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

Estd. On 2-3-1945

GUJARAT MEDICAL JOURNAL

INDIAN MEDICAL ASSOCIATION, GUJARAT STATE BRANCH

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GMJ

STATE PRESIDENT AND HON. STATE SECRETARY'S MESSAGE

Dear Friends,

Hope this Gujarat Medical Journal finds you in the best of your health and spirit. While this journal reaches to your hand, it might have completed year 2016 and year 2017 begins with a new Hope. **May this New Year brings joy and prosperity in your life.**

Friends, as you all are aware that the whole medical fraternity is passing through a very hard phase. With the advent of new draconian and absurd laws enforced, sorry state of medical education, rapid commercialization, onslaught of corporate hospitals, falling image we doctors have become a favorite whipping boy of the society, government and press. Nobody is ready to look at the issue comprehensively and address the problems considering the total scenario. Everybody tries to find a temporary and quick solution and it is creating more and more problems.

But working in this field in various capacity and various sector as medical activist, we strongly feel that we can definitely overcome this current turbulent phase. A strong IMA Hospital Board of India can really help in solving the problems of hospitals. Gujarat State Branch, IMA is going to form IMA Hospital Board of India, Gujarat Chapter, which may help to solve the hurdles at various levels.

Apart from this, Gujarat State Branch, IMA has also launched the long awaiting schemes like **Hospital Protection Scheme** and **Family Welfare Scheme**, their details are already published in our previous bulletin & Form has been posted to All Eligible Members.

So on behalf of Gujarat State Branch, I.M.A. office bearers, we request you to become the member of this scheme in large numbers.

Together, Let's All Work to bring back the Glory of Our Profession.

Long Live I.M.A.

DR. YOGENDRA S. MODI
(President, G.S.B.I.M.A.)

DR. KAMLESH B. SAINI
(Hon. State Secy. G.S.B.I.M.A.)

FROM THE DESK OF EDITORS



Dear friends,

We are thankful to all the central council members of GSB IMA for putting their faith, trust and confidence in us and giving the charge of prestigious Gujarat Medical Journal (GMJ) for this year also. On our side, we promise to see that the faith and trust that is put in us is full filled and for that, we shall try our best. GMJ is published since last 70 years. We are well aware that in these years GMJ has carved out its name as a journal of research oriented and academic minded people, in the medical field. All the editors in past, have tried their best to give a name and fame to this journal and we are enjoying their fruits. But we are aware, that increases our responsibility also. Moreover, our journal is an Indexed Journal, Indexed in InMed. We shall have to maintain that standard of our journal. We shall have to work hard and will have to be vigilant.

Due to some unavoidable technical reasons we are forced to published GMJ very late and we regret for that.

Our country and particularly, Gujarat has entered in the field of medical tourism. People from developed and under developed countries come here for treatment and we provide world best treatment to them at a cheaper rates then that is available in developed countries. Apart from big cities of Gujarat like Ahmedabad, Surat. Vadodra and Rajkot-Bhavnagar, even small centers like Anand and Nadiad provide world class treatment in the field of cardiology and nephrology. Our hospitals and expertise are world class and that pushes the medical tourism in Gujarat far ahead. From our own domestic population also we get large number of patients. This provides opportunities for research to our doctors. Now we have better infrastructure facilities for data collection and access to world data, for comparison. It has provided a big boost to research wok in our state. We appeal our colleagues to send their research articles and papers for publication in GMJ. This will help our other colleagues and also government in handling and controlling certain diseases. Government will also be able to determine where more efforts are required.

Without making any compromise with our laid down policy, we have made all the efforts 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. Yogendra Modi and hon. secretary Dr. Kamlesh Saini for encouragement and suggestions and giving us free hand in publication of this journal. We are grateful to GSB past presidents Dr Kirtibhai Patel, Dr. Jitubhai 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 is providing us time to time.

With regards,

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

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

DR. HARSHAD C. PATEL
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CONTENTS

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

Vol. : 11 **DECEMBER-2016** **Issue : 2**

CONTENTS

CASE REPORT

ORIGINAL ARTICLE

Comparative study of Tympanolasty with or without Mastoidectomy in chronic suppurative otitis Media (Quiescent stage)

Kumar Prayatna, Panchal Ajay

KEY WORDS : Tympanoplasty, Cortical mastoidectomy, Chronic otitis media.

ABSTRACT

OBJECTIVE : To compare outcome of tympanoplasty with and without cortical mastoidectomy in cases of, chronic otitis media , tubotympanic (quiescent stage).

STUDY DESIGN: Single-blinded, randomised, controlled study in SMIMER Surat.

METHODS : 70 cases were randomly divided into two groups. In group A, 35 ears underwent type one tympanoplasty alone. In group B, 35 ears underwent type one tympanoplasty along with cortical mastoidectomy. Outcome measures were as follows: perforation closure and graft uptake, hearing improvement.

RESULTS : There is no statistically significant difference in tympanic membrane perforation closure and hearing improvement, comparing the two groups at 6 weeks and 12 weeks post-operatively.

CONCLUSION : Tympanoplasty with cortical mastoidectomy was not found to be superior to tympanoplasty alone over a short term follow-up period. Hence, it may not be necessary to undertake routine mastoid exploration in this stage of disease.

