correction.

Figure: Suggested management protocol for suspected spontaneous hemoperitoneum in a patient with acute abdomen.



Imaging in patient with SH has two primary functions, (1) diagnosis of blood in peritoneal cavity, (2) to identify underlying cause. In the hemodynamically unstable patient, Focused Assessment with Sonography for Trauma (FAST) examination may be useful to detect intra-abdominal hemorrhage. It also has a large role in diagnosis of SH secondary to gynaec causes. However, CT scan represents the most important imaging technique. The use of intra venous contrast is currently recommended if the patient is stable enough for the delay associated with administering oral contrast. CT angiography of vessels has proven useful as a screening tool using small amounts of contrast to elucidate sites of active bleeding. MR is complementary to CT and is useful in confirming the diagnosis and in special cases like pregnancy.^{5,6}

CONCLUSION

Spontaneous Hemoperitoneum SHOULD BE considered in the differential diagnosis of patients who have acute abdomen with falling hematocrit and with signs and symptoms of hypovolemia with associated positive history of a predisposing condition to avoid a dangerous diagnostic delay and culmination into a catastrophe.

REFERENCES

- Lucy bc, et al, spontaneous hemoperitoneum, a bloody mess, emergency radiology, june 2007, vol 14, issue 2, page 65-75
- Subee WJ Jr, Cunningham PL, Massive Spontaneous hemoperitoneum of unknown etiology: a case report: Block R2, Am Surgery, 1998 Dec, 64(12):1177-8
- Ami schattner, MD, Natasha Kozack MD, Joshna Friedman, MD Arch. Idiopathic Spontaneous hemoperitoneum; international med 2001; 161 (7); 1009-1010
- Jadav M, Ducheine Y, Brief D, Carter L, McWhite T, Hardy J.; Abdominal Apoplexy: A Case Study of the Spontaneous Rupture of the Gastroepiploic artery.
- Brian c lucy, et all, spontaneous hemoperitoneum- causes and significance: Current problem in diagnostic radiology vol- 34, issue 5, page 182-195 sept 2005
- Alessandro Furlan, Saced Falcliram and Michal P Fedeele- Spontaneous abdominal hemorrhage; Casues, CT Findings, and Clinical Implication: Citation – Americal Journal of Roentgenology, 2009; 193:1077-1087
- LX Zhu, GS Wang, SI Fan –spontaneous rupture of hepatocellular carcinoma-British Journal of Surgery , 1996, V.83, N.5, P.602-607
- LX Zhu, XP Geng, SI Fan -spontaneous rupture of hepatocellular carcinoma and vascular injuries-Archives of Surgery, 2001, V.136, N.6, P.682-687
- Fugii M, Miyake H, Takamura K, Tashiro S; management of spontaneous rupture of hepatocellular carcinoma-Nihon-Geka-Gakkai-Zasshi; 2004 APR; 105(4) 292-5. PMID-15112491
- Li WH, Chen EC, Kwok PC, Cheung MT-Survival after transarterial embolization for spontaneous ruptured Hepatocellular carcinoma, J Hepaticobiliary Pancreatic-SURG, 2009; 16, 508-12
- Spontaneous hemoperitoneum in Non Cirrhotic Portal Hypertention : A Case Report- Program No P-264, ACG-2012. Annual scientific Meeting Abstract, Las Vegas, NV: American College of Gastroenterology
- Sucandy I, Akamal YM, Gabrielsen JD. Spontaneous massive hemoperitoneum: a potentially Life Threatening Presentation Of Wandering Spleen. North American Journal of medical science 2011; 3:99-102
- Picozzi Stefano, Carlo Maria, Masino Mauri, Luca camignani. A unique case of hemperitoneum; spontaneous rupture of splenic hemangiopericytoma; International Journal of Emergency Medicine 2011 4:13 doi 10.1186/1865-1380-4-13
- Karim Ibn Majdoub Hassani, Ali Bounckar, Jean Manuel Grun. Spontaneous rupture of right gastro epiploic artery: unusual cause of acute abdomen and shock; World Journal Of Emergency Surgery 2009/4:24: DOI 10.1186/1749-1922-4-24
- Kye BH, kim HJ, yoon Jh, Pack DC, Lec SJ. Spontaneous hemoperitoneum caused by a Diverticulum of the sigmoid colon; International journal of Med sci 2011; 8(6): 467-469 DOI 10.7151/ijms.8.467
- Subedi s, Banerjee B, Spontaneous hemoperitoneum masquerading Placental Abruption: a case report. NJOG 2012, Jul-Dec 7(2): 50-51
- Jennifer lu Gao, BHSc, Karine, Lortie, MD, Sukhbirsonysing MD. Laparoscopic Internal Iliac Artery Ligation For Post Partum Spontaneous hemoperitoneum; JOAC Dec 2010, 1172-1175
- D Y H Yap, T P S Yip, S L Lui and W K Lo. Ruptured Abdominal Aortic Aneurysm as a cause of spontaneous hemoperitoneum in a patient on Peritoneal Dialysis international, 31(5), 600-602, 2011
- NKT Chandrashekar, et all, Spontaneous hemoperitoneum in dengue fever with normal coagulation profile: JGlob Infect dis. 2013 Jan-Mar; 5(1): 29-30. Doi 10.4103/0974-777X. 107172
- Austin T Nelson, BS, Joshna D. Hartzell, MD, Kenneth Moe, MD, Steven J Durning MD. Ingestion of superwarfarin leading to coagulopathy: A case report and review of literature: MedGenMed 2006; 8(4): 41. Published online 2006 nov 28.
- Jeri Patoka, Georg Petroionu, Kamilkula, Toxic Potential of Spuperwarfarin Brodifacoum; Mil Medical Science lett (VojZdrav Listy) 2013, vol 82(1) p 32-38, ISSN 0372-7025
- G. Stephnnace, Alfred L, Geerge JR, William J Stone Hemoperitoneum – Red Flag in CAPV; journal of international society for peritoneal dialysis; Peric Dial Int. January/March 1985/ vol 5 no 1 page 42-44
- Back, Jong Bum, Seon, SeungHorn; Won, Dal Yeon, Bae, Keum, Kim, IK Young. Spontaneous hemoperitoneum in Patients taking Anticoagulants; JKorean Surg Soc. 2010 jun 07 ; 78(6) 369-375.
- Jayesh Saga, Vikas Kumar, Dharmendra K Shah and Ashok Bhatnagar. Spontaneous intra peritoneal bleeding secondary to warfarin , presenting as an acute appendicitis: A Case report and review of literature; BMC Blood Disorders 2006, 6:7, DOI 10.1186/1147-2326-6-7

25. Sankar Subramanian, Subramanian ManappaGoundee, ArunkumarThirunarayanan, Anand Kumar, and NandigamVenu. Retroperitoneal hemorrhagic shock in a patient on warfarin therapy. *J. Emergency Trauma Shock*, 2009 may 2(2):137-138
26. R.Dibbad, M.Deshmukh, H. Merchant, S. Shikhan, Spontaneous Intramural Hematoma of The Small Bowel- A complication Of Anticoagulation Therapy. *The International Journal Of Radiology*; 2010 vol 12 No.1: DOI: 10.5580/c6c
27. Rad MN, Abdollahi O, Golshahi H, FardRMN(2013). Idiopathic spontaneous Intraperitoneal Hemorrhage: A Case Report ; *J Clinical Case Reports* 3:289; DOI 10.4172/2165-7920.1000289
28. AmramHadary MD, IgoeDashkovskyy MD, AerkadyRapaport MD and Juan C. Lozakov. Non Traumatic Rupture of Spleen-Can Spleennctomybe Applied Selectively? ; *IMAJ*, Vol 10, Dec 2008 889-891Battula N
29. , Madanur M, Priest O, Srinivasan P, O'Grady J, Heneghan MA, Bowles M, Muiesan P, Heaton N, Rela M –spontaneous rupture of Hepatocellular carcinoma; western experience.*Am J. Surg.*2009 Feb;197(2);164-7.VOL 10.1016.J.amj surg.2007.10,10,016 Epub 2008.OCT 16.
30. Bird D, Vowles K, Anthony PP. Spontaneous rupture of a liver cell adenoma after long term methyltestosterone: Report of a case successfully treated by emergency right hepatic lobectomy. *Br J Surg*. 1979;66:212–3.
31. Mokka R, Seppala A, Huttunen R, et al. Spontaneous rupture of liver tumors. *Br J Surg*. 1976;63:715–7.
32. Nagasue N, Inokushi K. Spontaneous and traumatic rupture of hepatoma. *Br J Surg*. 1979;66:248–50.
33. Hsieh JS, Huang CJ, Huang YS, et al. Intraperitoneal hemorrhage due to spontaneous rupture of hepatocellular carcinoma: treatment by hepatic artery embolization. *AJR Am J Roentgenol*. 1987;149:715–7.
34. Schoedel KE, Dekker A. Hemoperitoneum in the setting of metastatic cancer to the liver. *Dig Dis Sci*. 1992;37:153–4.
35. Czarnecki DJ. Intraperitoneal hemorrhage diagnosed by technetium-99m labeled RBC imaging. *ClinNucl Med*. 1986;11:61
36. Urdaneta LF, Nielson JV. Massive hemoperitoneum secondary to spontaneous rupture of hepatic metastases: Report of two cases and review of the literature. *J SurgOncol*. 1986;31:104–7.
37. Ong GB, Taw HL. Spontaneous rupture of hepatocellular carcinoma. *Br Med J*. 1972;4:146–9.
38. Chung SCS, Lee TW, Kwok SPY, Li AKC. Injection of alcohol to control bleeding from ruptured hepatomas. *Br Med J*. 1990;301:421
39. James T. Loynes, . Spontaneous hemoperitoneum resulting from rupture of Hepatocellular Carcinoma; Hospital physician; Feb 2001, PG 68-71
40. Ronald A, Squires, Russell G. Postier; Sabiston textbook of surgery, 19th edition, vol 2 chapter 47, PG 1140-59

Prehypertension, Heart rate, Obesity and their co-relation; Survey on Medical Students

Dr. Lavina Sinha*

*Assistant Professor, Department of Medicine,
GMERS Hospital and Medical College, Sola, Ahmedabad.

KEY WORDS : Prehypertensives¹, Obesity² and blood pressure³

ABSTRACT

A common man may be of the opinion that a medical student is generally aware about his health condition, always has hygienic food, stays in good and clean environment and his daily routine is such that it keeps him in healthy condition. But on the contrary, there is need for better health awareness and a healthy lifestyle even among the medical students, along with the society in general. The objective of this research is to study the prevalence of prehypertension, hypertension and obesity among the medical students and factors associated with them. The present study, is a cross sectional study which is focused on 265 medical students of C. U. Shah Medical College, Surendranagar, Gujarat State, who voluntarily gave their consent to participate. Clinical examination, Temperature, Pulse, Blood Pressure, Respiratory Rate, Heart Rate and Body Mass Index were studied for this group of students. Family history of hypertension, diabetes, obesity and ischemic heart disease were also noted in the study. Predesigned and prevalidated format has been used as methodology for this research. Based on the above mentioned tests and the evaluation of the data collected, the statistical analysis done established a significant co-relation between prehypertension, heart rate and obesity. The result clearly indicates that prehypertension was highly prevalent in the group of medical students under the present study.

INTRODUCTION

There is growing incidence of hypertension occurring in younger ages as compared to older age group as in the past. Hypertension is an independent predictor of cardiovascular disease and cerebrovascular events affecting nearly a quarter of the adult population worldwide. Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC7)¹ defines hypertension as blood pressure >140/90 mmHg. Persons with systolic blood pressure of 120-139 mm Hg or diastolic blood pressure of 80-89 mm Hg are defined as having "prehypertension". Subjects with pre-hypertension have a greater risk of developing hypertension later and increased risk of major cardiovascular events independent of other cardiovascular risk factors. Cardiovascular diseases contribute to atleast one third of deaths in India every year. Although hypertension has been well studied, there is lack of enough data on the prevalence and risk factors for prehypertension in India.

With growing urbanization and the effect of Westernization in India there has been an increase in lifestyle disorders. These disorders are seen affecting the younger age groups and adolescents at an increasing

rate. Physical inactivity, stress on technology, computers and alarming consumption of junk food in the childhood has led to youth suffering from obesity and prehypertension. Prehypertension tends to go unnoticed. However the need of the hour is to identify these cases intervene early on to prevent the sequelae viz. hypertension and other cardiovascular complications⁶. Rising affluence has also modified the dietary pattern characterized by increased consumption of diets rich in fat, sugar and calories⁴. The all cause mortality has also been shown to be 50% higher in the prehypertensive adults compared to the normotensive counterparts.

Drug therapy for prehypertension is not recommended for various reasons ^{2,3}. Our study population consisted of students enrolled in a medical school and had similar dietary habits & lifestyle. Early identification of prehypertension in this subgroup plays an important role in screening for metabolic syndrome and identifies modifiable factors required for proposing prevention strategies for prevention of cardiovascular accidents.

OBJECTIVE

To study the prevalence of prehypertension, hypertension and obesity in medical students of our institute and factors associated with them.

Correspondence Address : **Dr Lavina Sinha**
Department of Medicine, GMERS Hospital and Medical College, Sola, Ahmedabad.

METHODOLOGY

A cross-sectional study was done and 265 students from medical institute of Surendranagar (Gujarat) in the age group 18-22 years were enrolled in the study. Written informed consent was taken. Their demographic data was collected which included their height, weight, habits. Family history of hypertension, diabetes, obesity and ischemic heart disease was noted in the study. BMI was calculated using the standard international formula. To measure the blood pressure a standardised mercury sphygmomanometer was used. A cuff bladder encircling at least 80% of the arm circumference was applied to the non dominant arm. The disappearance of phase V Korotkoff sounds was taken as the diastolic reading. The mean of three readings, recorded 2 min apart, was taken. If these readings differed by more than 5 mm Hg a further three readings were recorded at 2 min intervals and the mean of all six readings was taken. All the students were subjected to measurement of pulse, BP, RR, HR and BMI and a thorough clinical examination was performed. The radial pulse rate was manually recorded over a 1 min period. The individuals were categorized as normotensives, prehypertensive and hypertensive. Known hypertensives & students on antihypertensive management & non consented students were excluded from the study. SPSS version -2 was used for data analysis, Student t-test was used to calculate the correlation coefficient.

RESULTS

From among a target group of 265 students, 106 were males, 159 were females. Prevalence of normotension was 28.8%, prevalence of prehypertension- 61.1% out of which 40.7% were males while 59.3% were females. The prevalence of hypertension was 10.1% out of which 59.3% were male while 40.7% were female. The association between gender and BP (prehypertensive and hypertensive) is significant (p-value=0.039664).

Table 1: Prevalence of Prehypertension and Hypertension in Males and Females

	Males	Females	Total
Normotensive	24	52	76
Prehypertensive	66	96	162
Hypertensive	16	11	27
Total	106	159	265

According to their BMI 171 (64.5%) students had normal weight, 65 were underweight (i.e. 24.5%), 25 were preobese (i.e. 9.43%) and 4 students were obese (i.e. 1.5%). Amongst the preobese students 76% were males

and 24% females while amongst the obese students 25% were males and 75% were females. The association between gender and weight is highly significant (p <0.0001). Out of the prehypertensive students 94.44% were obese while all (100%) hypertensives were obese. Thus there exists a significant correlation between obesity and prehypertension with value of p=0.0042.

Table 2: Gender Wise Weight Distribution

	Males (% of Total Students)	Females (% of Total Students)	Total (% of Total Students)
Underweight	12 (4.5%)	53 (20%)	65 (24.5%)
Normal Weight	74 (27.9%)	97 (36.6%)	171 (64.5%)
Preobese	19 (7.16%)	6 (2.27%)	25 (9.43%)
Obese	1 (0.37%)	3 (1.13%)	4 (1.5%)
Total	106	159	265

Maximum prehypertensives students i.e. 55.55% had a heart rate between 80-90/min and out of the hypertensives students 37.01% had heart rate between 80-90/min. 35.19% prehypertensives had a positive family history of hypertension and 12.96% of students had a positive family history of obesity.