INTRODUCTION

Chronic otitis media is a long standing infection of a part or whole middle ear cleft characterized by ear discharge and permanent perforation .There is not much dilemma about the atticoantral type of (COM) management and pathology, but management of Chronic Otitis Media tubotympanic type (COM) is still not clear. It is well accepted that the main purpose of operation is to obtain permanently dry ear and close the perforation and improve the hearing. The use of cortical mastoidectomy, in which mastoid air cell system is exenterated and disease is cleared keeping posterior canal wall intact, as a means to establish drainage of a complicated infection of the ear has no controversy. Some argue that tympanic membrane perforations should be repaired by tympanoplasty alone in which tympanic membrane and ossicular chain are repaired without operating on mastoid bone, regardless of the status of the mastoid air cell system. While others advocate cortical mastoidectomy coupled with tympanic membrane and ossicular chain repair. Mastoid air cells system forms an air reservoir to minimize pressure fluctuation in middle ear. The functional status of the Eustachian tube has been correlated to the pneumatization of the mastoid air cells by some authors, whereas, others do not agree with it .However, the ears with chronic suppurative otitis media have consistently shown a reduction in the size of mastoid air cell system. The goal is to determine role of cortical mastoidectomy in tubotympanic type of otitis media (quiescent stage) in postoperative results; closure of perforation and hearing improvement.

AIMS AND OBJECTIVES

Otologists currently have two schools of thought regarding the importance of cortical mastoidectomy in the treatment of Tubotympanic chronic otitis media(quiescent stage).Some authors have thought that cortical mastoidectomy is justified in cases of chronic otitis media Tubotympanic type (quiescent stage). Other authors have argued that closure of tympanic membrane perforations by performing tympanoplasty without cortical mastoidectomy is sufficient.

The goal is to the compare between TYMPANOPLASTY and TYMPANOPLASTY WITH CORTICAL MASTOIDECTOMY in tubotympanic type COM (quiescent stage) in postoperative results; in terms of

1. Closure of perforation
2. Hearing improvement

MATERIALS AND METHODS

This study comprises of patients with chronic otitis media tubotympanic type in quiescent stage. Group A included patients operated with Tympanoplasty (type 1) and Group B included patients operated with Tympanoplasty (type 1) +cortical mastoidectomy. Randomization was done on alternate basis of admission. Hearing was assessed preoperatively and postoperatively at 6th and 12th week

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interval by PTA (pure tone audiometry). Hearing parameters used were change in air-bone gap. Air-bone gap was calculated as the average difference between air conduction and bone conduction at 0.5,1,2,4, kHz. Closure of perforation was assessed clinically by Otoscope. All the cases were operated in the department of ENT, SMIMER College Surat.

INCLUSION CRITERIA:

1. Chronic otitis media (Tubotympanic type quiescent stage) with intact and mobile ossicular chain
2. Age group of 15 years to 60 years

EXCLUSION CRITERIA :

1. Chronic otitis media (Atticoantral type)
2. Chronic otitis media (Tubotympanic type active stage)
3. Acute otitis media
4. Previously operated cases
5. Chronic otitis media (Tubotympanic type) with ossicular chain necrosis

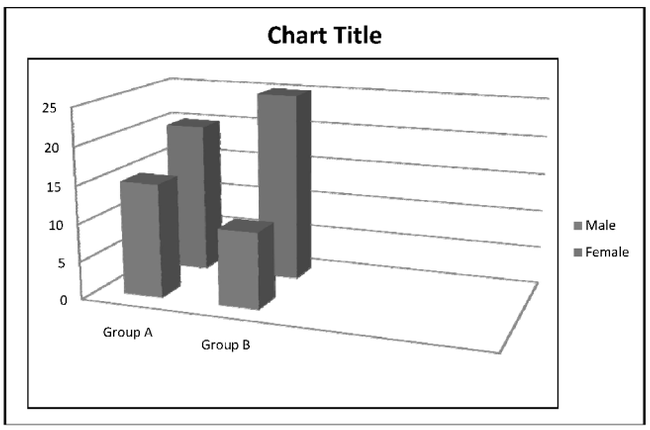
OBSERVATION

The study included 70 patients who were divided randomly into two groups; Group A patients were operated with Tympanoplasty alone included 35 patients where 15 were males and 20 were females. Group B patients were operated with cortical mastoidectomy + tympanoplasty included 35 patients where 10 were males and 25 were females.

Table I

Sex Ration

	Male	Female
Group A	15	20
Group B	10	25



Hearing Improvement

Average preoperative Air-Bone gap was 45.15± 9.91 in group A, whereas it was 45.41± 7.83in group B. Average A-B gap 6 weeks postoperative in group A was 25.00 ±6.07 whereas it was 26.81± 7.63 in group B. Average A-B gap 12 weeks postoperative in group A was 24.75±5.54, whereas it was 26.20± 8.72in group B .There was no statistically significant difference between group A and group B regarding A-B gap difference preoperatively and postoperative. P-value >0.05 i.e. there is no difference in between mean of hearing improvement in group A and B.

Table I I

	Group	N	Mean	Std. Deviation	P-value	95% Confidence Interval of the Difference	
Preop-PTA	B	35	45.4167	7.88353	.901	-3.98764	4.52041
	A	35	45.1503	9.91775			
@6_wk	B	35	26.8681	7.63322	.260	-1.40197	5.12379
	A	35	25.0071	6.07051			
@12_wk	B	35	26.2014	8.72902	.410	-2.01791	4.90640
	A	35	24.7571	5.54410			
Hearing_improveme	B	35	19.0903	6.47720	.606	-3.89750	2.29234
nt_							
	A	35	19.8929	6.59107			

P-value >0.05 i.e there is no difference in between mean of hearing improvement in group A and B.

- **Independent t-test applied**

Table III

Group	Group A	Group B
Hearing Improvement	19.89	19.01

Table IV

Group -A		Mean	Std. Deviation	Mean of difference	P-Value	95% Confidence Interval of the Difference	
						Lower	Upper
Pair 1	Preop_PTA	45.41	7.88	18.54±5.55	P<0.001	16.66	20.42
	@6_wk	26.86	7.63				
Pair 2	Preop_PTA	45.41	7.88	19.21±6.77	P<0.001	16.92	21.50
	@12_wk	26.20	8.72				

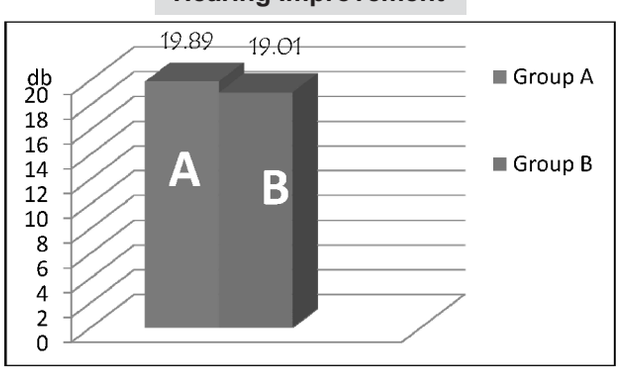
*** paired t-test applied**

Table V

Group -A		Mean	Std. Deviation	Mean of difference	P-Value	95% Confidence Interval of the Difference	
						Lower	Upper
Pair 1	Preop_PTA	45.15	9.91	20.14±8.66	P<0.001	17.16	23.11
	@6_wk	25.00	6.07				
Pair 2	Preop_PTA	45.15	9.91	20.39±8.34	P<0.001	17.52	23.25
	@12_wk	24.75	5.54				

*** paired t-test applied**

Hearing Improvement



Graft uptake :

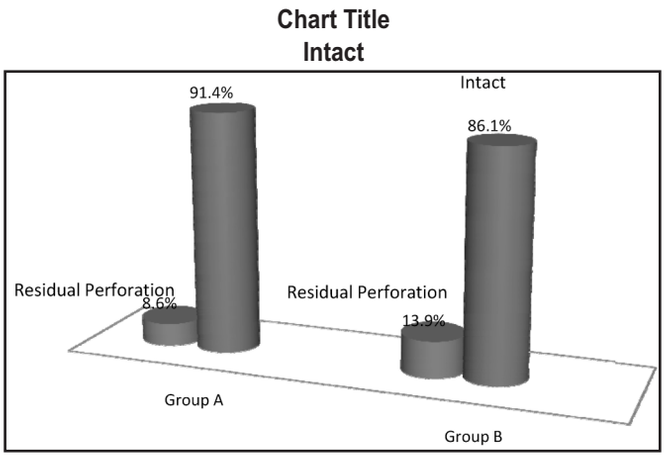
Graft success rates were 91.4% in group A, 86.1% in group B with P value 1.00 There was no statistically significant difference between group A and group B

NeoTM_status * Surgery_Crosstabulation

Table VI

		Surgery_		Total
		Group A	Group B	
NeoTM_ Status	Intact	32	31	63
		91.4%	86.1%	88.7%
	Non Intact	3	4	7
		8.6%	13.9%	11.3%
Total		35	35	70
		100.0%	100.0%	100.0%

*** test of prevalence (Z-test)**
P-value >0.05 i.e there is no difference in between the prevalence of intact in between intact.



Graft Take up rate in Group A and Group B

DISCUSSION

Mastoidectomy is one of the most common otological operations performed today. Indications for Cortical mastoidectomy range from eradication of chronic infection from mastoid air cell system to approaches for various neurotological procedures. Mastoidectomy was first described by Louis Petit in the 1700s, although the concept did not gain wider acceptance until 1958, the cortical mastoidectomy was popularized by William House. This procedure was attempted to avoid the common problems with radical mastoidectomy.

Tympanoplasty is an operative procedure, in which the reconstructive procedure is limited to repair of tympanic membrane perforation. Implicit in the definition is that the ossicular chain is intact and mobile, and the middle ear is disease free. There are a number of studies in the literature highlighting the advantages and disadvantages of performing cortical mastoidectomy in the surgical treatment of mucosal type of chronic otitis media.

Our study emphasizes the fact that overall satisfactory hearing outcome with adequate air–bone closure can be achieved irrespective of cortical mastoidectomy in tubotympanic type CSOM (quiescent stage). We found Average A-B gap preoperative was 45.15± 9.91 in group A, whereas it was 45.41± 7.83in group B. Average A-B gap 6 weeks postoperative in group A was 25.00 ±6.07 whereas it was 26.81± 7.63 in group B. Average A-B gap 12 weeks postoperative in group A was 24.75±5.54, whereas it was 26.20± 8.72in group BThere was no statistically significant difference between group A and group B regarding A-B gap difference pre- and postoperative..