Chart 1: Prevalence Of Prehypertension And Hypertension In Males And Females

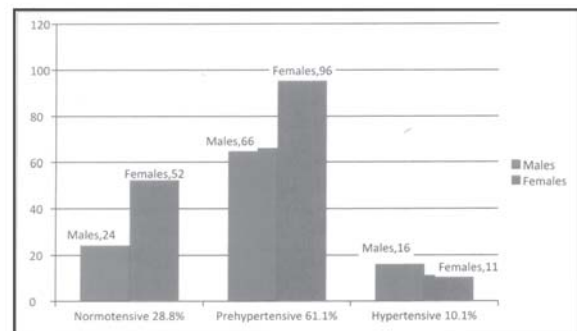


Chart 2: Genderwise Weight Distribution

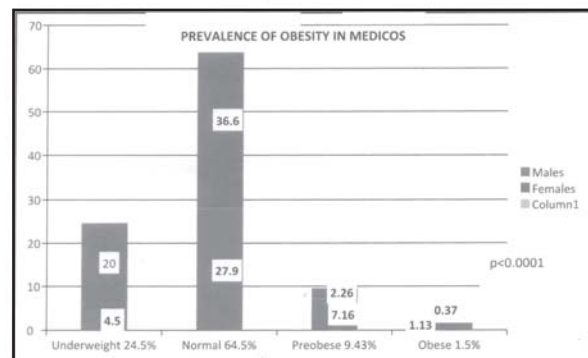


Chart 3: Obesity And Its Corelation With Blood Pressure

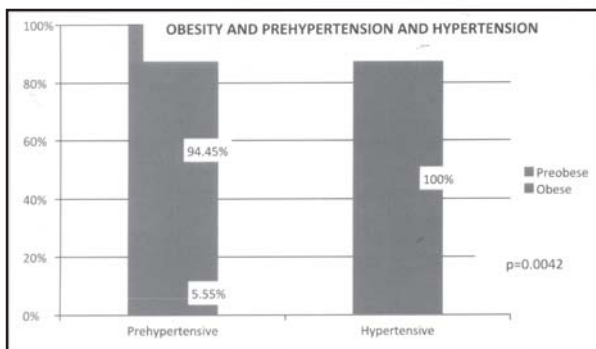
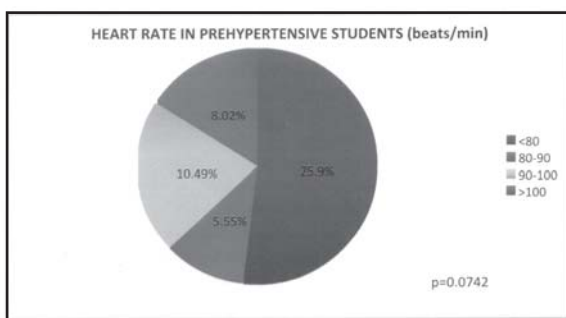


Chart 4: Heart Rate In Prehypertensive Students



DISCUSSION

Hypertension, a known risk factor for cardiovascular accidents⁵ has been well studied across the globe. Prehypertension is a potential modifiable risk factor preceding hypertension. From our analysis nearly 61.1% of our students fell in the prehypertensive range which is more than the general population. The medical students undergo similar or more stress during their training period with little physical activity, altered food habits and recreational social habits like smoking & alcohol which might explain the increased prevalence of prehypertension. All the students belonged to the same socioeconomic strata & consumed the same kind of food. Physicians across the globe are found to have higher prevalence of metabolic syndrome than previously expected. There is an urgent need to estimate the risks among the healthcare professionals.

In the present study, all hypertensives were categorized as obese with a positive correlation with high blood pressure. While among the prehypertensives 94.4% were found to be obese. Influence of gender in hypertension prevalence has been observed among men who showed higher prevalence of both systolic and diastolic hypertension in young age⁷. The present study identifies a subgroup, vulnerable but unnoticed, which needs

immediate attention and efforts to avoid progression to hypertension and possible cardiovascular disease.

In our study group 35.19% had a positive family history of hypertension. This obviously implies that a strong family history is an independent risk factor & those students are prone to develop prehypertension prematurely. Thus tackling them should be our prime priority to prevent or delay future comorbidities. Moreover 12.96% students had a positive family history of obesity. This indicates that not just genetic and familial factors play a role but also the dietary habits, the type of food consumed by the family may also have a role in determining the future impact of lifestyle diseases. Hence healthy dietary habits inculcated right from childhood by the parents would go a long way in preventing and delaying the onset of such lifestyle diseases and most importantly obesity.

It has been estimated that a 5 mmHg reduction in systolic blood pressure in the population will produce a 14% reduction in the risk of stroke and a 9% reduction in the risk of coronary heart disease⁸. The present study stresses the need to target this subgroup of prehypertensives more aggressively to prevent the epidemic of hypertension and its sequelae like coronary artery disease and chronic kidney disease. It has been shown that overweight status and increasing age are potential risks for future development of hypertension thus obesity management and lifestyle modifications are potential factors in the prevention of hypertension⁹ and we stress the need to inculcate this in the early training period among the medical students to keep the health care fraternity in better shape. Prehypertension and hypertension are also significant risk factors for the development of insulin resistance^{11,13} and metabolic syndrome which is quite prevalent in India. Presently pharmacological therapy has not been recommended to treat prehypertension except for those with other comorbid risk factors¹⁰.

The study involves a homogenous group of people with mostly similar backgrounds and socioeconomic parameters. The study has displayed facts based on a single visit, small sample size, lack of follow up data, any comparison in change in values obtained with a change in situation (eg. data collected maybe during exam times) has not been accounted for in this study, and remains a limitation.

CONCLUSION

We conclude that we have an alarming number of prehypertensive even among the medical students, the future healthcare providers. Similarly this subgroup in the community would also be large, which needs to be

identified and informed of the future complications. Prehypertension is potentially modifiable with simple lifestyle modifications. Thus there is need for better health awareness and a healthy lifestyle even among the medical students, along with the society in general.

REFERENCES

1. "The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report". *JAMA* 289 (19): 2560–72
2. King DE, Everett CJ, Mainous AG. Long term prognostic value of resting heart rate in subjects with prehypertension. *Am J Hypertens*. 2006 Aug;19(8):796-800
3. Ishikawa Y, Ishikawa J, Ishikawa S et al. Prehypertension and the risk for Cardiovascular disease in the Japanese general population: The Jichi Medical School Cohort Study. *J Hypertens*. 2010 Aug;28(8):1630-37
4. Elliot WT, Black HR. Prehypertension. *Nat Clin Pract Cardiovasc Med*. 2007 Oct;4(10):538-48
5. National Heart, Lung and Blood Institute (<http://www.nhlbi.nih.gov/hbp>)
6. Qureshi AI, Suri MF, Kirmani JF, Divani AA, Mohammad Y (September 2005). "Is prehypertension a risk factor for cardiovascular diseases?".
7. Mathavan A, Chockalingam A, Chockalingam S, Bilchik B, Saini V. Madurai Area Physicians Cardiovascular Health Evaluation Survey(MAPCHES)- an alarming status. *Can J Cardiol*. 2009 May;25(5):303-8
8. Gonzalez-Ortiz M, Martinez-Abundez E, Hernandez-Salazar E, Robles-Cervantes JA. Obesity and dyslipidemia in primary care physicians. *Acta Cardiol*. 2008 Jun;63(3):331-4
9. Yadav S, Boddula R, Genitta G et al. Prevalence and risk factors of prehypertension and hypertension in an affluent north Indian Population. *Indian J Med Res*. 2008 Dec;128:712-20
10. Strachan MVJ, Walker BR in Nicholas A Boon, Nicki R Colledge, Brian R Walker, John A A Huner editors. *Davidson's Principles and Practice of Medicine*, 20th edition: Elsevier publication; 2006:766
11. Vasan RS, Larson MG, Leip EP et al (November 2001). "Impact of high-normal blood pressure on the risk of cardiovascular disease"
12. American Heart Association (2005, August 6). Prehypertension Triples Heart Attack Risk.
13. Harrison's Principles of Internal Medicine.

Visual Outcome and Major Surgical Complications of Manual Small Incision Cataract Surgery versus Extra capsular Cataract Extraction performed by resident doctors in a Regional Institute of Ophthalmology.

Agrawal Garima*, **Thakker Hansa****, **Audich Kamini*****

Assistant Professor, Professor, Director & Professor, Department of Ophthalmology
M & J Institute of Ophthalmology, B. J. medical College, Civil Hospital, Asarwa, 380016

KEY WORDS : ECCE, MSICS, Major Surgical Complications

ABSTRACT

Introduction: Cataract surgery poses its own learning curve to the resident doctors. Our resident doctors receive training in Extracapsular cataract extraction (ECCE) and Manual small incision cataract surgery (MSICS). We designed a study to compare the visual outcome and major surgical complications of resident performed ECCE versus MSICS at our regional institute of Ophthalmology.

Material and Methods: 203 eyes of 203 patients with visually significant cataract presenting to our OPD were enrolled. Data of consecutive resident cataract surgeries done from December 2014 to February 2015 was analyzed. JR1 was allotted ECCE, JR2 and JR3 were allotted MSICS.

Observations: Both the surgeries in all the three resident groups had good visual outcome. The rate of major surgical complications was 3.23% for JR3 performing MSICS, 12.37% for JR2 performing MSICS and 4.55% for JR1 performing ECCE. The overall major surgical complication rate for MSICS was 8.8% while that for ECCE was 4.55%.

Conclusion: We conclude that both surgeries can be taught to beginner surgeons with good visual outcome. ECCE has a lower learning curve as evidenced by a low rate of major surgical complications. MSICS is technically more demanding with a higher rate of complications by the beginner JR2. Once expertise is achieved in MSICS the complication rate again drops in JR3 performed surgeries.

INTRODUCTION

Cataract surgery presents its own learning curve to the resident doctors. At our center residents are trained in Extracapsular cataract extraction (ECCE) and Manual small incision cataract surgery (MSICS). We designed a study to compare the major surgical complications and visual outcome of MSICS versus ECCE done by resident surgeons at our center.

AIM

To study the visual outcome and major surgical complications of Manual Small Incision Cataract Surgery (MSICS) versus Extracapsular Cataract Extraction (ECCE) performed by resident doctors in a Regional Institute of Ophthalmology.

MATERIAL AND METHODS

203 eyes of 203 patients with visually significant cataract presenting to our OPD were enrolled. The study was carried out at M&J Western Regional Institute of Ophthalmology, B J Medical College, Ahmedabad.

Informed consent for cataract surgery was taken from every patient. Data of consecutive resident cataract surgeries done from December 2014 to February 2015 was analyzed. The preoperative, operative and postoperative data was recorded. The patients were done by Junior resident (JR) 1/2/3 under the guidance of one Assistant professor. Preoperatively complete anterior segment and posterior segment examination, IOL powering, Intraocular pressure measurement (IOP measurement) were carried out. Blood pressure was measured and routine blood/urine investigations were done. Patients with uncontrolled diabetes, uncontrolled hypertension and active infection anywhere in the body were excluded from the study. High risk patients with zonular weakness, posterior polar cataracts, corneal dystrophy/pathology, uniocular patients, post-uveitic cataracts, pseudoexfoliation, patients younger than 35 years, traumatic cataracts were excluded from the study. JR1 was asked to observe at least 30 cases of SICS/ECCE and be first assistant to 30 cases. Thereafter JR1 was assigned an ECCE. JR2 and JR3 were assigned MSICS. Phacoemulsification surgeries were

Correspondence Address : **Dr. Garima Agrawal,**
C-101, Gala Aura, South Bopal, Ahmedabad, 380058.

assigned to post MS fourth year residents and have not been included in our study. The surgeries were done under local anesthesia under the guidance of one Assistant Professor. The intraoperative complications (including the operative step involved) were documented. Post-operatively visual acuity and complete anterior and posterior segment examination were documented. The postoperative complications were documented. Major surgical complications were defined as 1) Posterior capsular rupture or zonulodialysis with vitreous loss with successful IOL implantation. 2) Suprachoroidal hemorrhage. 3) New Vitreous hemorrhage (VH)/ retinal detachment (RD)/ Endophthalmitis within 90 days of surgery. 4) Any complication requiring reoperation within 90 days of surgery including a) Vitreous loss with inability to implant IOL. b) Nucleus drop. c) IOL drop. d) Others. Statistical analysis was performed using the chi square test. $p < 0.05$ was taken as significant.

OBSERVATIONS AND RESULTS

During the 3 month period 203 consecutive patients had cataract surgery done by residents at M&J Western Regional Institute of Ophthalmology. Table I shows the distribution of cases among the surgeon groups. 21.68% of patients were done by JR1, 47.78% by JR2 and 30.54% by JR3. Greater number of surgeries were done by JR2 reflecting the greater number of JR2 residents (MS and diploma) as compared to JR3 (only MS students). 21.68% Extracapsular cataract extractions were performed and 78.32% MSICS were done. Table II shows that 8.81% of MSICS cases and 4.55% of ECCE patients had major surgical complications. P value is > 0.05 thus the surgical technique in our study did not significantly appear to affect the major surgical complication rate (percentage). Table III shows the rate of major surgical complications as per the surgeon group. The rate of major surgical complications was 3.23% for JR3, 12.37% for JR2 and 4.55% for JR1. JR2 had a statistically significant higher major surgical complication rate as compared to JR3. Table IV shows the factors associated with major surgical complications including age, gender, nuclear sclerosis grade, surgeon and type of surgery. P value is not significant for age, gender, type of cataract and type of surgery comparisons. P value for JR1 versus JR3 is not significant while that of JR2 versus JR3 is significant. Table V shows the comparison of major surgical complications in MSICS and ECCE patients. The rate of vitreous loss for MSICS surgeries was 8.17% while that of ECCE surgeries was 4.55%. The major surgical complication rate for MSICS was 8.8% while that for ECCE was 4.55%. Table VI shows the surgical step at which vitreous loss was documented for the surgeries. Overall 53% were noted during irrigation/aspiration or

cortex removal. It is noted that JR2 group had 45% vitreous loss at nucleus extraction step. Table VII shows the pre-operative and post-operative best corrected visual acuity (BCVA) in MSICS and ECCE groups. Overall 95% patients in JR3 group (MSICS) and 86% patients in JR2 group (MSICS) had BCVA $\geq 6/6$ to 6/12. 80% patients in JR1 group had BCVA $> 6/12$.

DISCUSSION

Cataract surgery has its own learning curve. At our center ECCE and MSICS were done by resident doctors with good visual results.

The rate of major surgical complications was documented. ECCE done by JR1 had a low rate of complications (4.55%) as compared to MSICS done by JR2 (12.37%). ECCE is an initial simpler surgery with a lower learning curve as evidenced by a lower complication rate despite being done by residents with no prior surgical experience. MSICS is a technically advanced surgery and requires more training and supervision in the learning stage. Once expertise is achieved in MSICS as in JR3 complication rate again drops (3.23%).

Majority of the complications were noted in the irrigation aspiration stage in ECCE done by JR1 thus this step requires careful supervision. MSICS done by JR2 had a high complication rate at the nucleus extraction step and irrigation aspiration step. These steps require training. JR3 had complications at the irrigation aspiration step.

Age, gender, grade of nucleus and type of surgery did not appear to significantly affect the complication rate. The complication rate is affected by the surgeon type being significantly higher in JR2 (compared to JR3) reflecting their learning curve of MSICS.

To the best of our knowledge this is the first study in western India that compares the outcomes of ECCE versus MSICS in beginner (resident) surgeons.

We have come across only one other study comparing MSICS versus ECCE in resident surgeries. Hari Priya et al report four surgeon groups i.e. staff, fellows, residents, visiting trainees. Surgical distribution was 26% phacoemulsification, 67% MSICS and 7% ECCE. Overall complication rate was 1.11% for phacoemulsification, 1.01% for MSICS and 2.6% for ECCE. The overall intraoperative complication rate was 0.79% for staff, 1.19% for fellows, 2.06% for residents and 5% for visiting trainees. 1

Browning DJ et al reported a 9% vitreous loss and 14% posterior capsular rent in the first 25 cases of the average residents experience with extracapsular cataract extraction. 2

Table I : Distribution of cases among surgeon groups.	
Surgeon	Number of cases n(%)
JR3	62(30.54)
JR2	97(47.78)
JR1	44(21.68)
Total	203(100)

Table II : Rate of Major surgical complications as per the surgical technique used.			
Technique	Surgeries without major surgical complications n (%)	Surgeries with major surgical complications n (%)	Total n (%)
MSICS	145(91.19)	14(8.81)	159(100)
ECCE	42(95.45)	2 (4.55)	44(100)
Total	187 (92.12)	16 (7.88)	203 (100)
P value 0.54 MSICS versus ECCE complication rate			

Table III : Rate of major surgical complications as per the surgeon group.			
Surgeon	Surgeries without major surgical complications n (%)	Surgeries with major surgical complications n (%)	Total n (%)
JR3	60 (96.77)	2 (3.23)	62 (100)
JR2	85 (87.63)	12 (12.37)	97 (100)
Jr1	42 (95.45)	2 (4.55)	44 (100)
p value is 0.04 comparing JR2 versus JR3 and p value is 0.87 comparing JR1 versus JR3 rate of major surgical complications.			

A number of studies reporting visual outcome and complications of phacoemulsification done by residents have been reported in literature.^{3,4,5,6,7,}

Meeks LA et al reported a 2.5% complication rate with phacoemulsification performed by beginner resident primary surgeon and 4.1% complication rate with ECCE.They concluded that phacoemulsification cataract surgery could be taught safely and effectively to residents with no cataract surgery experience as primary surgeon.³

Thomas et al reported the visual outcome and complications of residents learning phacoemulsification.⁴They documented a 10% overall incidence of vitreous loss.A BCVA of 6/12 or better was achieved in 94.8% eyes at 6 weeks follow up.