Balyan et al in 1997 did a retrospective study of 323 patients to evaluate the role of mastoidectomy in non-cholesteatomatous CSOM. They observed no statistically significant difference in hearing outcome when mastoidectomy was done.[41]

Mishiro et al in 2001 reviewed 251 cases of noncholesteatomatous chronic otitis media, to determine whether mastoidectomy is helpful when combined with tympanoplasty for these conditions. A total of 147 patients were treated by tympanoplasty with mastoidectomy and 104 were operated on without mastoidectomy. There was no statistically significant difference between the two groups.[39]

Bhat et al in 2008 compared outcomes for cortical mastoidectomy + tympanoplasty and for tympanoplasty alone in cases of quiescent, tubotympanic CSOM.[42] There were no statistically significant differences in hearing improvement. In 2012, Albu et al found that cortical mastoidectomy offers no additional benefit regarding hearing gain over myringoplasty.[43]

In contrast to our study, Jackler and Schindler in 1984 studied 48 patients with chronic otitis media with tympanic perforations who underwent myringoplasty with mastoidectomy. In their study, it was found that simple mastoidectomy was found to be an effective means of re-pneumatizing the sclerotic mastoid and restoring the hearing.

Our study revealed Graft success rates were 91.4% in group A, 86.1% in group B with P value 0.01. There was no statistically significant difference between group A and group B

Sheehy in 1985 recommended performing simple cortical mastoidectomy routinely for all tympanoplasties because it is “good practice” and because “it's better to be safe than sorry.”

Jackler and Schindler in 1984 found that simple mastoidectomy was found to be an effective means of re-pneumatizing the sclerotic mastoid and eradicating mastoid sources of infection. The study concluded that simple mastoidectomy is a safe and useful adjunct to myringoplasty.

McGrew et al in 2004 conducted a retrospective study of patients at a tertiary referral center, where 484 patients who underwent surgical repair of simple tympanic membrane perforations were identified and reviewed. Surgical outcome and clinical course were assessed to compare results of tympanic membrane perforation repair, with and without canal wall up mastoidectomy. They found that tympanic membrane repair was equally effective in both groups at 91%.[37]

Development of persistent ipsilateral otological disease requiring a subsequent ipsilateral procedure was approximately twice as common in the tympanoplasty group. They concluded that mastoidectomy was not necessary for successful repair of simple tympanic membrane perforations. However, mastoidectomy impacted the clinical course in patients by reducing the

number of patients requiring future procedures and by decreasing disease progression. This suggests that combining mastoidectomy with tympanoplasty during repair of simple perforations in patients with no active evidence of infection remains an appropriate option, and may be valuable in reducing the need for future surgery.

On the contrary, Bhat et al in 2008 concluded that there were no statistically significant differences in tympanic perforation closure, graft uptake, or disease eradication, comparing the two groups at 3 and 6 months postoperatively. Mastoidotympanoplasty was not found to be superior to tympanoplasty alone over a short-term follow-up period. [42]

Albu et al in 2012 presented a paper of 320 consecutive adult patients treated by either myringoplasty with cortical mastoidectomy or myringoplasty only. They found that three factors were significant in predicting success rate, that is, healthy opposite ear, a long dry period preceding the operation, and non-smoker status. The only factor attaining significance in the multivariate analysis was a dry period longer than 3 months. They concluded that cortical mastoidectomy offers no additional benefit in myringoplasty performed on patients with persistent or intermittent discharging CSOM and no evidence of cholesteatoma or mucosal blockage within the antrum. [43]

Comparison of Graft uptake rate with other studies and our study

Group A:Patient operated with Tympanoplasty

Group B: Patients operated with cortical mastoidectomy + tympanoplasty.

Table VII

Study	Group A	Group B
Mishiro et al	93.3%	90.5%
Balyan et al	90.5%	85.75%
Kamath et al	91.5%	90.3%
McGrew BM et al	91%	91%
Albu S, et al	76%	82.8%
Our study	91.4%	86.1%

Comparing Hearing improvement with other study

Group A:Patient operated with Tympanoplasty

Group B: Patients operated with cortical mastoidectomy + tympanoplasty.

Table VIII

Study	Hearing Improvment	
	Group A	Group B
Kamath MP et al	12.40	12.88
Balyan FR1 et al	17.2	20.1
Mishiro et al	20	20
Our study	19.89	19.01

CONCLUSION

We concluded at the end of study that, tympanoplaty alone is sufficient for chronic otitis media tubotympanic variety quiescent stage .

There is no any significant difference in closure of perforation and hearing improvement after tympanoplasty alone and tympanoplasty with cortical mastoidectomy.

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ORIGINAL ARTICLE

Peptic Ulcer Perforation (Pulp) score –predictor of mortality following Peptic Ulcer Perforation

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KEY WORDS : Peptic Ulcer, Pulp, As a Score, Surgery, Mortality

ABSTRACT

BACKGROUND - Accurate and early identification of high risk surgical patients with perforated peptic ulcer is important for triage and risk stratification. The objective of the present study was to replicate and re-evaluate the results of this score in our setup, as compared to the cohort study carried out in Denmark. **METHOD -** A study was conducted on 50 surgically treated patients for peptic ulcer perforation for period of one year.There was no age restriction. **OUTCOME MEASURE:** Predefined outcome measure was mortality within 30 days of surgical procedure for peptic ulcer perforation. VARIABLES USED: age, co-morbid disease, liver cirrhosis, steroids use, shock on admission, time lapse between perforation and admission, serum creatinine, ASA Score 2-5. **RESULTS -** A total of 50 patients were studied in single hospital over period of one year.Nearly 12% (6 patients)died within 30 days of surgery. Out of total deaths, most of deaths occurred in the patients with PULP score more than 7 and the risk of mortality increases with increase in PULP Score. So the risk stratification is classified into low and high risk. **CONCLUSION -** PULP Score can be used to predict 30-day mortality in patients operated for peptic ulcer perforation almost accurately and thus assist in risk stratification and triage.This study hencereplicates the cohort study in Denmark in our setup.

BACKGROUND

There is substantial mortality and morbidity in cases of perforated peptic ulcer and mortality around 10-15% have been reported in different studies. Number of prognosticfactors and clinical predictions for morbidity and mortality in cases of peptic ulcer have been reported, likeBoeyscore and ASA Score. At present no such clinical predictions are used extensively in clinical practice in cases of peptic ulcer perforation.Early and accurate identification of high risk patients of peptic ulcer perforation canhelp in risk stratification and triage e.g. any preoperative specific care, post operative ICU care, preoperative respiratory and circulatory stabilization, specific monitoring, etc.

AIM

To replicate and re-evaluate the results of this score in our setup, as compared to the cohort study carried out in Denmark.

METHOD

A study was conducted on 50 surgically treated patients for peptic ulcer perforation for period of one year.There was no age restriction.OUTCOME MEASURE: Predefined outcome measure was mortality within 30 days of surgical procedure for peptic ulcer perforation.VARIABLES USED: age, co-morbid disease, liver cirrhosis, steroids use, shock on admission, time lapse between perforation (beginning of symptoms) and admission, serum creatinine, ASA Score 2-5.

Assignment of points according to Peptic ulcer perforation score

VARIABLE	POINTS
Age >65 years	3
Co-morbid active malignant disease or AIDS	1
Co-morbid liver cirrhosis	2
Concomitant use of steroids	1
Shock on admission (BP<100mmhg, Pulse >100/min)	1
Time from perforation (beginning of symptoms)	1
to admission >24h	
Serum Creatinine>1.5mg/dl	2
ASA Score 2	1
ASA Score 3	3
ASA Score 4	5
ASA Score 5	7
Total PULP Score	0-18

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ASA SCORE

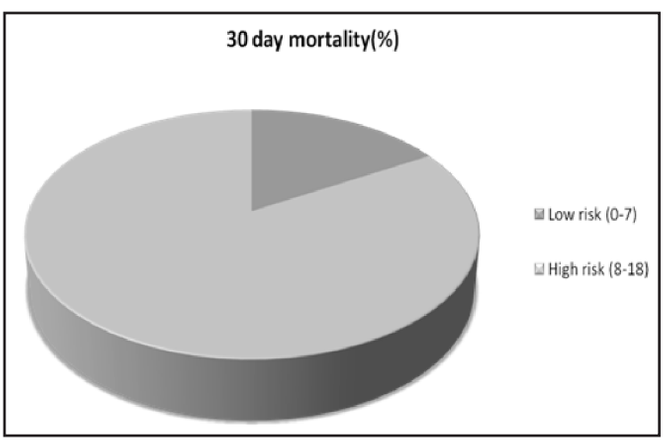
ASA SCORE	Description
1	Healthy Patient
2	Mild systemic disease
3	Severe systemic disease
4	Severe systemic disease that is constant threat to life
5	A moribund patient who is not expected to survive without operation

RESULT

A total of 50 patients were studied in single hospital over period of one year.Median age was 46 yrs (19-83 years). 12% (6 patients) died within 30 days of surgery.In the present study no patient scored 0,1,17, 18 PULP score. Out of total deaths, most of deaths occurred in the patients with PULP score more than 7 and the risk of mortality increases with increase in PULP Score.So the Risk stratification is divided into two classes:-

Risk of mortality	PULP SCORE
Low Risk	0-7
High Risk	8-18

Risk Stratification according to PULP Score



DISCUSSION

In the present study on PULP Score which was aimed to replicate and evaluate the score used in Cohort Study in Denmark; we studied around 50 patients operated for peptic ulcer for 30 day mortality. All these operated patients were without any intra-operative complications.As the PULP Score increases there is increase in mortality and morbidity as well.ASA Score is subjective phenomenon but ASA Score more than 3 has increased risk of mortality.ASA Score more than 3, age older than 65, co-morbid liver cirrhosis, and elevated serum creatinine had got highest prognostic impact.The cohort study carried out in Denmark used a large number of patients over a period of some years, but this study was carried out over a period of one year in single hospital on small sample size.The study carried out in Denmark had 27% mortality and most of them had PULP score above 7. This study has 12% mortality. Patients with low risk(0-7 score) have 3.33% mortality and patients with high risk(8-18 score) have 25% mortality.PULP Score combines readily available predictors of patient's baseline health status and acute disease severity with ASA scoring system.Based upon risk of dying within 30 days of surgery, we could classify patients as low and high risk as mentioned earlier.PULP score can thus assist in risk stratification and triage of patients with peptic ulcer perforation like preoperative respiratory and circulatory stabilization, post operative ICU Stay, specific monitoring and certain peri-operative care.

CONCLUSION

This study demonstrates PULP Score can be used to predict 30-day mortality in patients operated for peptic ulcer perforation almost accurately and thus assist in risk stratification and triage.This study thus replicates the cohort study in Denmark in our setup.

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ORIGINAL ARTICLE

Comparative Study Between Laparoscopic Hernia Repair Vs Open Hernia Repair

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KEY WORDS :

BACKGROUND

Despite a large number of clinical studies in recent years no consensus has been achieved on the surgical technique of inguinal hernia repair for various reasons. "Experts" believe that their own preferred open methods have the lowest possible recurrence and complication rates. This review article aimed to compare Laparoscopic Hernia Repair versus Open Hernia Repair.