Unal et al reported resident experience of phacoemulsification with topical anesthesia .They reported a BSCVA of 20/40 or better in 84.9% patients in the topical group. Posterior capsular rupture with vitreous

Table IV : Factors associated with major surgical complications.				
Variable	Cases without major surgical complications	Surgeries with major surgical complications n (%)	Total	P value
Age				
</=60	108	6	114	0.11
>/=60	79	10	89	
Gender				
Male	88	9	97	0.47
Female	99	7	106	
Nuclear sclerosis				
</=NS2+	24	1	25	0.78
Ns3+	72	4	76	
Ns4+	91	11	102	
Surgeon				
Jr3	60	2	62	-
Jr2	85	12	97	0.04
Jr1	42	2	44	0.87
Type of Surgery				
MSICS	145	14	159	0.54
ECCE	42	2	44	

Table V : Comparison of Major Surgical Complications in MSICS and ECCE patients.			
Complication	Both techniques n	MSICS n	ECCE n
Posterior capsular rupture or Zonulodialysis with vitreous loss with succesful IOL implantation	6	5	1
Suprachoroidal Hemorrhage	1	1	-
New VH/RD/Endophthalmitis within 90 days of surgery	-	-	-
Any complication requiring reoperation within 90 days of surgery			
a) Vitreous loss with inability to implant IOL	9	8	1
b) Nucleus drop			
c) IOL drop			
d) Others			
Total	16	14	2

Table VI : Surgical step at which vitreous loss was documented for the surgeries.				
Number(percentage)				
	MSICS		ECCE	
Surgical Step	JR3	JR2	JR1	Overall
Nucleus prolapse	-	2(18.18)	-	2(13.33)
Nucleus extraction	-	5(45.45)	-	5(33.33)
Cortex Removal I/A	2(100)	4(36.36)	2(100)	8(53.33)
IOL Implantation	-	-	-	
Other steps	-	-	-	
Total	2(13.33)	11(13.33)	2(13.33)	15(100)
I/A=Irrigation/Aspiration				

Table VII : BCVA in MSICS and ECCE Group n(%)						
MSICS			ECCE			
JR3	JR2	JR1				
BCVA	Pre operative	Post operative	Pre operative	Post operative	Pre operative	Postoperative
6/6-6/12	-	59(95.16)	-	83(85.57)	-	35(79.55)
<6/12-6/60	11(17.74)	2 (3.22)	32(32.99)	9 (9.28)	5(11.36)	7(15.90)
<6/60	51(82.26)	1 (1.61)	65(67.01)	5(5.14)	39(88.64)	2(4.55)
Total	62(30.54)	62(30.54)	97(47.78)	97(47.78)	44(21.68)	44(21.68)

loss was seen in 6.1% eyes and loss of lens fragments was documented in 2.7% eyes in the topical group. The rate of postoperative complications was 15% in the topical group.⁵

Randleman JB et al reported the resident surgeon phacoemulsification learning curve .BCVA was 20/40 or better in 97.8% cases.Inraoperative complications were seen in 5% cases with a significant reduction in vitreous loss rates after first 80 resident cases (5.1% versus 1.9% p=0.03).⁶

Randleman JB et al reported phacoemulsification with topical anesthesia performed by resident surgeons.Vitreous loss was seen in 4.1% cases.Postoperative complication rate was 9.9%.86.6% cases achieved a BCVA of 20/40 or better.⁷

Tarbet KJ et al reported 6.3% overall rate of surgical complications in in phacomulsification performed by residents.90.6% of all eyes had BCVA 20/40 or better.⁸

Cruz OA et al reported a 9.9% incidence of PCR and BCVA 20/40 or better in 92.6% eyes of resident performed phacoemulsification.⁹

Allison RJ et al reported a 14.7% incidence of vitreous loss in cases of third year residents performing phacoemulsification.¹⁰

Badoza et al reported a mean postoperative 1 month BCVA of 0.19+/-0.19.They documented a low incidence of vitreous loss of 2.8% in resident performed phacoemulsification cases.¹¹

Bhagat N et al reported a 6.7% rate of posterior capsular disruption in resident performed phacoemulsification cases.¹²

Biomquist PH et al reported that 91% of patients had a BCVA of 20/40 or better (after excluding pre existing ocular pathology patients).13

Corey RP et al reported rate of posterior capsular rupture with vitreous loss in resident early cases to decrease to no cases of PCR in late surgeries of resident performed phacoemulsification cases.14

Quillen DA et al reported post operative BCVA of 20/40 or better in 89% eyes and vitreous loss in 4.8% cases of resident performed phacoemulsification without prior planned ECCE experience.15

Rutar T et al reported that mature nuclei and zonular pathology carried increased intraoperative risk in resident performed phacoemulsification cases.16

Our complication rates and visual outcome are comparable to the above studies.

CONCLUSION

We conclude that ECCE and MSICS can be taught to resident doctors with good visual results and a comparable rate of major surgical complications..ECCE is an initial simpler surgery with a lower learning curve as evidenced by a lesser complication rate despite being done by JR1 with no previous surgical experience. MSICS on the other hand is a technically demanding surgery requiring more training and supervision in the learning stage as evidenced by a higher complication rate in JR2. Once expertise is achieved as in JR3 the complication rate again drops.

REFERENCES

1. HariPriyaA, ChangDF, ReenaM, Shekhar M. Complication rates of phacoemulsification and manual small incision cataract surgery at Aravind Eye Hospital. J Cataract Refract Surg 2012;38(8):1360-1369.
2. BrowningDJ, CoboLM. Early experience in extracapsular cataract surgery by residents. Ophthalmology 1985;92:1647-1653.
3. Meeks LA ,BlomquistPH, SullivanBR. Outcomes of manual extracapsular versus phacoemulsification cataract extraction by beginner resident surgeons. J Cataract Refract Surg 2013;39(11):1698-1701.
4. Thomas R, NaveenS, JacobA, BraganzaA. Visual Outcome and complications of residents learning phacoemulsification. Indian J Ophthalmol 1987;45:215-219.
5. UnalM, YuceII, Sarici, ArtunayO, DevranoqluK, AkarY, AltinM. Phacoemulsification with topical anesthesia: resident experience. J Cataract Refract Surg 2006;32:1361-1365.
6. Randleman JB, WolfeJD, Woodward M ,Lynn MJ, CherwekDH, SrivastavaSK. The resident surgeon learning curve. Arch Ophthalmol 2007;125:1215-1219.
7. RandlemanJB, Srivastava SK, Aaron MM. Phacoemulsification with topical anesthesia performed by resident surgeons. J Cataract Refract Surg 2004;30:149-154.
8. Tarbet KJ, MamalisN, TheurerJ, JonesBD, Olson RJ. Complications and results of phacoemulsification performed by residents. J Cataract Refract Surg 1995;21:661-5.
9. Cruz OA, WallaceGW, GayCA, MatobaAY, KochDD. Visual results and complications of Phacoemulsification with intraocular lens implantation performed by ophthalmology residents. Ophthalmology 1992;99:448-52.
10. Allison RW, MetrikinDC, FanteRG. Incidence of vitreous loss among third year residents performing phacoemulsification. Ophthalmology 1992;99:726-30.
11. Badoza DA, JureT, Zunino LA ,ArgentoCJ. State-of-the-art phacoemulsification performed by residents in Buenos Aires. Argentina. J Cataract Refract Surg 1999;25:1651-5.
12. Bhagat N, NissiriosN, Potderin L et al. Complications in resident performed phacoemulsification cataract surgery at New Jersey Medical School. Br J Ophthalmol 2007;91:1315-17.
13. BiomquistPH, RugwaniRM. Visual outcomes after vitreous loss during cataract surgery performed by residents. J Cataract Refract Surg 2002;28:847-52.
14. CoreyRP, OlsonRJ. Surgical outcomes of cataract extractions performed by residents using phacoemulsification. J Cataract Refract Surg 1998;24:66-72.
15. Quillen DA, PhippsSJ. Visual outcomes and incidence of vitreous loss for residents performing phacoemulsification without prior planned extracapsular cataract surgery experience. Am J Ophthalmol 2003;135:732-3.
16. RutarT, PorcoTC, NaseriA. Risk factors for intraoperative complications in resident performed phacoemulsification surgery. Ophthalmology 2009;116:431-6.

Diagnostic Role of Bone Marrow Aspiration and Trepine Biopsy in Haematological Practice

Dr. Shilpa Patel*, Dr. Pooja Nathani**, Dr. Nailesh Shah***, Dr. C.K. Shah****

* Junior Lecturer, **1st Year Resident Doctor, ***Professor and H.O.D, ****

Professor Department of Pathology, Sheth V.S.G.Hospital, N.H.L Municipal Medical College, Ahmedabad-66

KEY WORDS : Bone marrow aspirate, Trepine Biopsy, Diagnostic role.

ABSTRACT

Aims and Objectives:- To determine, evaluate and correlate the diagnostic role of bone marrow aspiration and trephine biopsy in routine hematological practices.

Material and Methods:- A study of 120 cases of bone marrow examination, was conducted at the Department of Pathology, N.H.L Municipal Medical College, Ahmedabad from January 2013 to December 2014. Patients were reviewed irrespective of their age and sex. In each case a detailed history along with systemic, general and local examination and routine hematological investigation was carried out prior. All the bone marrow smears and biopsy sections were then examined in detail. The diagnosis and findings of both the aspirate and biopsy were evaluated and compared with each other.

Results:- Out of those 120 cases, in 110 cases both bone marrow aspiration and biopsy was performed and in the remaining 10 cases only bone marrow aspiration was done. Out of those 110 cases, 96 (87.3%) cases showed a close diagnostic correlation between both aspiration and biopsy. On the other hand, 12 (10.9%) cases of dry tap were recorded as well as two (1.8%) cases were recorded which showed absolutely no correlation between the diagnostic interpretation of both aspiration and trephine. One of these cases was that of a tuberculous granuloma while the other one was that of a lymphoma.

Conclusion:- The results suggest that both aspiration and biopsy are valuable and complementary to each other. Bone marrow aspirate smears are ideal for the study of cytological details of haematopoietic cells. Trephine biopsies are helpful for the assessment of cellularity, extent and pattern of tumor infiltration and cell type. Nutritional anaemias, haematological malignancies and immune thrombocytopenia can be readily diagnosed by bone marrow aspiration alone. On the other hand, trephine biopsy is necessary for diagnosing granulomatous inflammations, hypoplastic or aplastic anaemias, myelofibrosis and lymphomatous infiltrations.

INTRODUCTION

Bone marrow examination is a useful and cost effective diagnostic procedure in haematological practice for the diagnosis of both neoplastic and non-neoplastic haematological diseases. These procedures are also employed for typing of anaemias, evaluation of cytopenias and pyrexia of unknown origin. The bone marrow examination may either confirm the clinically suspected disease or may provide the previously unsuspected diagnosis.¹⁻⁴ Bone marrow aspiration alone is usually sufficient to diagnose nutritional anaemias, most of the acute leukaemias and Immune Thrombocytopenias because of ease of the application of pearl iron stain and other cytochemical stains on aspirate smears but it does not provide important diagnostic information in patients with granulomatous disease, myelofibrosis and bone marrow infiltration.⁵ These procedures are also valuable for a regular follow up of patients undergoing

chemotherapy, bone marrow transplantation and other forms of medical treatment. Involvement of marrow by metastatic tumour, may have an effect on clinical treatment and prognosis. Similarly involvement of the marrow by granulomatous lesion especially tuberculous granulomas may be easily identified in bone marrow biopsies. For bone marrow examination two types of samples – BMA (Bone marrow aspiration) and BMB (Bone marrow biopsy) can be obtained. Marrow biopsy by surgical trephine is an older procedure than needle aspiration. It is only since the late 1950s that core needle biopsy of the bone marrow has been used⁶ but bone marrow aspiration is a simple and minimally invasive technique. However bone marrow aspiration has lower sensitivity in detecting solid tumour metastasis and lymphoma involvement in comparison to Bone marrow biopsy. Bone marrow biopsy is a more reliable method for detecting marrow infiltrate, the pattern of marrow involvement and the cellularity of marrow.

Correspondence Address : Dr. Shilpa Patel,

Department of Pathology, N.H.L Municipal Medical College, V.S. Hospital, Ahmedabad
Email id – shilpapatel220@yahoo.in

When both the procedures are performed simultaneously, they are complementary to each other and there is more material to study the morphology and the pattern of distribution of the cells. This study was conducted in our institute to evaluate the complementary role of both the procedures done simultaneously and to see the advantages and disadvantages of these procedures. Nowadays aspirate and trephine biopsy specimens are considered complementary and when both are obtained, they provide a comprehensive study of bone marrow.

AIMS AND OBJECTIVES

To determine, evaluate and correlate the diagnostic role of bone marrow aspiration and trephine biopsy in routine hematological practices.

MATERIALS AND METHODS

A study of 120 cases of bone marrow examination was conducted at the Department of Pathology, Sheth V.S. General Hospital, N.H.L Municipal Medical College, Ahmedabad from January 2013 to December 2014.

Patients were reviewed irrespective of their age and sex. In each case a detailed history along with systemic, general and local examination and routine hematological investigation was carried out prior to the bone marrow trephine biopsy. All the bone marrow smears and biopsy sections were then examined in detail. The diagnosis and findings of both the aspirate and biopsy were evaluated and compared with each other.

For preparing the Bone marrow aspiration the standard technique of using Salah's needle was applied for collection. About 0.25-0.5 ml of aspirate was obtained from the posterior superior iliac spine into a syringe and delivered onto a clean glass slide. After the smears were made the slides were air dried and were stained subsequently with (Romanowsky) leishman stain.

Trephine biopsies were performed using the Jamshidi needle with length of biopsy ranging from 1 to 2 cm. The biopsies were fixed in Bouins fluid, decalcified for 48 hours and embedded in paraffin from which 4 μ thin sections were made and stained with hematoxylin and eosin (H&E) stain. Other special stains like reticulin, periodic acid-Schiff (PAS) and Perls were done, wherever indicated. Bone marrow aspiration was labeled as unsatisfactory when marrow particles were absent or there was dilution of samples with peripheral blood. Trephine biopsies were considered inadequate when there were <3 intertrabecular spaces or when there was inadequate/total absence of hematopoietic elements.

OBSERVATION AND RESULTS

Out of 120 cases, in 110 cases both bone marrow aspiration and biopsy was performed and in the remaining 10 cases only bone marrow aspiration was done. Out of

those 110 cases, 96 (87.3%) cases showed a close diagnostic correlation between both aspiration and biopsy. On the other hand, 12 (10.9%) cases of dry tap were recorded as well as two (1.8%) cases were recorded which showed absolutely no correlation between the diagnostic interpretation of both aspiration and trephine. The age of the subjects ranged from three years to seventy years with a male predominance (2.4:1).

The indications for performing a bone marrow biopsy are anaemia, pancytopenia, hepatosplenomegaly, pyrexia of unknown origin and petechia etc.

Table I: Various Indications of Bone Marrow Biopsy and The No. of Cases Recorded.

Indications	No. of Cases	% of Cases
Anaemia	53	44.2
Pancytopenia	30	25.0
Petechia	10	8.3
Pyrexia Of Unknown Origin	10	8.3
Hepatosplenomegaly	05	4.2
Miscellaneous	12	10.0
Total	120	100

Table I shows that maximum no. of cases were recorded of anaemia (44.2%) and pancytopenia (25%) being the clinical indication for performing a bone marrow examination.

The miscellaneous indications included sternal tenderness, bleeding manifestations as in gum bleeding and lymphadenopathy.

Figure I. Indications for Bone Marrow Biopsy (N =120).

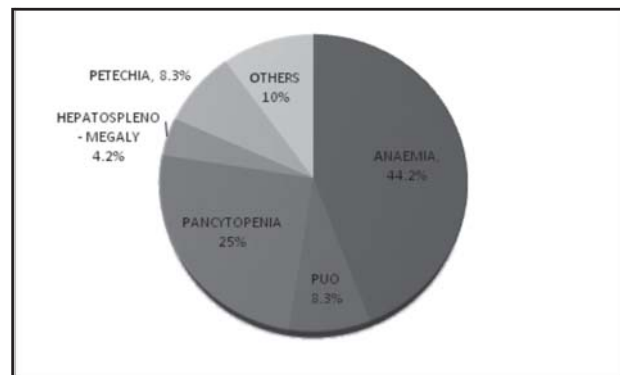


Figure I shows the commonest indication for a bone marrow examination was anemia (44.2%) followed by and pancytopenia (25%).

Figure II: Diagnosis of Bone Marrow Examination in Different Cases

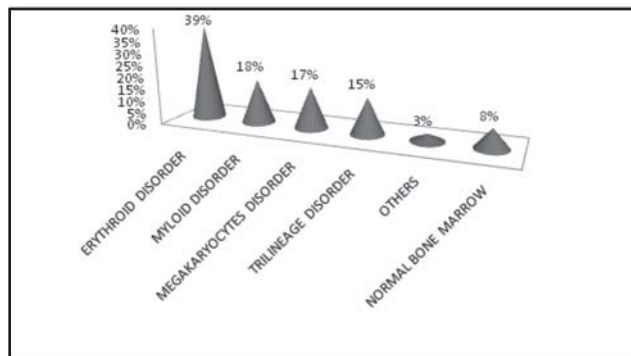


Figure II shows that disorders related to erythroid series were present in 39% of cases. In 18%, myeloid disorders, in 17% megakaryocyte disorders and 15% diseases affecting trilineage cells were diagnosed. 8% cases revealed normal marrow findings.

Table II : Diagnosis of Bone Marrow Examination in Different Cases.(N-120).

Disorders	Bonemarrow Aspiration	% of Cases
Megaloblastic Anaemia	16	13.3
Iron Deficiency Anaemia	06	5.0
Dimorphic Anaemia	08	6.7
Erythroid Hyperplasia	10	8.3
Pure Red Cell Aplasia	02	1.7
Congenital Dyserythropoietic Anaemia	01	0.8
Aplastic Anaemia	04	3.3
Myeloid Hyperplasia	06	5.0
Myelodysplastic Syndrome	03	2.5
Mylofibrosis	02	1.7
Acute Leukaemia	07	5.8
Muliple Myeloma	03	2.5
Essential Thrombocythemia	03	2.5
Immune Thrombocytopenic Purpura	11	9.2
Decreased Megakaryocytes	06	5.0
Hypercellular Marrow	08	6.7
Hypocellular Marrow	10	8.3
Granulomatous Disease	01	0.8
Lymphoma	02	1.7
Haemophagocytosis (Drug Induced)	01	0.8
Normal Marrow	10	8.3
Total Cases	120	100

12 cases of dry tap and 2 cases of inadequate biopsy material were recorded.

Table II reflects that megaloblastic anaemia (13.3%) was the commonest finding followed by immune thrombocytopenic purpura (9.1%) and hypocellular marrow (8.3%) among all the cases. In case of acute leukemia peripheral smear examination was in conclusive but bone marrow aspiration and biopsy showed > 80% blast cells. In cases of myelofibrosis, reticulin stain was also performed. One case of Myelodysplastic syndrome diagnosed as 5q- was later confirmed at the Gujarat Cancer & Research Institute, Ahmedabad.

Table Iii: Bone Marrow Trepine Biopsy Diagnosed Cases in Dry Taps on Bone Marrow Aspiration (N-12).

Sr No.	Diagnosis	No of Cases	% of Cases
1	Myelofibrosis	2	16.7
2	Lymphoma	1	8.3
3	Acute Leukemia	1	8.3
4	Granulomatous Lesion	1	8.3
5	Hypocellular Marrow & Aplastic Anaemia	6	50
6	Myelodysplastic Syndrome	1	8.3

As shown in Table III in 12 patients, (10%) aspiration was dry so no diagnosis was given.

Out of Total 120 cases, in 110 cases both aspiration and biopsy was performed. In two cases diagnosis was different. On aspiration, megaloblastic anaemia was diagnosed and trephine biopsy was suggestive of lymphoma. Another case of granuloma on trephine biopsy was diagnosed as reactive changes on aspiration. In 2 cases (1.7%) biopsy material was insufficient to report. One case of Myelodysplastic syndrome was diagnosed as 5q- syndrome on both aspiration as well as on biopsy material and later it was confirmed in cancer hospital. In suspected cases of Immune thrombocytopenic purpura, a 100% correlation between bone marrow aspiration and trephine was recorded.

In the present study following criteria were used to define pancytopenia.

Criteria for pancytopenia :

Parameters	Value
Hemoglobin	<10.0 gm/dl
Total WBC count	<3500/ul
Platelet count	<1,00,000/ul

Table IV: Cases Presenting as Pancytopenia

Sr No.	Diagnosis	No of Cases	% of Cases
1	Megaloblastic Anaemia	10	29.4
2	Dimorphic Anaemia	1	2.9
3	Erythroid Hyperplasia With Trilineage Haematopoeisis	5	14.7
4	Congenital Dyserythropoietic Anaemia	1	2.9
5	Aplastic Anaemia	4	11.8
6	Myeloid Hyperplasia (Reactive)	1	2.9
7	Myelofibrosis	1	2.9
8	Myelodysplastic Syndrome	1	2.9
9	Decreased Megakaryocytes	1	2.9
10	Lymphoma	1	2.9
11	Hypocellular Marrow	8	23.5
	Total	34	34

As shown in Table IV, in 34 (28.33%) cases out of 120, pancytopenia was present. The most common finding in a patient with pancytopenia was Megaloblastic anaemia (29.4%) and Hypocellular Marrow (23.5%).

DISCUSSION

Bone marrow examination is an important investigation carried out in the routine practice for the diagnosis of various haematological and non haematological disorders. It is a well known fact that bone marrow

aspiration and bone marrow biopsy complement each other. Nowadays both specimens are routinely obtained at the same time and usually same site.

In our study, we did a comparative evaluation of all such bone marrow aspiration and bone marrow biopsy cases, to see the complementary role of both the procedures, to study the advantages and disadvantages of both the procedures done simultaneously.

There was 85.45% positive correlation between bone marrow aspiration and bone marrow biopsy in our study. The cases that were diagnosed as reactive marrow and erythroid hyperplasia showed the highest positive correlation. Those cases which were clinically sent for evaluation of anaemia showing erythroid hyperplasia with either macronormoblastic or micronormoblastic proliferation were further worked up. Perl's stain was done and the biochemical parameters were taken into consideration. The diagnosis was given either as iron deficiency anaemia or megaloblastic anaemia.

The commonest haematological disorder in the present study was anaemia with 47 cases (39.16%) belonging to this subset. Megaloblastic anaemia was the commonest anaemia diagnosed on bone marrow examination (13.33%). In the present study, 55% cases of hypoplastic anaemia were diagnosed on bone marrow aspiration while additional 45% were diagnosed on bone marrow biopsy. In 6 cases aspiration was limited either by dry tap or dilution by peripheral blood. In suspected cases of immune thrombocytopenic purpura, 100% correlation between bone marrow aspiration and trephine was done. More than 90% concordance was noted in case of hypercellular marrow.

Table IV: A Comparison with Various Other Studies of Pancytopenia

Sr No	Study	Year	No of Cases	Commonest Cause of Pancytopenia
1	International Agranulocytosis and Aplastic Anaemia Study ⁷	1987	389	Aplastic Anaemia (52.7%)
2	Keisu M ⁸	1990	100	Neoplastic Disease Radiation (32%)
3	Hossain Et Al ⁹	1992	50	Aplastic Anaemia
4	Verma Et Al ¹⁰	1992	202	Aplastic Anaemia (40%)
5	Tilak Et Al ¹¹	1999	77	Megaloblastic Anaemia (68%)
6	Khodke Et Al ¹²	2001	50	Megaloblastic Anaemia (44%)
7	Sandip Shah		20	Megaloblastic Anaemia
8	Present Study	2014	34	Megaloblastic Anaemia

In comparison to various other studies conducted, our study statistics showed a close correlation to the studies done by Tilak et al, Khodke et al and Dr. Sandip Shah which highlighted that megaloblastic anaemia was the most common cause of pancytopenia.

In our study the second most common cause of pancytopenia was aplastic anaemia with hypocellular marrow.

On the other hand studies done by Hossain et al, Verma et al and the international agranulocytosis and aplastic anaemia study, where 50,20, 389 cases were recorded showed that aplastic anaemia was most common cause. The study done by Keisu M reflected neoplastic radiation disease as the most common cause.

SUMMARY

Male: Female ratio was 2.4:1 with male predominance. The youngest patient was 3 years and the oldest was 70 years of age.

In 12 cases bone marrow aspiration was a dry tap and in 2 cases the diagnosis given on aspiration and biopsy showed no correlation.

Megaloblastic anaemia (13.3%) was the commonest finding followed by ITP (9.1%) and hypocellular marrow (8.3%) among all the cases

Out of 120 cases, 34 cases showed pancytopenia. The most common finding in a patient with pancytopenia was Megaloblastic anaemia (29.4%) and Hypocellular Marrow (23.5%).

There was 85.45% positive correlation between Bone marrow aspiration and Bone marrow biopsy.

CONCLUSION

Bone marrow aspiration is better in making out individual cell morphology whereas biopsy is useful in study of bone marrow architectural pattern and distribution. Morphology of megaloblasts are best seen in bone marrow aspiration but bone marrow biopsy is useful in suspected cases so as to rule out non-megaloblastic macrocytic anaemia. Valuable information on the status of hematopoiesis and changes in stromal components is also provided by trephine biopsy specimens. Trephine biopsy is necessary for diagnosing Granulomatous Inflammation, Hypoplastic/Aplastic Anaemia, Myelofibrosis and Lymphomatous infiltration. Bone marrow biopsy is particularly required for the diagnosis of NHL infiltration, myeloproliferative disorders, aplastic/hypoplastic anaemia, metastatic tumours and granulomatous lesions. Hence both aspiration and biopsy are imperative tools for diagnostic evaluation and treatment of any haematological or non haematological condition.

REFERENCES

1. Bird AR, Jacob P. (1982). Trephine biopsy of the bone marrow. *SAfr Med J*; 64: p 271 – 276.
2. Toi PC, Varghese RG, Rai R. (2010). Comparative evaluation of simultaneous bone marrow aspiration and bone marrow biopsy: an institutional experience. *Indian J Hematol Blood Transfus*; 26: p 41 – 44.
3. Chandra S, Chandra H. (2011). Comparison of bone marrow aspirates cytology, touch imprints cytology and trephine biopsy for bone marrow evaluation. *Hematol Rep*; 3:e22
4. Khan TA, Khan IA, Mahmood K. (2014). Diagnostic role of bone marrow aspiration and trephine biopsy in haematological practice. *J Postgrad Med Inst*; 28(2): p 217 – 221.
5. Winfield DA, Polacz SV. (1992). Bone marrow histology. 3: valve of bone marrow core biopsy in acute leukaemia, myelodysplastic syndromes, and chronic myeloid leukaemia. *J Clin Pathol*; 45: p 855 – 859.
6. Pandya A, Patel T, Shah N. (2012). Comparative utility of bone marrow aspiration and bone marrow biopsy. *Journal of Evolution of Medical and Dental Sciences*. Volume (1):6: p 987 – 993.
7. International agranulocytosis and aplastic anaemia study. Incidence of Aplastic anaemia, the relevance of diagnostic criteria. *Blood* (1987); 70: p 1718 – 1721.
8. Keisu M, Ost A. (1990). Diagnosis in patients with severe pancytopenia suspected of having aplastic anaemia. *Eur J Haematol*; 45: p 11 – 14.
9. Hossain MA, Akond AK, Chowdhary MK et al. (1992). Pancytopenia – a study of 50 cases. *Bangladesh Journal of Pathology*; 1: p 9 – 12.
10. Verma N Dash S. (1992). Reappraisal of underlying pathology in adult patients presenting with pancytopenia. *Trop Geogr Med*; 44: p 322 – 327.
11. Tilak N, Jain R. (1999). Pancytopenia – a clinical haematological analysis of 77 cases. *Indian journal of Pathology and Microbiology*; 42: 4: p 399 – 404.
12. Khodke K, Marwah S, Buxi S et al. (2001). Bone marrow examination in cases of pancytopenia. *Indian Academy of Clinical Medicine journal*; 2: p 55 – 59.

Study of Acute Kidney injury in H1N1 Patients (Year2015)**Dr. Jagdish Patel ***, **Dr. Abhijit Khadtare ****, **Dr. Ishwar Parmar****, **Dr. Arjun Kelaiya****, **Dr. Chintan Prajapati******AP in medicine, **2nd year Resident, **2nd year Resident, **2nd year Resident, ***3rd year Resident
Medicine Department, B.J.Medical College and Civil Hospital Ahmedabad**KEY WORDS** : Acute Kidney injury in H1N1**ABSTRACT**

We have done study of acute kidney injury in H1N1 positive patients in 2015 epidemic. Total number of patients admitted with H1N1 positive influenza were 1071 uptill (jan-april 2015), out of which 192 (positive) patients were expired. We had done study of 208 patients having AKI, out of which majority patients were Females 114(57%) and males are about 94 (43%). Patients were of age range from 14 to 75 years. Among 208 patients of AKI, 122 patients had AKI due to Sepsis, 50 patients had AKI due to Pre Renal causes, 20 patients had AKI which was drug (Vancomycin ect) induced and 16 patients had AKI due to secondary bacterial infection. So prevalence of AKI in total number (1071) of patients admitted with H1N1 is 19.41%, and out of total expired (192 H1N1 positive patient) 46.73% patient having main cause of death is acute kidney injury. Mortality was associated with multiple organ failure, sepsis, shock, prolonged ventilator requirement, ICU stay and acute kidney injury.

INTRODUCTION

Influenza viruses are members of the Orthomyxoviridae family, of which influenza A, B, and C viruses constitute three separate genera. Cases of novel influenza A (H1N1) virus infection have resulted in rapidly progressive lower respiratory tract disease resulting in respiratory failure, development of acute respiratory distress syndrome (ARDS), and prolonged intensive care unit (ICU) admission. There have been a few studies on acute kidney injury (AKI) in patients infected with the pandemic influenza A (H1N1) virus. The objective of this study was to assess the occurrence of acute kidney injury among the critically ill patients with H1N1 infection.

MATERIALS AND METHODS

All patients with H1N1 admitted in the Medical Intensive Care unit at civil hospital, ahmedabad between January 2015 to April 2015, where the diagnosis of H1N1 was confirmed by RT-PCR in samples from throat swab were included in this observational study. The data were noted in a standardized form, the hematological and biochemical reports, treatment and outcome of these critically ill patients with H1N1 infection were studied. The occurrence of acute kidney injury (AKI) in these patients was studied. Descriptive statistics like percentage distribution and mean, standard deviation for the clinical

variables was done. Nasopharyngeal-swab specimens were collected at admission. Specimens were placed in transport medium and kept at a temperature from 2 to 4°C. RT-PCR testing was done in accordance with published guidelines from the U.S. Center for Disease Control and Prevention (CDC).

DISCUSSION

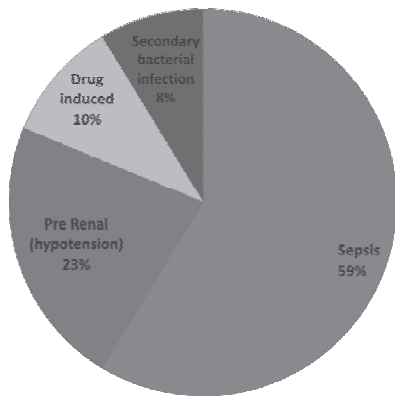
Influenza viruses are members of the Orthomyxoviridae family, of which influenza A, B, and C viruses constitute three separate genera. The designation of influenza viruses as type A, B, or C is based on antigenic characteristics of the nucleoprotein (NP) and matrix (M) protein antigens. Influenza A viruses are further subdivided (subtyped) on the basis of the surface hemagglutinin (H) and neuraminidase (N) antigens; individual strains are designated according to the site of origin, isolate number, year of isolation, and subtype. A spectrum of disease ranging from non-febrile, mild upper respiratory tract illness to severe or fatal pneumonia has been described¹. The most commonly reported symptoms have included cough, fever, sore throat, malaise and headache. Some cases have experienced gastrointestinal symptoms (nausea, vomiting and/or diarrhoea). Clinical diagnosis (based on the acute onset of fever and cough) can be increasingly predictive of the new influenza A (H1N1) virus infection as the prevalence of infections increases⁴. Commercially available rapid tests for seasonal influenza have uncertain sensitivity and lack specificity for detection of

Correspondence Address : **Dr. Jagdish Patel,**
20, Shayona Prajad, Shayona City RCTI Road, Ghatlodia,
Ahmedabad-380063.

Table 1: Study of ACUTE KIDNEY INJURY(AKI) in total admitted number of patients.

Parameters	Indian tertiary referral centre, study in BJMC,CHA 2015
Total patient number(H1N1 positive)	1071
Number of Patients with AKI	208
Sex	Male -94 & Female – 114
Age	14 to 75 years
RRT	44 (21.15%)
Duration of ICU stay	Approximately 20 days
Albuminuria	94 (47%)
Cause of renal failure	Sepsis : 122 (59%)
	Pre renal :50 (23%)
	Drug Induced : 20(10%)
	Secondary bacterial infection : 16(8%)
Death in patients with renal failure	96(48%)
Discharge in patients with renal failure	112(52%)

Causes of Renal Failure in 208 patients



the new influenza A (H1N1) virus. If these tests are performed, both positive and negative results should be interpreted with caution. Samples for laboratory tests should be taken from the deep nasal passages (nasal swab), nasopharynx (naso - pharyngeal swab), throat or, if available, bronchial aspirate. Upper respiratory tract sampling using a combination of a nasal or nasopharyngeal and a throat swab is advised and may facilitate virus detection. It is not yet known which clinical specimen gives the best diagnostic yield for this specific infection. Specimen collection should be done with appropriate precautions since this may expose the collector to respiratory secretions from patients⁴.

Hospitalization or antiviral therapy is not likely to be required for most patients. Supportive care includes antipyretics, such as Paracetamol or Acetaminophen for fever or pain, and rehydration with fluids. Salicylates (such as aspirin and aspirin-containing products) should not be used in children and young adults (aged <18 years) because of the risk of Reye's syndrome The specific risk factors that predict increased risk of progressive disease are incompletely understood. The underlying underlying comorbidities (such as pregnancy, immunocompromised conditions, pre- existing chronic lung disease or cardiovascular disease, diabetes mellitus) are likely to predispose for severe H1N1 infection. Pregnant women are known to be at increased risk of complications from seasonal, avian H1N1 and pandemic influenza infection. Several hospitalizations including fatal outcomes have been reported in pregnant women infected with the new H1N1 virus. Consequently, pregnant women with suspected or confirmed new influenza A (H1N1) infections warrant closer observation and, if in accordance with national policies, treatment with antivirals . A similar study showed that kidney failure was associated with increased death, whereas the need for dialysis was associated with an increase in length of hospital stay. Mortality was mainly associated with multiple organ failure, oligoanuria, acute renal injury and a lack of recovery of renal function. Specific antiviral

therapy available for influenza infection is the neuraminidase inhibitors Zanamivir, Oseltamivir, and Peramivir (for both influenza A and influenza B). A 5-day course of Oseltamivir or Zanamivir reduces the duration of signs and symptoms of uncomplicated influenza by 1 to 1.5 days, if treatment is started within 2 days of the onset of illness. Oseltamivir is administered orally and gives higher systemic level. Zanamivir is delivered by inhalation with low systemic absorption. Oseltamivir is the recommended treatment for lower respiratory tract complications. Rare neuropsychiatric symptoms such as confusion or abnormal behaviour have occurred after beginning treatment for seasonal influenza with Oseltamivir, particularly in children and adolescents. Zanamivir may exacerbate bronchospasm in asthmatic patients. Peramivir, an investigational neuraminidase inhibitor that can be administered intravenously, is being evaluated in clinical trials, as is an intravenous form of Zanamivir¹. Antibiotic chemoprophylaxis should not be used. When pneumonia is present, treatment with antibiotics should generally follow recommendations from published evidence based guidelines for community-acquired pneumonia. Past influenza pandemics have been associated with an increased risk of secondary *Staphylococcus aureus* infections, which may cause, rapidly progressive, necrotizing pneumonia and, in some areas, by methicillin-resistant strains. The results of microbiological studies, wherever possible, should be used to guide antibiotic usage for suspected bacterial coinfection in patients with the new influenza A (H1N1) virus infection. Paracetamol or acetaminophen can be given orally or as suppository. Avoid administration of salicylates (aspirin and aspirin containing products) in children and young adults (< 18 years old) due to risk of Reye's syndrome. If Oxygen therapy is used, monitor oxygen saturation and Sao₂ is to be maintained over 90% (95% for pregnant women) with nasal cannulae or face mask⁴. In our study, all patients received Oseltamivir within 48 hours of presumed diagnosis and duration of treatment was 5 days. Dose modifications were done in severe renal failure. Treatment with Oseltamivir did not cause any worsening of renal functions.