INTRODUCTION

Repair of inguinal hernia is one of the commonest surgical procedures worldwide.

There are three important landmarks in the history of repair of inguinal hernia.

1. Tissue repair Eduardo Bassini 1888
2. Onlay mesh Irving Lichtenstein1984 [tension-free] repair
3. Laparoscopic Ger, Shultz, hernia repair Corbitt etc. 1990

AIMS

The aim of this study was to compare the effectiveness and safety of laparoscopic and conventional open repair in the treatment of inguinal hernia.

The following parameters were evaluated for both laparoscopic and open procedures.

- Patient selection criteria
- Operative technique
- Operating time
- Intra-operative and postoperative complications
- Postoperative pain and amount of narcotics used
- Postoperative recovery
- Recurrence
- Bilateral assessment and treatment.
- Cost effectiveness
- Learning curve

MATERIALS

A literature review was performed & criteria for selection of literature were the number of cases at-least 25 each, methods of analysis, operative procedure. We have considered 25 cases each of laparoscopic and open hernia repair.

Method of patient selection

Anesthetic consideration:

The general anesthesia and the pneumoperitoneum required as part of the laparoscopic procedure do increase the risk in certain groups of patients. Patients with Cardiac diseases and COPD should not be considered as a good candidate for laparoscopy. The laparoscopic hernia repair may also be more difficult in patients who have had previous lower abdominal surgery. The elderly may also be at increased risk for complications with general anesthesia combined with pneumoperitoneum.

Various Operative techniques available:

1. Open suture repair of inguinal hernia

Following methods of suture repair of inguinal hernia is practiced:

- Bassini's repair
- Halsted repair
- Tanner [relaxing incision to reduce suture line tension]
- McVay repair
- Shouldice's repair

1. Open mesh repair of inguinal hernia

PPM remains most popular both in open and laparoscopic surgery. Recently some of the prosthetic biomaterials have been combined together to form various composite mesh in an attempt to minimize the undesirable side effects. Ingrowths of fibrous tissue and collagen provide strength to the repair.

2. Tension-free repair of inguinal hernia

Tension free repair requires a mesh. Placement is either by open anterior, open posterior approach or by laparoscopic means.

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a. Giant prosthetic reinforcement of the visceral sac [GPRVS], Reni Stoppa

b. Lichtenstein Onlay patch repair

c. Patch and plug repair

d. Kugel patch

3. Laparoscopic hernia repair

The present day techniques of laparoscopic hernia repair evolved from Stoppa's concept of pre-peritoneal reinforcement of fascia transversalis over the myopectineal orifice with its multiple openings by a prosthetic mesh. In the early 1990's Arregui and Doin described the trans-abdominal pre-peritoneal repair [TAPP], where the abdominal cavity is first entered, peritoneum over the posterior wall of the inguinal canal is incised to enter into the avascular pre-peritoneal plane which is adequately dissected to place a large [15 x 10 cm] mesh over the hernial orifices. After fixation of the mesh, the peritoneum is carefully sutured or stapled. TAPP approach has the advantage identifying missed additional direct or femoral hernia during the first operation itself.

Around the same time Phillips and McKernan described the totally extra-peritoneal [TEP] technique of endoscopic hernioplasty where the peritoneal cavity is not breached and the entire dissection is performed bluntly in the extra-peritoneal space with a balloon device or the tip of the laparoscope itself. It appears to be the most common endoscopic repair today.

Relative contraindication for laparoscopic approach:

a. Obesity with BMI >30

b. Significant chest disease

c. Patient on anticoagulants

d. Adhesions

e. Massive hernias

f. Pregnancy

g. Unfit for GA

Inguinal hernia repair in obese patients:

Operations in patients with BMI above 27 may be difficult for less experienced surgeons, particularly when trying to encircle an indirect sac. Patients with BMI of above 30 should be encouraged to lose weight or should even be turned down for the laparoscopic approach.

Inguinal hernia repair in recurrence:

Generally, the short-term recurrence rate of laparoscopic inguinal hernia repair is reported to be less than 5%. In both the open and laparoscopic repair procedures, the aim is to cover the whole inguino-femoral area by a pre-peritoneal prosthetic mesh, and recurrences should not occur.

Operating time:

Operating times of surgical techniques varies between surgeons and also vary considerably between centers. It reduces with experience. It is less important to the patient than a successful operation. The operative time to perform unilateral primary inguinal repair has frequently been reported as longer for laparoscopic compared to open repair.

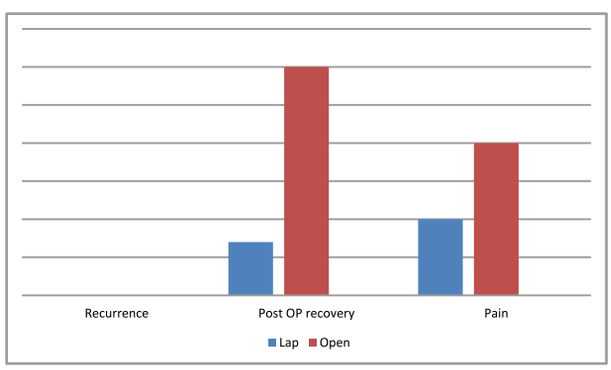
Postoperative pain and amount of narcotics used:

Assessing post-operative pain between open tension-free repairs and laparoscopic repairs, report less pain in the laparoscopic groups. In many cases this also results in less analgesia being consumed by the patient.