RESULT

Total number of patients admitted with H1N1 positive influenza were 1071 uptill, out of which 192 (positive) patients were expired. We had done study of 208 patients having AKI, out of which majority patients were Females 114(57%) and males are about 94 (43%). Patients were of age range from 14 to 75 years. Out of this 208 patients with Acute kidney Injury 96 patients were expired and 112 patients were discharged. 46 patients had undergone Renal Replacement Therapy Whose creatinine were above 5 mg /dl. Among expired patients 12 patients had already Chronic Kidney Disease and Acute kidney injury

overlapped on it. Among 208 patients of AKI, 122 patients had AKI due to Sepsis, 50 patients had AKI due to Pre Renal causes, 20 patients had AKI which was drug (Vancomycin) induced and 16 patients had AKI due to secondary bacterial infection. So prevalence of AKI in total number (1071) of patients admitted with H1N1 is 19.41%, and out of total expired (192 H1N1 positive patient) 46.73% patient having main cause of death is acute kidney injury.

CONCLUSION

Primary viral pneumonia is the main cause of ICU admission in (H1N1) infected patients, developing severe respiratory failure and it is associated with a relatively high mortality. In the critically ill due to H1N1, renal insufficiency is one of the complications. Sepsis, hypotension, Drug (vancomycin) induced, secondary bacterial infection are major causes for renal failure. Mortality was associated with multiple organ failure, sepsis, shock, prolonged ventilator requirement, ICU stay and acute kidney injury.

REFERENCES

1. Longo, Fauci, Casper, Hauser, Jameson, Lozcalso : Influenza In Harrison's Principles Of Internal Medicine. 18th ed.
2. Perez-Padilla R, de la Rosa-Zamboni D, Ponce de Leon S, Hernandez M, Quiñones-Falconi F, Bautista E, et al. Pneumonia and respiratory failure from swine-origin influenza A (H1N1) in Mexico. *N Engl J Med*.
3. Centre of disease control and prevention
4. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P. Acute Dialysis Quality Initiative workgroup. Acute renal failure-Definition, outcome measures, animal models, fluid therapy and information technology needs: The Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Crit Care*. 2004;8:R204–12.
5. R. L. Mehta, J. A. Kellum, S. V. Shah et al., "Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury," *Critical Care*, vol. 11, article R31, 2007.
6. C. V. Thakar, A. Christianson, R. Freyberg, P. Almenoff, and M. L. Rander, "Incidence and outcomes of acute kidney injury in intensive care units: a Veterans Administration study," *Critical Care Medicine*, vol. 37, no. 9, pp. 2552–2558, 2009. 7)M. Ostermann and R. W. S. Chang, "Acute kidney injury in the intensive care unit according to RIFLE," *Critical Care Medicine*, vol. 35, no. 8, pp. 1837–1843, 2007.
8. H. Gammelager, C. F. Christiansen, M. B. Johansen, et al., "One-year mortality among Danish intensive care with acute kidney injury: a cohort study," *Critical Care*, vol. 16, article R124, 2012.
9. M. Joannidis, B. Metnitz, P. Bauer et al., "Acute kidney injury in critically ill patients classified by AKIN versus RIFLE using the SAPS 3 database," *Intensive Care Medicine*, vol. 35, no. 10, pp. 1692–1702, 2009
10. T. Mandelbaum, D. J. Scott, J. Lee, et al., "Outcome of critically ill patients with acute kidney injury using the Acute Kidney Injury Network criteria," *Critical Care Medicine*, vol. 39, article 2659, 2011.

Grey hair turning to black following Mesenchymal Stem Cell Therapy in patients with neurological disorders: A Report of 4 Cases

RajniVyas, Dr. Bhaskar Vyas**.

* Stem cell scientist, Research Director,TPC, ** Stem cell scientist, TPC

KEY WORDS : Mesenchymal stem cells, Human hair, Melanocyte. Melanin

ABSTRACT

While treating neurodegenerative disorders with autologous mesenchymal stem cells for their regenerative and rejuvenating properties, accidentally hair colorations from grey to black was observed in 4 patients. That this may be through positive up regulation of melanocyte stem cells is explained. This is not reported so far in the literature.

INTRODUCTION

Neurodegenerative diseases are caused by ageing. Mesenchymal stem cells (MSCs) are likely to emerge as a promising therapy for neurological regeneration^(1,2,3,4,5) Such therapy has an advantage as it shows no immune reaction.^(6,7) We have been conducting a clinical trial with autologous MSCs for neurodegenerative disorders. The treatment for such conditions is not available.

The trial was conducted as per Guidelines given by Indian Council of Medical Research (ICMR). Approvals for clinical trials were obtained from Institutional Ethics Committee (IEC) and Institutional Stem Cell Research Committee (ISCRC). The trial was informed to National Apex Committee for Stem Cell Research and Therapy (NAC-SCRT).

MATERIALS & METHODS

The patient's preparation for the implantation of cells was done after detailed clinical and laboratory investigations that include liver, kidney functions and hematological investigations etc.

The autologous MSCs were harvested from 200 cc of lipoaspirate by liposuction from lower abdomen subcutaneous tissue under local anesthesia. The bone marrow derived MSCs were harvested from iliac crest by aspiration of 200 ccs. Of bone marrow. Stem cells from adipose tissue and bone marrow were isolated following a fastidious protocol in our GMP Cl V laboratory. The yield was about 100 m + cells. About 5 m cells were put for culture and passaging for 2 passaging. That yielded about

40 m cells. MSC cell markers used for identification in our lab were CD29, CD90, CD 44, CD 105 positive markers & negative markers are CD 45, CD 31, CD 34 and HLA-DR. (Fig 1,2)

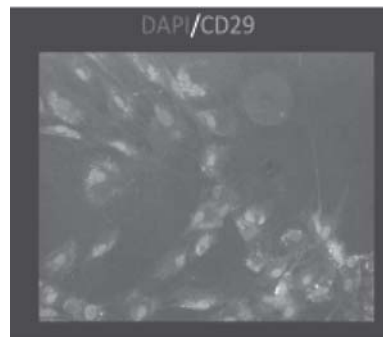


Figure 1 : DAPI/CD 29/Positive Marker



Figure 2 : CD 45/Negative Marker

Each patient received more than one session within a time frame of 2-3 weeks. Each time the MSCs were administered by spinal intrathecal route and intravenously after dividing into equal proportions over a period of 20 minutes.

Correspondence Address : Dr. Bhaskar Vyas

Total Potential Cells (P) LTD, Parimal, Pratap Road, Raopura, Vadodara- 390 001.
Email: bhaskarvyas2007@gmail.com

RESULTS

We treated a total number of 14 patients between the age group 38 - 75 years, Of them 4 subjects had completely grey hair. Three subjects were males and one female. There were no notable complications. Apart from remarkable improvement in the neurological status (being reported separately) only adverse effect was transient headache.

About 50% of the hair were seen turning black after duration of 3 - 6

weeks. This was observed on the scalp and on the beard as well.

The effect is not permanent; as the age progresses grey process continues. The longest period of follow up is 20 months. The black hair are still there but less in density and number.

Table. 1: MSCs therapy in patients with neurological diseases.

Name of Patient	Gender	Disease	Age	Number of Sessions	Route of Injection
A	F	Cerebral Palsy	38	4	I.V. (2) & Spinal (2)
B	M	Acute Spine Injury	67	2	I.V. (1) & Spinal (1)
C	M	Progressive Supranuclear Palsy	70	4	I.V. (2) & Spinal (2)
D	M	Progressive Autoimmune Disease of Anterior Horn of Motor Nerves	75	2	I.V. (1) & Spinal (1)

An Illustrative Case Report

Mr. C, aged 70 years, was diagnosed for progressive supra nuclear palsy. He had fixed gaze, unstable gait, frequent falls, slurred speech, difficulty in swallowing, uncoordinated limb movement was increased, frequency of urination with loss of bladder control etc. This patient was treated with adipose derived mesenchymal stem cells (ADMSCs) as the first session. A second session with cultured ADMSCs following two passages was done after 3 weeks. Bone marrow derived mesenchymal stem cells (BMMSCs) were similarly given by 3rd & 4th sessions. During first three weeks of his treatment, we observed improvement in his neurological status. Besides this, an interesting finding was observed that many of his grey hair turned black during this course. (Figure 3,4,5,6). Comparable results were obtained with three other patients. The details of the case studies are listed in Table 1 as above.



Figure 3 : Patient C before treatment of Progressive Supranuclear Palsy



Figure 4 : Patient C after the treatment of Progressive Supranuclear Palsy
Before Treatment After Treatment



Figure 5 : Patient D before the treatment of Progressive Autoimmune Disease of Anterior Horn of Motor Nerves



Figure 6 : Patient D after the treatment of Progressive Autoimmune Disease of Anterior Horn of Motor Nerves

DISCUSSION

We do not view MSCs as a treatment modality for grey hair. This is a sensational finding, so far not reported in the literature. We noted it accidentally during the course of treatment for neurodegenerative disorders.

The production of melanin in the hair follicles of the scalp is regulated by several molecules like alpha melanocyte stimulating hormone, adrenocorticotrophic hormone, basic fibroblast growth factor⁽⁸⁾ nerve growth factor, hepatocyte growth factor OA1, P, MATP, ATP7A, BLOC-1^(9,10,11) to synthesise eumelanin and pheomelanin^(12,13). Microtubules in the hair are involved in transport of melanin from follicular bulb to hair.^(14,15,16)

The honing properties of stem cells propagates the MSCs to target the neurodegenerative tissues.⁽¹⁷⁾ The elaborate mechanisms that correlates with the functions of MSCs are briefly summarised as follows:

MSCs have 1) Multilineage translation, 2) Facility to migrate to affected tissue and translate to it, 3) Anti inflammatory property by virtue of paracrine secretions, 4) Regenerative capability, 5) They are capable of vasculogenesis and angiogenesis. This is illustrated by figure 7.

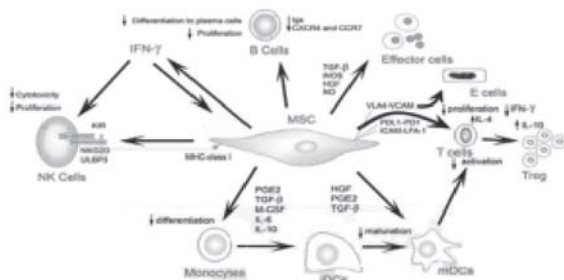


Figure 7 : Illustrating a few cytokines released by MSCs that up regulate regeneration

Hardly any information is available on the causes of grey hair. It is obvious that melanin pigment is responsible for the coloration of skin and hair. It is produced by the melanocytes. Melanoblasts are the precursors to melanocytes that originate in the neural crest and travel to hair follicular bulb and get differentiate in to melanocytes. A recent study shows that loss of colour is due to dysfunction of melanocyte stem cells in hair follicular bulb. It can be derived that MSCs travel to the diseased, dysfunctional brain area and affect the neural crest through their cytokines. This in turn will up-regulate the melanin cycle. MSCs might be positively influencing all of these factors and help in up regulating these pathways that are involved in proliferation and differentiation of hair follicular bulb.

We postulate a hypothesis that MSCs find their way to the hair follicles in the scalp and beard to up regulate the production of melanin by activating all the above factors. We wish to reiterate that this accidental finding is a so far not reported observation about the regenerative property of mesenchymal stem cells. Remaining ten patients had only partial restoration to black colour.

SUMMARY

This is a report of 4 cases of neurodegenerative disorders. While treating them with MSCs, it was incidentally discovered that their grey hair started turning black. This generates an interesting hypothesis, that MSCs play an important role in melanin regulating pathways, biosynthesis and transport in to hair follicular bulb. The hypothesis is elucidated. The cytokine pathways remains to be further researched.

REFERENCES

1. Jieli Chen and Michael Chopp, Neurorestorative Treatment of Stroke: Cell and Pharmacological Approaches, NeuroRx. 2006 ; 3: 466–473.
2. Ye Xiong, Asim Mahmood, and Michael Chopp, Angiogenesis, neurogenesis and brain recovery of function following injury, Curr Opin Investig Drugs. 2010; 11, 298–308.
3. Rosaria Giordano, Margherita Canesi, Maurizio Isalberti, Ioannis Ugo Isaias, Tiziana Montemurro, Mariele Vigan1, Elisa Montelatici, Valentina Boldrin, Riccardo Benti, Agostino Cortelezzi, Nicola Fracchiolla, Lorenza Lazzari and Gianni Pezzoli, Journal of Translational Medicine 2014, 12:14
4. Guojun Chen1, Yali Wang1, Zhenyu Xu2, Feng Fang, Renmei Xu, Yue Wang, Xiaoli Hu, Lixing Fan and Houqi Liu Neural stem cell-like cells derived from autologous bone mesenchymal stem cells for the treatment of patients with cerebral palsy Journal of Translational Medicine 2013, 11:21
5. Satake, K; Lou, J; Lenke, Migration of mesenchymal stem cells through cerebrospinal fluid into injured spinal cord tissue, 49th Annual Meeting of the Orthopaedic Research Society Paper #0078
6. Weiss man, I.L. Stem cells: units of development, units of regeneration, and units in evolution. Cell 2000, 100:157-168.
7. Barry, F.P. and Murphy, J.M. Mesenchymal stem cells: clinical applications and biological characterization. Int.J. Biochem. Cell Biol. 2004, 36, 568-584.
8. M., Naldini, L., Gaudino, G., Tamagnone, L., Coffer, A., and Comoglio, P.M. (1992). Hepatocyte growth factor is a potent angiogenic factor which stimulates endothelial cell motility and growth. J. Cell Biol. 119, 629–641.
9. Imokawa, G., Kobayashi, T., Miyagishi, M., Higashi, K., and Yada, Y. (1997). The role of endothelin-1 in epidermal hyperpigmentation and signaling mechanisms of mitogenesis and melanogenesis. Pigment Cell Res. 10, 218–228
10. Nishimura, E.K., Jordan, S.A., Oshima, H., Yoshida, H., Osawa, M., M., Jackson, I.J., Barrandon, Y., Miyachi, Y., and Nishikawa, S. Dominant Role of the Niche in Melanocyte Stem Cell Fate Determination. 2002. Nature 416, 854–860.

-
11. Yoshida H, Kunisada T, Kusakabe M, Nishikawa S, Nishikawa S.I. Distinct stages of melanocyte differentiation revealed by analysis of nonuniform pigmentation patterns. *Development*. 1996;122:1207–1214.
 12. Chakraborty, A.K., Funasaka, Y., Slominski, A., Ermak, G., Hwang J., Pawelek, J.M., and Ichihashi, M. (1996). Production and release of proopiomelanocortin (POMC) derived peptides by human melanocytes and keratinocytes in culture: regulation by ultraviolet B. *Biochim. Biophys. Acta* 1313, 130–138.
 13. Takeuchi, T. Genetic analysis of a factor regulating melanogenesis in the mouse melanocyte. *Jpn. J. Genet.* 1968, 43, 249–256.
 14. Wilkie A.L, Jordan S.A, Jackson I.J. Neural crest progenitors of the melanocyte lineage: coat colour patterns.
 15. Fang, D., T. Kute, and V. Setaluri. 2001. Regulation of tyrosinase-related protein-2 (TYRP2) in human melanocytes: relationship to growth and morphology. *Pigment Cell Res.* 14:132-139. *Development*. 2002;129:3349–3357.
 16. Jordan S.A, Jackson I.J. MGF (KIT ligand) is a chemokinetic factor for melanoblast migration into hair follicles. *Dev Biol.* 2000;225:424–436.
 17. Boquest AC, Noer A, Collas P (2006) Epigenetic programming of mesenchymal stem cells from human adipose tissue. *Stem Cell Rev* 2:319–329.
 18. Patricia A. Zuk, Min Zhu, Peter Ashjian, Daniel A. De Ugarte, Jerry I. Huang, Hiroshi Mizuno, Zeni C. Alfonso, John K. Fraser, Prosper Benhaim, and Marc H. Hedrick . Human Adipose Tissue Is a Source of Multipotent Stem Cells. *Mol Biol Cell.* 2002 12: 4279–4295.
 19. Stakeuchi, T. Genetic analysis of a factor regulating melanogenesis in the mouse melanocyte. *Jpn. J. Genet.* 1968. 43, 249-256.
 20. Chakraborty, A.K., Funasaka, Y., Slominski, A., Ermak, G., Hwang J., and Ichihashi, M. Production and release of proopiomelanocortin (POMC) derived peptides by human melanocytes and keratinocytes in culture; regulation by ultraviolet B. *Biochem. Biophys. Acta* 1996, 1313, 130-138.

CASE REPORT

Anaesthetic Management of Bilateral Carotid Body Tumor Resection

Dr. Saeed Ahmed Vohra*, Dr. Rini Majumdar**, Dr. Vinay Solanki***, Dr. B. M. Shah****, Dr. J. M. Thakkar*****,
Dr. B. M. Patel*****

*Jr, Resident, **Sr. Resident, ***Consultant Anesthetist, ****Asst Professor, *****Professor, *****Professor & HOD
Anaesthesia Department, Gujarat Cancer & Research Institute Civil Hospital, Ahmedabad

KEY WORDS : Paraganglioma, Carotid body tumor.

ABSTRACT

Eighteen year old female patient was planned for carotid body tumor resection. Patient was examined for cranial nerve involvement, compression of either carotid sinus or internal carotid arteries and functional status (catecholamine secretive state) of tumor. Doppler ultrasound neck examination, CT scan, MRI neck, bilateral carotid angiography and urinary VMA studies were done. Tumor resection difficulty and need for vascular reconstruction was assessed according to Shambling classification. Left carotid tumor (Shambling II) resection required external carotid artery ligation and carotid clamping for surgical repair of internal carotid artery while tumor resection. Pharmacologic cerebral protection from ischemic insult was done with barbiturates. In the view of prolonged postoperative bradycardia during previous surgery, right carotid tumor resection (Shambling I) was done with prophylactic temporary transvenous pacemaker insertion according to cardiology advice. Postoperative hoarseness of voice was treated with steroids and speech therapy.

CASE REPORT

Eighteen year old female patient was admitted with bilateral carotid body tumor present since last six years. Systemic examination was normal. Evidence of cranial nerve involvement like dysphagia, choking, hoarseness of voice, Horner syndrome, syncopal attacks suggestive of compression of either carotid sinus or ICA or inappropriate catecholamine secretion was not there. No family history of carotid paraganglioma was there. Routine hematological, biochemical, radiological investigations, coagulation profile, ECG were normal. Doppler ultrasound examination revealed well defined hypo echoic masses with low flow pattern vascularity measuring 30x25x24mm on left side(Shambling II) and 26x22x16 on right side(Shambling I).CT scan examination showed bilateral intense enhancing cervical masses along the course of carotid artery. Carotid angiography showed presence of bilateral carotid body tumors without any definite evidence of independent feeders from external carotid, internal carotid or vertebral artery on respective neck side with intact circle of Willis.

Left carotid tumor resection was planned. Tumor was of Shambling II i.e. lesion more adherent to the adventitial layer and partially encircle the vessel at bifurcation that may need vascular reconstruction and/or intravascular shunt placement.⁽¹⁾ Baseline blood pressure was 120/76 mm of Hg. Monitoring of HR, ECG, IBP, SPO2%, ETCO2

was done. Anesthesia induction was done with Inj Fentanyl, Thiopentone Na, and Succinylcholine and maintained with O2, N2O, Isoflurane, Vecuronium bromide. While tumor handling, frequent episodes of bradycardia, HR 40-48 beats/min were there which were corrected with Atropine and surgical site infiltration with Xylocaine 2%. While dissecting the tumor there was a rent in the ICA. ICA was clamped. While carotid clamping blood pressure rose to 200/110 mmHg which was lowered down to 160/90 mm of Hg with SL Nifedepine 10mg. BP was maintained in the same range to maintain the cerebral perfusion pressure. During surgical repair of ICA, cerebral protection from ischemic insult was done with Thiopentone, steroids, diuretics and normocapnia. Intraluminal stent was not needed as the rent was repairable without need of vascular reconstruction. Carotid clamping lasted for 18minutes. At the end of ICA repair, Heparin 2500units IV was given. Surgical site at ICA vessel was infiltrated with Papaverine. During tumor resection, ECA was ligated to reduce tumor vascularity. Patient had no neurological deficits in the post operative period. In the immediate post operative period frequent episodes of bradycardia, HR 36-40/min without any complain of palpitation, giddiness, chest pain, dyspnoea, syncope or fever were there. Bradycardia was treated with Atropine, Isoprenaline infusion. Cardiology advice of close cardiac monitoring, ECG at time of bradycardia, not to touch the right carotid artery

Correspondence Address : Dr. Saeed Ahmed Vohra

20/457, Govt A Colony, Nr. Dariakhan Ghummat, Shahibaugh, Ahmedabad 380004

E mail : vohrasaeed@yahoo.com

and not to do the carotid massage if sudden tachyarrhythmia's occurs, was followed. On fourth post operative day HR was between 60-70/min.

Right carotid tumor resection was planned after ten months. Tumor was of Shambling I i.e. Tumor can be easily isolated and dissected from carotid vessels. CT scan neck showed enhancing mass right of the neck causing splaying of internal and external carotid artery and displacing IJV posteriorly. MRI neck also showed carotid body tumor causing splaying of vessels .ECG showed single coarse episode of atrial fibrillation with heart rate 100/min. 2D Echo showed normal left ventricular function and ejection fraction 60%. In the view of prolonged postoperative bradycardia during previous surgery and cardiologist's advice, prophylactic temporary pacemaker insertion was planned. Pacemaker insertion was done through femoral vein under local anesthesia with fluoroscopic guidance. While dissecting the tumor, in spite of surgical infiltration with Xylocard 2%, episode of bradycardia, HR 40 beats/min was there. Immediately pacemaker function was taken over. After Atropine administration pacing rhythm on monitor was replaced by sinus rhythm. Tumor resection was completed within 2 hrs. Post operative vitals were normal. Temporary pacemaker was kept in-situ for further 48 hrs according to cardiology advice. On follow up examination after one month hoarseness of voice was present. IDL showed reduced movement of right vocal cord. Oral steroids and speech therapy resulted in 50% recovery.

DISCUSSION

Carotid body tumors are paragangliomas arising from chromaffin cells. Carotid body paraganglioma is the most common extra adrenal parasympathetic paraganglioma. In 5% of cases tumors can be associated with pheochromocytoma. It preferentially affects women living at high altitude. They are most likely to occur during fifth decade of life. Familial cases such as MEN syndromes tend to develop in younger patients and are more frequently bilateral and multifocal. De novo germ line mutation in tumor susceptibility gene can occur or it can occur spontaneously in hyper plastic glands.⁽²⁾ Carotid body has both baroreceptive function i.e. increase or decrease of heart rate, arterial pressure, vasomotor tone and chemoreceptive function i.e. detects reduction of pH and of oxygen pressure, increase in carbon dioxide pressure in addition to producing an increase in respiratory frequency, depth and minute volume by stimulation of glossopharyngeal nerve. Response to hypoxia and carbon dioxide is due to pH change in carotid body which contains carbonic anhydrase enzyme.⁽²⁾

Patient can present with dizziness, palpitation, hypertension, dysphagia, hearing loss, ptosis of eyelid.⁽¹⁾ Frequency of preoperative peripheral nerve deficits like

vagus, hypoglossal nerve paralysis and Horner syndrome is 11%. Carotid body tumors grow with progressive involvement of ICA and ECA without constricting arterial lumens. They can extend to the base of skull. Radiological imaging are helpful in determining size, extent, vascularity of tumor, status of carotid, vertebral vessels and circle of Willis which is essential for vascular clamping, reconstruction or even preoperative embolization of feeding vessels⁽⁴⁾Invasive arterial line placement was done in the view to maintain blood pressure within specified range, perform blood gases and coagulation studies. Normocapnea was preferred because cerebral vessels in the area ischemic are already dilated. Hypercapnea may result in dilatation of normally responsive vessels outside the ischemic area. This phenomenon termed, "steal", may divert blood flow away from ischemic area, further compromising perfusion. Hypocapnea may cause vessels in the ischemic area to undergo constriction, converting marginally ischemic areas to truly ischemic areas.⁽²⁾

Clamping of ICA causes pressure drop beyond the clamp, i.e. focal ischemia. Cerebral vasculature beyond the clamp loses its auto regulatory ability and undergoes vasodilatation. Cerebral blood flow here becomes passive and depends on systemic arterial pressure for adequate perfusion. The combination of carotid occlusion and hypotension produces ischemia in arterial boundary zones. Electroencephalography, Somatosensory evoked potentials, Trans cranial Doppler, ICA stump pressure measurement can measure cerebral perfusion and guide for intra luminal shunt placement.⁽³⁾ Blood pressure during this period was maintained in specific predetermined range of approx 20 % higher than baseline value to prevent ischemic cerebral insult⁽⁵⁾ Rise in blood pressure while carotid clamping can be body's own physiological protective mechanism to optimize cerebral circulation. Abolishing cerebral electrical activity and reducing CMRO2 are the factors of importance during focal ischemia. Barbiturates have an additional effect of doing redistribution of cerebral blood flow which has protective role in focal ischemia. Cerebral metabolic activity depression is uniform in both cortical and sub cortical regions with barbiturates.⁽⁶⁾ Brain edema frequently follows ischemic insult. It is combination of cytotoxic and vasogenic edema. The major detrimental effect of edema is the impingement of potential collateral flow. Mannitol's proposed action includes reduction in cytotoxic edema.⁽⁶⁾

Preoperative optimization of serum potassium and intra operative normocapnia was maintained in view of pace maker function optimization. Bipolar electro cautery was used to prevent electro magnetic interference that may result in inhibition of ventricular synchronous pulse generator and failure of pace maker function.⁽⁷⁾ Vecuronium was used for muscle relaxation as

fasciculation's produced by succinylcholine may result in malfunction of pace maker. Frequent episodes of bradycardia and preoperative single episode atrial fibrillation may be the result of sensitization of dysfunctional carotid baroreceptors⁽⁸⁾ Carotid tumor hypersensitivity causing nocturnal attacks of epilepsy due to cardiac asystole produced by altered sinus reactivity, cured by permanent pace maker implantation has been documented. The likely mechanism must be that tumor changes the pressure transduction mechanisms within the carotid body leading to hypersensitivity.⁽⁹⁾ Post operative incidence of cranial nerve palsy lasting for more than 6 months is around 12 %. Mortality wasn't noticed in any case.⁽¹⁰⁾

In summary, carotid tumors are rare. Resection of tumor may require clamping of carotid artery and have risks similar to those of patients undergoing carotid endarterectomy. Barbiturate therapy along with temporary shunting can be used as means of cerebral protection. Altered carotid sinus receptor activity can again be a point of major concern. Interventional measures like temporary Trans venous pacing can be life saving. The mortality rate is reduced to near zero, since still incidence of neurological deficit is high.

REFERENCES

1. John H. Sampson, Robert H Wilkins. Paragangliomas of carotid body and temporal bone. Neuro surgery. Robert H Wilkins, Setty S Rengachary. Second edition, vol. 4: 1996.1559-61.
2. Stoelting RK, Dierdorf SF. Disease of nervous system In: Anesthesia and Coexisting disease.4thedition.New York, Churchill Livingstone 2002;233-2
3. M R Tinkham, RN, BSN. Care of the patient undergoing surgical excision of a carotid body chemodectoma. AORN, Inc J , 2010; 91: 117-128(January)
4. Clark NJ, Stanley TH. Anesthesia for vascular surgery. Miller RD, editors. Anesthesia-4th edition. New York: Churchill Livingstone, 1994:1851-95.
5. Allian R, Marone LK, Meltzer J, Jeyabalam G. Carotid endarterectomy. Int Anesthesiology Clin 2005; 43(1):15-38.
6. McConkey, Kien ND. Cerebral protection with thiopentone during combined carotid endarterectomy and clipping of intraluminal aneurysm. Anesthesia Intensive Care. 2007; 30:219-22.
7. Shapiro WA, Roizen MF, Singleton MA. Intraoperative pace maker complications. Anesthesiology 1985; 63:319-22.
8. BoveEL, Fry WJ, Gross WS, et al. Hypotension and hypertension as consequences of baroreceptor dysfunction carotid endarterectomy. Surgery 1979; 85:633-7.
9. Turner M, Marshall A J.Chronic bilateral carotid body tumors causing carotid sinus hypersensitivity; abolition of symptoms by permanent cardiac pacing. Heart, images in cardiology 2000, 84:196(August).
10. Mitchell RO, Richardson JD, Lambert GE. Characteristics, surgical management and outcome in 17 carotid body tumors. Ann Surg.1996; 62:1034-37.

CASE REPORT

A Pseudoaneurysm of the External Jugular Vein- A case report

Dr. Nina M. Shah*, Dr. Vishal S. Shah**, Dr. Parth R. Dalal***, Dr. Prafull Mishra****

*Head of Unit, **2nd Year Surgery Resident, ***Assistant Professor,**** 1st Year Surgery Resident

KEY WORDS : External jugular vein, Pseudoaneurysm

ABSTRACT

Venous pseudoaneurysms are one of the uncommon causes of neck swellings. External jugular vein pseudoaneurysms may be fusiform or saccular. We present a case of male who presented with a non-tender partially compressible swelling in the right supraclavicular region, which was found to be the external jugular vein pseudoaneurysm on Doppler ultrasound. Fusiform pseudoaneurysms of the external jugular vein are uncommon. Ultrasound can allow early detection of this entity.

INTRODUCTION

Venous pseudoaneurysms are rare when compared to arterial ones^[1]. The commonest site of aneurysm in the neck is the internal jugular vein^[2]. Fusiform venous pseudoaneurysm of the external jugular vein is a very rare entity and only a few cases have been reported in the English literature[3]. We report a case of a 25- years-old male with a fusiform pseudoaneurysm of the right external jugular vein diagnosed by colour doppler.

CASE REPORT:

A 25 year old male patient, soldier by occupation presented to the surgical O.P.D. with chief complain of a swelling on lower part of right side of neck since 2 months. Initially the swelling used to appear on activities like running and exercising and loud speaking and used to reduce spontaneously. But since the last 20 days, the swelling became firm and did not reduce spontaneously. On manual reduction it decreased in size. Patient had a history of repeated trauma to neck due to the belt and buckle of his rifle. There was no history of tingling and numbness in the hand especially in the little and ring finger. There was no cyanosis of the tip of the fingers. There was no history of pain in the swelling. There was no history of pain even on hyperabduction of the arm nor did the swelling increase in size on this maneuver indicating that the swelling did not compress the neurovascular bundle of the thoracic outlet. There was no oedema the right upper limb or dilated veins over the face. There was no history of ecchymosis or discolouration of skin suggestive of rupture of external jugular vein. It indicated pseudo aneurysm had developed slowly and progressively. There was no past history of cervical

lymphadenopathy or Anti Koch's Treatment (AKT) taken.

On examination there was a single 7cm× 6cm non warm, non-tender swelling, located in the supraclavicular region with smooth surface and overlying normal skin. The swelling was firm in consistency, non-pulsatile, partially reducible and had limited mobility in all the planes. The swelling was not fixed to the skin and became tense on Valsalva maneuver. External jugular vein above the swelling was normal. There was no raised jugular venous pressure (non-pulsatile external jugular vein). Upper limb arterial pulsations were normal. There was no evidence of any neurological involvement. Adson's and Allen's test were negative. Fluctuation and transillumination were negative.

Duplex scan showed a fusiform dilatation of the external jugular vein with internal colourflow confirming it to be pseudoaneurysm of the external jugular vein.

The aneurysm was approached by a supraclavicular linear incision about two finger breadths above and parallel to the clavicle. The clavicular head was not divided since the aneurysm was limited to the supraclavicular region. Pseudoaneurysm of about 7cm×6cm of the external jugular vein was found. Vein found to be collapsed proximally. Two tributaries namely transverse cervical and transverse scapular veins found at the distal end at 9 and 7 o' clock positions draining into the pseudoaneurysm. Both the tributaries were ligated doubly and cut. External jugular vein was ligated doubly proximally and distally and cut. Sharp dissection was done around the swelling and

Correspondence Address : Dr. Vishal S. Shah
35/209, Ellora Park, Race Course Circle, Vadodara.
E-mail : drvishal_2007@yahoo.co.in

swelling delivered in toto. The anterior jugular vein and the arch vein were found to be patent without thrombosis and hence there was no need of an interposition graft.

Histopathological examination was suggestive of a dilated vascular structure with the presence of hemorrhage and thrombosis with disruption in endothelium consistent with the clinical diagnosis of external jugular vein pseudoaneurysm.



Fig I : Supraclavicular swelling

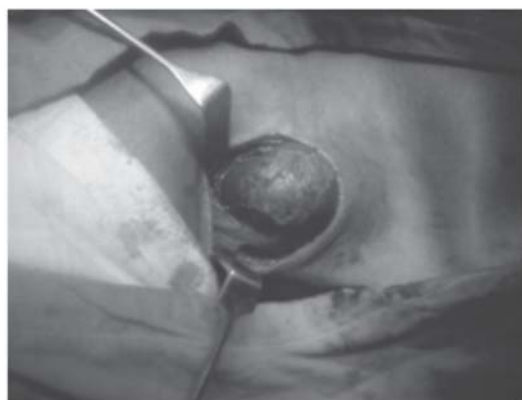


Fig II : Intraoperative image of the pseudoaneurysm of right external jugular vein

DISCUSSION

The external jugular vein is formed by the junction of the posterior division of the retromandibular vein with the posterior auricular vein. It crosses the sternocleidomastoid obliquely, and in the subclavian triangle perforates the deep fascia, and ends in the subclavian vein lateral to or in front of the scalenus anterior muscle. External jugular veins develop as secondary channels and not derived from the anterior cardinal vein. The external jugular vein can be used to measure the CVP. The external jugular vein is easier to visualise than the internal jugular vein and may give a reliable estimate of

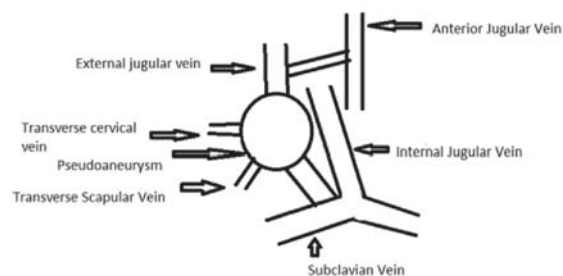


Fig III- Schematic diagram of the pseudoaneurysm

CVP. It can also be used to insert a central venous catheter for central venous access. Venous aneurysm is fusiform or saccular dilatation of a vein. It can be primary (congenital) or acquired [4]. Primary venous aneurysms are true venous aneurysms as they contain intact venous wall and are usually fusiform or localized dilatation of a vein[5]. Acquired aneurysms are caused by several factors such as inflammation, trauma, venous valve insufficiency, tumours, arteriovenous fistula which may occur due to blunt trauma or penetrating trauma or bullet injury. Internal jugular venous aneurysm is more common than external jugular venous aneurysm. These can be differentiated by MR venography preoperatively for better planning for surgery. It also shows extent of thrombosis into the anterior jugular vein. Superficial venous aneurysm of the neck can be mistaken for lymph node enlargement and variety of cystic swellings such as cold abscess, cystic hygroma, cavernous haemangioma, laryngocele or with arterial aneurysms. Complications of superficial venous aneurysms include thrombosis, rupture, thrombophlebitis and pulmonary embolism although very rare.