Table-I

Out of 25 Each Patients	Recurr-ence	Post Op Recovery (in days)	Pain*
Open Hernia	0	4-5 days on avg.	4-5 on avg.
Lap Repair	0	1-2 days on avg.	1-2 on avg.

*with 1, being minimal pain and 10, being severely painful



Complication rates:

Complications in laparoscopic inguinal hernia surgery are more dangerous and more frequent than those of open surgery, especially in inexperienced hands and hence are best avoided.

Complications of laparoscopic repair of inguinal hernia can be divided into:

Intra-operative complications and precaution to avoid these complications

1. During creation of pre-peritoneal space: This is the most important step for beginners.

- A wide linea alba may result in breaching the peritoneum.

- Improper placement of balloon trocar causing dissection of muscle fibers
- Entry into peritoneum causing pneumoperitoneum
- Rupture of balloon in pre-peritoneal space
- The Hassan's trocar must snugly fit into the incision to avoid CO 2 leak

2. Precautions during port placement

The trocars should be short and threaded in proportion to less workspace and to ensure a snug fit respectively. The patient should empty their bladder before surgery as the suprapubic trocar could injure a filled bladder. The pressure in the pre-peritoneal space must be such as to offer sufficient resistance during trocar insertion to avoid puncturing the peritoneum.

3. Correct identification of the anatomical landmarks

The next most important and crucial step in any hernia surgery is the correct identification of anatomical landmarks. This is difficult for beginners as the anatomy is different from that seen in open surgery. The first most important step is to identify the pubic bone. Once this is seen, the rest of the landmarks are traced keeping this as reference point.

4. Bladder injuries

Bladder injury most commonly occurs during port placement, dissecting a large direct sac or in a sliding hernia. It is mandatory to empty the bladder prior to an inguinal hernia repair to avoid a trocar injury. The diagnosis is evident when one sees urine in the extra-peritoneal space.

5. Bowel injuries

Bowel injury is rare during hernia surgery. It can occur when reducing large hernias. Injury is best avoided in such circumstances by opening the hernial sac as close as possible to the deep ring.

6. Vascular injury

This is one of the commonest injuries occurring in hernia repair and often a reason for conversion. The various sites where it can occur is rectus muscle vessel injury during trocar insertion; inferior epigastric vessel injury; bleeding from venous plexus on the pubic symphysis; aberrant obturator vein injury; testicular vessel injury; and the most disastrous of all, iliac vessels, which requires an emergency conversion to control the bleeding and the immediate services of a vascular surgeon to repair the same.

7. Injury to vas deferens

Injury occurs while dissecting the hernia sac from the cord structures. The injury causes an eventual fibrotic narrowing of the vas. A complete transaction of the vas needs to be repaired in a young patient. An injury to the vas is best avoided.

8. Pneumoperitoneum

It is a common occurrence in TEP which every surgeon should be prepared to handle. Putting the patient in Trendelenburg position and increasing the insufflation pressures to 15 mmHg helps. If the problem still persists, a Veress needle can be inserted at Palmer's point.

Postoperative complications

1. Seroma / hematoma formation

It is a common complication after laparoscopic hernia surgery, the incidence being in the range of 5-25%. They are specially seen after large indirect hernia repair. Most resolve spontaneously over 4-6 weeks.

2. Urinary retention

This complication after hernia repair has a reported incidence of 1.3 to 5.8%. It is usually precipitated in elderly patients, especially if symptoms of prostatism are present. These patients are best catheterized prior to surgery and catheter removed the next day morning.

3. Neuralgias

The incidence of this complication is reported to be between 0.5 and 4.6% depending on the technique of repair. The intraperitoneal onlay mesh method had the highest incidence of neuralgias in one study and was hence abandoned as a form of viable repair. The commonly involved nerves are lateral cutaneous nerve of thigh, genitofemoral nerve and intermediate cutaneous nerve of thigh. They are usually involved by mesh-induced fibrosis or entrapment by a tack.

4. Testicular pain and swelling

It occurs due to excessive dissection of a sac from the cord structures, especially a complete sac. Reported incidence is of 0.9 to 1.5%. Most are transient.

5. Mesh infection and wound infection

Wound infection rates are very low. Mesh infection is a very serious complication and care must be taken to maintain strict aseptic precautions during the

entire procedure. Any endogenous infection must be treated with an adequate course of antibiotics prior to surgery.

6. Recurrence

It is the most important endpoint of any hernia surgery. It requires a proper and thorough knowledge of anatomy and a thorough technique of repair to help keep the recurrence in endoscopic repair to a minimum.