Doppler ultrasound is the first imaging technique performed because it is non-invasive and can differentiate vascular from non-vascular cause of neck swelling. CT and MRI can more accurately demonstrate the size and extent of aneurysms. Another modality of diagnosis is MR venography which shows well-defined, hypo echoic masses with heterogeneous echo texture, cystic or sinusoidal spaces, and possible phleboliths. It typically shows intermediate signal intensity (slightly higher than that of muscles) on T1-weighted images, very high signal intensity on T2-weighted images, and variable enhancement after intravenous gadolinium administration. Colour Doppler of both lower limbs and abdominal ultrasonography to see the great veins should be done to rule out any other venous malformations.

Asymptomatic aneurysms and pseudoaneurysms less than two centimeters are diagnosed incidentally and can

be managed conservatively with reassurance and regular follow-up. Surgical excision is offered for either cosmetic reasons or a painful pseudoaneurysm secondary to thrombosis or phlebitis of the jugular venous system.^[6] Surgical resection also eliminates the theoretical risk of aneurysmal rupture, pulmonary embolism and allows for histopathological diagnosis. Asymptomatic fusiform jugular venous pseudoaneurysm can be safely managed by excision and ligation, while exclusion and bypass is indicated in both saccular and fusiform dilatation. If MRI shows extension of thrombosis into the anterior jugular vein (arch), modality of treatment is ligation of vein proximally and distally with or without excision and by pass graft with external jugular and subclavian vein.^[7] Greenfield filters are not recommended in the neck as it is nearer to the heart and veins are superficial^[8]

REFERENCES

1. Karapolat S, Ekut B, Unlu Y (2005) Multiple aneurysms of the left external jugular vein. *Turk j Med Sci* 35:43-45
2. McCready RA, Bryant MA, Divelbiss JL, Chess BA (2007) Subclavian venous aneurysm: case report and review of the literature. *J VascSurg* 45 (5): 1080-1082
3. Drakonaki EE, Symvoulakis EK, Fachouridi A, Kounalakis D, Tsafantakis E(2011) External jugular vein aneurysm presenting as a cervical mass. *Int J Otolaryngol* 485293. Epub May 16, 2011
4. REgueiro Mira F, GalbisCaravajal JM, canto Armengod A (2002) Thoracic venous aneurysms. Clinical observation. *J CardiovascSurg (Torino)* 43(4): 527-529
5. Al-Shaikhia , Kay S, Laberge JM (2003) External jugular venous aneurysm: an unusual cause of a neck mass in a young child. *J PediatrSurg* 38:1557-1559
6. Lee HY, Lee W Cho Yk, Chung JW, Park JH (2006) Superficial venous aneurysm: reports of 3 cases and literature review. *J Ultrasound Med* 25(6): 771-776
7. Stell and Maran's Textbook of Head and Neck Surgery and Oncology
8. Jatin Shah's Head and Neck Surgery and Oncology

CASE REPORT

Multiple Endocrine Neoplasia Type 1 (Wermer's Syndrome)

Dr. A. A. Ghasura, Dr. K. H. Patel, Dr. Bharat Prajapati, Dr. Hitesh Mahida, Dr. Vikramaditya Oza

KEY WORDS : Multiple Endocrine Neoplasia type 1 (MEN1), hyperparathyroidism, insulinoma, gastrinoma, pituitary tumor.

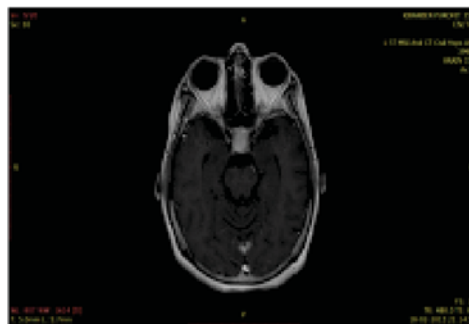
ABSTRACT

Multiple endocrine neoplasia type 1, inherited as an autosomal dominant disorder, leads to hyperplastic/neoplastic changes in parathyroid, pituitary and endocrine pancreas along with other characteristic tumors. Hyperparathyroidism is the most common manifestation of MEN1. Our case was a female patient aged 35 years who, diagnosed with parathyroid adenoma, coincident with Pituitary macroadenoma & Pancreatic Neuroendocrine tumors. Hyperparathyroidism was noticed initially & parathyroidectomy with transcervical thymectomy and hemithyroidectomy was performed. Elevated prolactin levels were also noted, and sella MRI revealed a macroadenoma, was treated conservatively. A multidisciplinary approach involving endocrinologists, surgeons, oncologists, and radiologists is pivotal for optimizing patient treatment. Treatment consists of surgery and/or drug therapy, often in association with radiotherapy or chemotherapy.

CLINICAL PRESENTATION

A 35-yr-old female with history of several hospital admissions due to abdominal pain, nausea, and vomiting was admitted to our hospital. On examination patient was afebrile, hemodynamically stable. The abdomen was soft, non distended, without guarding or rigidity. Patient was unmarried and had secondary Amenorrhoea. Other findings were normal RBS, mild Anemia and hypercalcemia.

- CECT ABDOMEN + PELVIS showed a diffusely edematous pancreas & peri pancreatic/mesenteric fat stranding, dilated main pancreatic duct with calculi suggestive of acute on chronic pancreatitis. Heterogeneously enhancing lesion adjacent to head of pancreas suggestive of (Gastrinoma/paraganglioma/ Nodal mass)
- Bilateral renal and pancreatic parenchymal calcification



- MRI Brain – intensely enhancing sellar and suprasellar region Suggestive of Pituitary Macroadenoma
- USG NECK – 4×2 cm inferior pole of Rt. Lobe of thyroid Suggestive of Parathyroid Adenoma. Thyroid lobes are normal in size & vascularity.
- S. PTH – 312.4 pg/ml (14 – 72)
- TSH – 0.21 mU/ml (0.25 – 5.0)
- FSH – 0.31 ng/ml (1 – 90)
- PO4 – 2.1 mg/dl (3.4 – 6.2)
- S. Gastrin – 129 pg/ml (<150)
- S. Insulin – 1.4 microIU/ml (6.0-27)
- EUS guided FNAB – Well differentiated neuroendocrine tumour
- Synaptophysin (Neuroendocrine marker) Positive
- CK (Epithelial marker) positive
- KI-67 (Proliferation marker) Low

Correspondence Address : Dr. A. A. Ghasura
Dept. of Surgery, B. J. Medical College, Civil Hospital,
Ahmedabad.

S. Calcium– (Avg.)(mg/dl) Total	On Admission	On Discharge	Follow up
ionised	14.2 7.4	9.50 5.00	9.00 5.3

S.Prolactin (Avg.)(ng/ml)	On Admission	On Discharge	Follow up
	4800	754	736

Patient was given I.V. antibiotics & fluids, oral Cabergoline(0.5mg

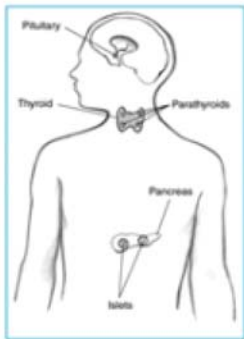
twice in a week) with iv. Pamidronate(90mg). She underwent total 4 gland parathyroidectomy with transcervical thymectomy and hemi thyroidectomy due to thyroid adenoma found intraoperatively. Patient was given supplementary calcium post operatively. She had hypocalcemia on 6th post operative day with mild perioral tingling, mild hoarseness of voice and weakness. We continued supplementary Calcium & Dopamine agonists. After discharge, follow up PTH & S.Calcium level came within normal limits. Patient continued supplementary calcium, Cabergoline, Pancreatic enzymes.

Biopsy : parathyroid adenoma with normal thyroid, thymus & lymph nodes.

Every monthly follow up revealed S.Calcium within normal limits, S.prolactin was raised so Cabergoline (0.5mg) continued thrice a week with supplementary calcium and pancreatic enzymes. Follow up CECT scan and MRI brain show same findings as of above without any significant symptoms.

DISCUSSION :

- rare autosomal dominant hereditary syndrome
- tumours of Parathyroids, Endocrine pancreas and Anterior pituitary.



- Very high penetrance, equal sex distribution
- Prevalence is 0.04-0.2 cases/ 1000 population/ year.
- Types - sporadic and familial.

- Sporadic- any 2 of the 3 among parathyroid adenomas, entero-pancreatic tumours, pituitary tumours.
- Familial - Atleast one first degree relative showing one of the characteristic endocrine tumours.
- Others - adrenal cortical tumours, carcinoids of bronchi, gastrointestinal tract and thymus, lipomas, angiofibromas, collagenomas.
- The mutated gene on chromosome 11q13 encodes a protein, menin.

Hyperparathyroidism :

- Usually First & most common presentation.
- Parathyroid tumors increased PTH Hypercalcemia, can exist for many years before being found incidentally or through screening for MEN1
- Altered mental status, lethargy
- Anorexia, constipation, nausea, vomiting
- Polyuria, polydipsia, dehydration, hypercalciuria kidney stones
- Increased bone resorption
- Hypertension
- Hypercalcaemia may increase the secretion of gastrin gastrinoma.
- Parathyroid adenoma are generally characterised by Multiglandular hyperplasia.
- Parathyroid Localization can be done by USG, CT, MRI Intraoperative PTH assay etc.

Management :

- Total Parathyroidectomy including Cervical Thymectomy followed by daily calcium supplements & vitamin D.
- 3 ½ Gland resection with forearm implant with Cervical Thymectomy.
- Serial monitoring of S.Ca.

Pancreatic Neuroendocrine Tumors (PNETs) :

- In 30–80% of MEN1 patients.
- Produce excessive hormones (gastrin, insulin, glucagons, somatostatin, neurotensin or vasoactive intestinal polypeptide
- Most Common functional : gastrinomas and insulinomas.
- Non-functional tumours and insulinomas are located within the pancreas
- Gastrinomas are present around the pancreas and in duodenal submucosa

Endoscopic ultrasonography is most sensitive(75%) for detection of small (≤ 10 mm) PNETs.

Gastrinomas :

- more than 50% of all pancreatic tumours.
- Biochemical diagnosis : increased basal gastric acid secretion.
- elevated basal S. gastrin (normal <100)



Typically present in Passaro's Triangle :- Junction of Cystic duct & CBD, 2nd & 3rd part of duodenum, neck & body of pancreas.

Management :

- non-metastatic surgical resection.
In duodenum/head of pancreas Pylorus Preserving Partial Pancreticoduodenectomy
Body/Tail pancreas Distal pancreatectomy with excision of tumor in head of pancreas
- Multiple and disseminated human somatostatin analogue (octreotide), PPIs or H2-receptor blockers, chemotherapy.

Zollinger-Ellison syndrome

- One or more tumors in pancreas or duodenum.

Symptoms : Upper abdominal pain, diarrhoea, oesophageal reflux, vomiting and acid-peptic or duodenal ulcers and, weight loss.



Treatment

PPIs are first choice.

Single gastrinoma without dissemination Surgical resection

Gastrectomy control acid production.

Insulinomas

Characterised by fasting hypoglycaemia

High plasma or S.insulin together with high plasma or S.C-peptide.

Surgery is the main treatment.

Chemotherapy is used for metastatic disease

VIPoma

Vasoactive intestinal peptide secreting tumours occur as WDHA syndrome: Watery Diarrhoea, Hypokalaemia and Achlorhydria. increased plasma VIP concentration (Normal <75 pg/ml).

Surgical excision is curative in many cases.

Glucagonoma

Excess Glucagon → Glucose intolerance, hyperglycemia, excessive thirst, frequent urination, weight loss Usually malignant.

Tests

CT abdomen, S.Glucose, S.Glucagon

- Surgery is treatment of choice

Anterior pituitary tumours

Diagnosed by CT scanning and MRI.

Prolactinoma (60%)

increased S.Prolactin

- Symptoms : Galactorrhoea, amenorrhoea,infertility in women, and hypogonadism, sexual dysfunction, gynecomastia in men.
- Mass compression Headache, blurred vision etc..
- Medical treatment :- dopamine agonists such as cabergoline, bromocriptine, pergolide.

Associated endocrine tumours:

Adrenal cortical tumours

- Majority are non-functioning
- Functioning → elevated S. Cortisol → hypercortisolaemia and Cushing's syndrome.
- Mostly require surgical removal of tumours > 3 cm in diameter

Thyroid & Thymus tumours

- Thyroid adenoma, colloid goitres and carcinomas in over 25% of MEN1 patients.

Associated Non-endocrine tumours

Carcinoids :

May be located in the bronchi, GIT, pancreas, thymus.

- mostly asymptomatic. Rarely oversecrete ACTH, calcitonin, GHRH, serotonin or histamine. Diagnosed by X-ray/ CT examination.
- Others : Facial angiofibroma & collagenomas, Lipomas, Meningiomas, Somatostatinomas.

CASE REPORT

Pregnancy In Rudimentary Horn: A rare entity

Hina V. Oza*, Hafsa M. Vohra**, Nisarg H. Patel**, Daisy Y. Shethna***

*Associate Professor and Head of unit, **Assistant Professor, ***3rd Year Resident
Department of Obstetrics and Gynecology, Civil Hospital Asarwa, Ahmedabad.

KEY WORDS : Unicornuate uterus, rudimentary horn pregnancy

ABSTRACT

Pregnancy in non-communicating rudimentary horn is very rare and life threatening. It is often not diagnosed unless it terminates by rupture in the 2nd trimester. Prerupture diagnosis of rudimentary horn pregnancy with USG is technically difficult, with sensitivity of 30%. Here we present a case of unruptured non-communicating rudimentary horn pregnancy at 15 weeks presenting in our emergency department as a case of intrauterine death and induction failure. Laparotomy was carried out and excision of rudimentary horn was done.

INTRODUCTION

Mullerian duct anomalies in female results from defective fusion or resorption during embryonic life. Rudimentary horn is the rarest uterine anomaly and pregnancy in non-communicating rudimentary horn is even rarer form of ectopic gestation¹. The only possible explanation for pregnancy to occur in this case is by trans-peritoneal migration of spermatozoa or embryo through contralateral tube². Incidence of rudimentary horn pregnancy is between 1 per 76000 to 1 per 140000 pregnancies^{2,3}. Natural history of rudimentary horn pregnancy is usually rupture of pregnant horn during 2nd and 3rd trimester resulting in life threatening intraperitoneal haemorrhage. Therefore early pre-rupture diagnosis is of major importance. We report a case of ectopic pregnancy in non-communicating rudimentary horn.

CASE HISTORY

A 26 year old primigravida patient presented to our hospital on 8/03/2014 with H/O 4 months amenorrhoea with c/o abdominal pain for 2 days. Her last menstrual period was before 4 months. She had an AML of 7 years and had taken treatment for infertility. Patient had history of diagnostic laparoscopy before 8 months at private hospital. Findings of laparoscopy was unicornuate uterus with right side horn non communicating with uterine cavity with no spillage on right side. Left side horn was developed with free spillage on that side. Excision of non-communicating horn was advised, but patient had refused. In the current pregnancy, the patient attended another consultant and laparoscopy findings were not revealed. After conception, owing to IUD, induction with

PGE₁ was done failing of which patient was referred to CHA.

On examination, she was vitally stable. On per abdomen examination, uterus corresponded to 14-16 weeks pregnancy. On per speculum examination cervix was normal. On per vaginum examination, cervix was firm, 2.5cm long and os was closed. There was no adnexal mass felt. There was no active bleeding.

Her investigations showed Haemoglobin 10.2 gm/dl, WBC 8300/cu.mm, Platelet count 3.08 lac/cu.mm and coagulation profile was normal. Transabdominal USG at Radiology department showed a single intrauterine pregnancy with absent cardiac activity having 15 weeks maturity. Generalised foetal oedema and effusion seen. A clinical impression of intrauterine fetal death was made.

A repeat ultrasound was carried out by senior gynaecologist and co-related with laparoscopy findings, pregnancy in rudimentary horn was suspected. Patient was counselled and prepared for laparotomy.

Intra-op findings: Foetus in the right side non-communicating horn, right fallopian tube and ovary attached to right horn (figure I). Left horn of uterus with left fallopian tube and ovary were normal. Incision was kept on right side non-communicating horn of uterus. A 14-16 weeks fetus was delivered out along with placenta and its membranes (figure II). Right side fallopian tube, ovarian ligament, round ligament were clamped, cut and doubly ligated. Right side non communicating horn excised from main uterus and defect was repaired in two layers.

Postoperative period was uneventful. The stitches were removed and patient discharged on 10th postoperative

Correspondence Address : Dr. Daisy Y. Shethna

C-108, New P. G. Hostel, Civil Hospital Campus, Asarwa, Ahmedabad-380016.

E-mail : shethna.daisy@yahoo.com



Figure I: Unicornuate uterus with pregnancy in right side non-communicating rudimentary horn.



FIGURE II: After keeping incision on right side non-communicating horn, a 14-16 weeks fetus delivered out.

day. She was counselled about need for antenatal care and elective caesarean section for any future pregnancy.

HISTOPATHOLOGY REPORT

The gross specimen measured 6x5x3cm, endometrium and myometrium 0.4 and 0.6 cm in size. Uterine canal 4cm in length. Cervix not identified. Sections from uterus show histology of retained products of conception and consistent with clinical diagnosis.

DISCUSSION

Rudimentary horn with unicornuate uterus results from failure of complete development of one of the mullerian duct and incomplete fusion with the contralateral side. It is a class 2B anomaly according to the American Fertility Society Classification of Mullerian anomalies. In 83% of cases the rudimentary horn is non-communicating⁴. The use of ultrasonography, CT scan, MRI, 3D ultrasonography and laparoscopy may be helpful for diagnosing such abnormalities⁵. Sonographic diagnostic criteria suggestive are (1) Pseudo-pattern of an asymmetrical bi-cornuate uterus (2) Absent visual continuity between cervical canal and lumen of pregnant horn (3) Presence of myometrial tissue surrounding Gsac⁶.

Rupture of pregnancy in the rudimentary horn by the 2nd trimester is most common but silent rupture as the continuation of pregnancy as secondary abdominal pregnancy was reported by some studies. Cases of pregnancy progressing to the third trimester and resulting live birth after caesarean section have also been documented⁷.

A rudimentary horn pregnancy can never be delivered vaginally and mode of delivery is always a laparotomy. Surgical removal of the rudimentary horn is mandatory to avoid risk of uterine rupture with increased maternal morbidity. However laparoscopic excision of an unruptured rudimentary horn pregnancy has been increasingly carried out with safe and favourable outcome in many expert centres now⁸.

CONCLUSION

This case report highlighted the need for high level of suspicion of this rare entity. In this report we suggest criteria for early sonographic diagnosis of rudimentary horn pregnancy. High clinical suspicion, early diagnosis and timely laparotomy can reduce maternal morbidity and mortality.

In our opinion, routine excision of rudimentary horn should be undertaken during non pregnant state. However, those women who refuse should be adequately counselled regarding potential complications and if pregnancy occurs in rudimentary horn, first trimester laparoscopic excision should be done.

REFERENCES

1. Tufail A, Hasmi HA: Ruptured ectopic pregnancy in rudimentary horn of the uterus. *J Coll Physicians Surg Pak.* 2007, 17:105-106.
2. Johansen K. Pregnancy in a rudimentary horn: two case reports. *Obstet Gynecol* 1969; 34:805-808.
3. Nahum GG. Rudimentary uterine horn pregnancy: a case report on surviving twins delivered eight days apart. *J Reprod Med* 1997; 42:525-532
4. Heinonen PK: Unicornuate uterus and rudimentary horn. *Fertilsteril.* 1997, 68:224-230.
5. Nanda S, Dahiya K, Sharma N, Agarwal D, Singhal SR, Sangwan N. Successful twin pregnancy in a unicornuate uterus with one fetus in the non-communicating rudimentary horn. *Arch Gynecol Obstet* 2009; 280: 993-5.
6. Tsafir A, Rojansky N, Sela HY, Gomori JM, Nadjari M. Rudimentary horn pregnancy: first trimester prerupture sonographic diagnosis and confirmation by MRI. *J Ultrasound Med* 2005; 24: 219-23.
7. Amritha B, Sumangali T, Priya B, Deepak S, Sharadha R. *J Med Case Rep* 2009; 3: 38. doi:10.1186/1752-1947-3-38.
8. Henriot E, Roman H, Zanati J, Lebreton B, Sabourin JC, Loic M. Pregnant non-communicating rudimentary uterine horn with placenta praecreta. *JSL* 2008; 12: 101-3.

CASE REPORT

Hemoptysis as The Presenting Symptom in Ards in A Patient of Plasmodium Vivax Malaria (A Case Report).

Dr. Alpa Leuva* , Dr. Jyoti Mannari**, Dr. Chinmaye Sapre***

*Professor, ** Head of Department, ***Resident, Medicine department Shree Krishna hospital, Karamsad

KEY WORDS : vivax, ARDS , hemoptysis

ABSTRACT

Malaria is an important parasitic disease of humans, affecting more than 1 billion people worldwide, causing around 1 to 3 million deaths due to complication in plasmodium falciparum every year with prevalence in 103 countries.(1)

Plasmodium vivax causes benign clinical picture, but increasing reports of complication and deaths are being reported.(6)Here we present a case of benign tertian vivax malaria who developed ARDS as a late complication with only complaint of hemoptysis on its presentation. Timely Supportive treatment in form of oxygen supplementation helped us avoid invasive ventilation and mortality..

INTRODUCTION

Malaria is an important parasitic disease of humans, affecting more than 1 billion people worldwide, causing around 1 to 3 million deaths every year with prevalence in 103 countries. 1% of the patients with Plasmodium falciparum infections develop more severe manifestations resulting in the failure of organ systems. This is rarely seen with Plasmodium vivax.(1)

Definition of severe malaria includes (1):

One or more of the following criteria + the presence of asexual parasitaemia

- Cerebral malaria/unarousable coma
- Severe anaemia
- Renal failure
- Pulmonary oedema/adult respiratory distress syndrome (ARDS)
- Hypoglycemia
- Hypotension/shock
- Bleeding/disseminated intravascular coagulation (DIC)
- Convulsion
- Acidosis/acidaemia
- Macroscopic haemoglobinuria

These complications are also rarely seen with plasmodium vivax malaria. (1)

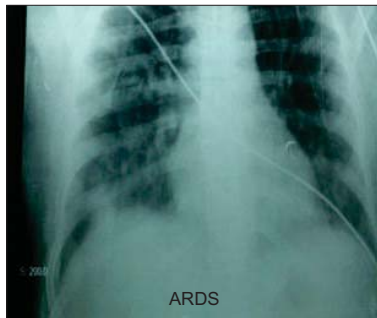
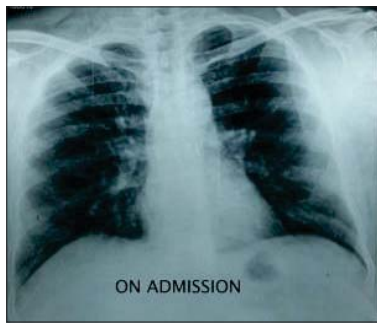
Severe complications of vivax malaria occur from around 6 hrs to 8 days after initiation of the anti-malarial treatment.(2) In adults severity of the spectrum of the disease of vivax malaria is diverse. (3)

Here we will discuss a case of a patient of vivax malaria developing ARDS presenting as hemoptysis on the third day of treatment.

CASE SUMMARY

A 54-year-old male patient, diabetic on treatment, presented with the history of fever with chills, remittent and cough with whitish expectoration with bodyache for 3 days. The patient was hemodynamically stable with no significant findings on systemic examination with Spo2= 98% on Room air. Investigations showed thrombocytopenia (PC=63000) with plasmodium vivax positive on malaria serology test and MP smear showing 2-3 rings and 1-2 trophozoites of plasmodium vivax. Thus tablet chloroquine was started. The patient had persistent fever spikes on day 2 but no hemodynamic instability. On day 3 of treatment the patient developed complaint of hemoptysis, difficulty in breathing sudden onset, with examination revealing tachycardia and tachypnoea with bilateral crepitation's present in the infrascapular region (right>left) with spo2= 93% on room air. The ABG showed paO2=58% and po2: fio2 ratio of 187. Chest x-ray showed bilateral haziness in the lower zones suggestive of moderate ARDS. 2D echo showed ejection fraction of 60%. Reports showed TC=9700 with 80% polymorphs and PC= 50000/microlitre with normal creatinine, LFT and electrolytes. CRP was 142 mg/l and procalcitonin =

Correspondence Address : **Dr. Chinmaye Sapre**
139/140 orchid bungalows, old padra road
Baroda-390020



0.79 ng/ml. O₂ supplementation was started. Antibiotics along with injectable artesunate was started. Over the next 2 days the patient had persistent hemoptysis but rest of the clinical and the laboratory parameters showed improvement. Blood and sputum culture were found to be negative. Following the episode of hypoxemia there was no fever spikes. There was no blood present in the sputum on day 5. The patient's antibiotics were gradually stepped down.

DISCUSSION

Malaria has a wide clinical spectrum. Data shows that the vivax infected RBC's cytoadhesin are present on the lung endothelial cells⁽⁴⁾ but to a lesser degree than falciparum. Hence there lies a possibility of developing ARDS even with plasmodium vivax. There are random reports of shock with ARDS,⁽⁵⁾ pulmonary edema⁽⁶⁾ in vivax malaria. The notion of vivax malaria being benign is not fully true.

Pulmonary involvement in malaria can be asymptomatic or with symptoms such as cough which may be ignored. Compromised respiratory function has been demonstrated in clinically uncomplicated cases of vivax malaria. Sarkar et al shows the presence of ARDS as a rare but emerging complication seen with plasmodium vivax malaria. All 3 cases who all developed ARDS during different times in the treatment & had complete recovery with the help of supportive measures like mechanical ventilation and PEEP.⁽⁷⁾

Here we report a case of complicated malaria where mixed infection was ruled out on the basis of peripheral smear and a one step Pf/Pv rapid antigen test.

The patient developed ARDS with hemoptysis after 48 hours of initiation of chloroquine therapy and showed improvement only with supportive measures. Patient had recovering trend in form of improving fever spikes, when he developed ARDS. Requirement of any non-invasive or invasive ventilatory support was not required.

Supportive therapy like O₂ supplementation with prompt recognition leads to the evasion of need of NIV or invasive ventilator support.

CONCLUSION

ARDS is a serious complication rarely seen with vivax malaria, which may present only with the symptom of hemoptysis. First line chloroquine therapy has not been found helpful in preventing this complication. Frequent vigilant monitoring is required to detect this. Use of antibiotics or anti-malarial have not helped in improvement of patient outcome. Supportive measures like O₂ supplementation/NIV have only been found useful in such cases. Further studies are needed to evaluate host or environmental characteristics leading to complicated P.Vivax malaria. Research may also be needed to detect any immune complex injury by benign tertian malaria.

REFERENCES

1. White NJ, Breman JG. Malaria and Babesiosis : Diseases caused by red blood cell parasites. In : Braunwald E, Fauci A, Kasper DL et al, (eds). Harrison's Principles of Internal Medicine. 15th edition. New York : McGraw-Hill Inc. 2001; 1: 1203-13.
2. Lomar AV, Vidal JE, Lomar FP, Barbas CV, Matos GJ, Boulos M. Acute respiratory distress syndrome due to vivax malaria: case report and literature review. *Braz J Infect Dis* 2005; 9:425-30
3. Saravu K, Rishikesh K, Kamath A, Shastry AB: Severity in Plasmodium vivax malaria claiming global vigilance and exploration-a tertiary care centre-based cohort study. *Malar J* 2014, 13:304.
4. Chotinavich K, Udomsangpetch R, Pukrittayakamee S, et al. The adhesion receptors of P. vivax-infected red cells (abstract P27). *Exp Parasitol* 2003;105:33-4.
5. Kumar S, Melzer M, Dodds P, Watson J, Ord RP. Vivax malaria complicated by shock and ARDS. *Scand J Infect Dis* 2007;39:255-6.
6. Torres JR, Perez H, Postigo MM, Silva JR. Acute non-cardiogenic lung injury in benign tertian malaria. *Lancet* 1997;350:31-2.
7. Sarkar S, Saha K, Das cS. three cases of ARDS: An emerging complication of Plasmodium vivax malaria (case report). *Lung India* 2010;27:154-7.

I.M.A. G.S.B. NEWS BULLETIN**GUJARAT MEDICAL JOURNAL**

Official Journal of Indian Medical Association, Gujarat State Branch

OFFICE : A.M.A. HOUSE, 2ND FLOOR, OPP. H. K. COLLEGE, ASHRAM ROAD, AHMEDABAD-380 009.

Fax / Phone : (079) 2658 73 70 E-mail : imagsb@gmail.com, gujaratmedicaljournal@gmail.com

Mandatory Submission Form

Title of article :

.....

Specification : Case Report / Original Article / Review Article / Short Communication /

Key Words :

Study / Sponsorship / Grant by (if any)

A signature below certifies compliance with the following statements :**Copyright transfer :**

In consideration of the acceptance of the above work for publication, I do hereby assign and transfer to GMJ all rights, title and interest in and to the copyright in the above-titled work. This assignment applies to all translation of said article as well as to preliminary display/posting of the abstract of the accepted article in electronic form before publication. If any changes in authorship (order, deletions, or additions) occur after the manuscript is submitted, agreement by all authors for such changes must be on file with the Publisher. an author's name may be removed only at his/her request.

Author Responsibilities :**I attest that :**

1. The manuscript is original work/compilation work, without fabrication, plagiarism, or fraud;
2. The manuscript is not currently under consideration elsewhere and the research reported will not be submitted for publication elsewhere unless a final decision is made by journal that the manuscript is not acceptable;
3. I have made a significant scientific contribution to the study.
4. I have read the complete manuscript and take responsibility for the content and completeness of the final submitted manuscript and understand that if the manuscript, or part of the manuscript is found to be faulty or fraudulent, I share responsibility.
5. **No Objection Certificate from the institute / hospital is mandatory, along with the article sent for the publication**
6. The first signed author should be Life member of Gujarat State Branch, I.M.A.

Signature of each author is required in the same order as on the manuscript title page (Fax signatuers, multiple forms are acceptable). For more than 6 authors, use an extra sheet.

Signature (1) GSB IMA Membership No.

Print name Date

Signature (2) Print name Date

Signature (3) Print name Date

Signature (4) Print name Date

Signature (5) Print name Date

Signature (6) Print name Date

Corresponding author Postal Mailing Address

STD Code No. Ph. (O)

(R).(M)

E-Mail

(One can use photocopy of this Mandatory Submission Form)

I.M.A. G.S.B. NEWS BULLETIN

GUJARAT MEDICAL JOURNAL

Official Journal of Indian Medical Association, Gujarat State Branch

OFFICE : A.M.A. HOUSE, 2ND FLOOR, OPP. H. K. COLLEGE, ASHRAM ROAD, AHMEDABAD-380 009.

Fax / Phone : (079) 2658 73 70 E-mail : imagsb@gmail.com, gujaratmedicaljournal@gmail.com

Instructions to Authors

The Gujarat Medical Journal, (GMJ) the official publication of Indian Medical Association, Gujarat State Branch (I.M.A., G.S.B.) welcome original articles, review articles, case reports and short communications of interest to medical fraternity. The official language is English. Articles are accepted on condition that these are contributed solely to the Gujarat Medical Journal and are not submitted elsewhere for publication. The editors reserve the right to reject or edit any article. Articles accepted will be the sole property of the journal and all copyrights will be in the name of I.M.A. G.S.B. The article must be submitted via Mandatory Submission Form, which is printed in this issue of journal. Please note that the manuscripts without forwarding letter and / or incomplete mandatory Submission Form will not be processed.

GMJ has adopted the "Uniform Requirements for Manuscripts (URM) submitted to Biomedical Journals" for its review and publication process. One can refer this on Internet at www.icmje.org

GMJ has now adopted the CONSORT Statement for the uniformity of statistical data presentation, One can refer this statement on www.consort-statement.org also.

Preparation of the Manuscript

The manuscript should be submitted to the editor on computer CD prepared in M.S. Word. However 2 copies of the manuscript printout should also be sent along with the CD. Out of that one should not bear name/names of the author and or institute.

Length of Article

The article submitted for publishing should not exceed the limit. The material should be typed in A-4 SIZE Paper only in double spacing.

Review Article : 4000 words excluding 50 references & abstract upto 250 words.

Original Article : 3000 words excluding 20 references & abstract upto 250 words.

Case Report & Brief Communication : 1000 words excluding abstract upto 150 words

Letter to Editor : 500 words and 5 references.

Title Page

This should include the following:

Title of the article with type of manuscript such as case report, original article or short communication etc. A brief running Title and key words (maximum 3). Author's names with designations, Name and address of the Institution, Name and address for correspondence.

Text

The text should have appropriate subheadings like Introduction, Material and Methods, Observation, Discussion, Acknowledgements etc. Reference numbers should be superscript and not written in parenthesis. Only standard abbreviations should be used and should be preceded by the full form on its first appearance. Generic name of the drugs are preferred to the trade names.

Tables, Figures and Illustrations

Each table and figure should be typed on separate page, be numbered in Roman numerical and have a brief descriptive title. Photographs should be glossy, clear and should be marked at the back with pencil containing name of the articles and author/s and should indicate the top of the figure by an arrow. Colour prints can be made at the author's expense. Legends should be typed on a separate page.

Photographs

Photographs should preferably be Post-card size (6"x4"). GMJ will bear the cost of only two photographs per article. Additional photographs will be included on payment of Rs. 100/- per photograph.

References

References should be in the numerical order in which they are first cited in the text. Please follow the style of Index Medicus for references.

Reprints

First author will receive two complimentary copies of the Journal.

Enquiry : Please contact the IMA GSB office for your queries/enquiry about the status of article submitted.