Postoperative recovery:

Marked variations are seen in post-operative recovery due to patient motivation, post-operative advice, and existing co-morbidity. Nevertheless all trials reporting this as an endpoint of study show a significant improvement in the laparoscopic group, with no real difference between the TAPP and TEP groups. [Table-1]

Recurrence

Recurrence rates are low with the use of mesh and not significantly different between open or laparoscopic techniques. [Table-1]

Causes of recurrence in laparoscopic inguinal hernia repair

1. Mesh size: The mesh size should be adequate to cover the entire myopectineal orifice. The established size in 2006 is 15 cm x 10 cm per unilateral hernia, with minor deviations.
2. Mesh material: The mechanical strength of available meshes exceeds the intra-abdominal peak pressures and by far even the lightweight meshes are strong enough for inguinal repair. The new macro porous compound meshes present both the successful reduction of the overall foreign body amount and the preservation of mesh elasticity after the scar tissue ingrowths, due to very limited shrinkage and reduced bridging effect.
3. Fixation of the mesh: In the early years of laparoscopic hernia repairs, a strong fixation seemed to be the most important factor in prevention of recurrence. With growing size of the mesh and true macro porous materials being used, the belief in strength reduced and gave way to the concern of acute / chronic pain possibly caused by fixation.
4. Technical experience: The long learning curve of endoscopic repairs contains the potential risk of technical errors leading to unacceptable rise of recurrence rate.
5. Collagen status: Inborn or acquired abnormalities in collagen synthesis are associated with higher incidence of hernia formation and recurrences.
6. Other factors: The negative effect on healing in hernia repair is often related with malnutrition, obesity, steroids, type II diabetes, chronic lung disease, jaundice, radiotherapy, chemotherapy oral anticoagulants, smoking, heavy lifting, malignancy and anemia. Laparoscopic inguinal hernia repair offers excellent results in experienced hands.

Cost effectiveness:

It is suggested that laparoscopic hernia repair is more expensive to perform than open hernia repair. The primary reason for this relates to the cost of extra equipment used for the laparoscopic repair with secondary costs attributed to perceived increases in operating time for the laparoscopic procedure.

Learning curve:

This period represents the developmental and learning curve for the consultant and the senior registrars. There is steep learning curve for laparoscopic repair. Initially everyone used to fix mesh with staples, but nowadays many surgeons are using sutures for it.

DISCUSSION

The TAPP operation represents an excellent alternative for primary inguinal hernia repair. Laparoscopic repair compared favorably with Lichtenstein repair for primary indirect and direct hernias, and unilateral and bilateral recurrent hernias, but was inferior for primary bilateral hernias. General anesthesia and higher costs are reasonable compromises for a shorter period of discomfort in patients with a low ASA index and busy job/sport activity.

There are minimum complication with Open Lichtenstein hernia repair, in terms of intraoperative and postoperative complications and short-term recurrence. In fact with extra care, complications can be nearly avoided. The laparoscopic operations caused significantly less pain in the early postoperative period, leading to earlier mobilization and earlier return to work than open mesh repair. Furthermore, laparoscopic TEP repair is associated with greater patient satisfaction and better cosmetic results than its open counterpart. On the basis of these early experiences, laparoscopic extra-peritoneal hernia repair seems to be as good as, if not superior to, the existing open Lichtenstein repair in terms of postoperative pain, hospital stay, return to work, and cosmoses provided the long-term recurrence rates also are comparable. It is possible to achieve high standards even during the

learning phase of the surgeon if there is strict adherence to the protocols. The TEP technique took no longer to perform, and was associated with less postoperative pain, a shorter period of sick leave and a faster recovery, compared with open Lichtenstein hernia repair.

TAPP is an easier procedure to learn and is less expensive than TEP repair done with balloon dissectors and their ports; however, the reverse is true if no balloon dissectors and staples are used during TEP repair. TEP repair has a longer learning curve.

Laparoscopic hernia repair may not be more expensive than open repair in terms of direct hospital costs or where a difference exists, this is relatively small. Societal costs due quicker recovery and return to employment show clear advantages for the laparoscopic repair and although not currently evaluated in detail, the reduction in chronic groin pain after laparoscopic repair is likely to lead to savings in both direct hospital costs and societal costs.

At present, the laparoscopic repair of hernias finds its clinical niche in patients with bilateral or recurrent hernias or in patients with unilateral hernia who desire a minimal period of postoperative disability.

Open hernia repair requires an incision at the point of maximum weakness, dividing of muscle and then suturing to repair the defect. This damage must heal before the wound become comfortable. Type of anesthetic used to affect the repair does not affect the period of discomfort. In a laparoscopic repair no incision is made in the groin. The small wounds which are made heal rapidly and have been shown to cause negligible post-operative pain. Further mesh is placed inside the groin muscle in the pre-peritoneal layer and this seems a more logical position to prevent peritoneal contents bulging out of a muscle defect than placing a mesh on the outside of the defect. Laparoscopic repair has no surgical weakness postoperatively.

As per current guidelines:

1. Patient should be given a choice of open and laparoscopic repair of hernia in all suitable cases i.e., even in primary unilateral inguinal hernias.
2. Laparoscopic hernia repair should be performed only by appropriately trained surgeons.
3. Patients should be told about TAPP and TEP repair and their risks so they choose an appropriate procedure
4. For repair of recurrent and bilateral inguinal hernia, laparoscopic repair should be considered
5. When laparoscopic surgery is undertaken for inguinal hernia, the totally extra peritoneal [TEP] procedure should be preferred

Recommendation:

The important points to be kept in mind during the surgery are:

- After dissecting direct sac, all peritoneal adhesions around the margin of the defect should be meticulously lysed.
- Always search for an indirect sac, even if a direct hernia has been reduced.
- Reflect the peritoneum off the cord completely
- Place an adequate size mesh to cover the myopectineal orifice completely, preferably the size of 15 x 15 cm.
- The lower margin of the mesh must be comfortably placed - medially in the retro pubic space and laterally over the psoas muscle.
- Perform a 2-point fixation of the mesh on the medial aspect over the Cooper's ligament.
- Avoid cutting of the mesh over the cord. This weakens the mesh and provides a potential site for recurrence.
- Ensure adequate hemostasis prior to placing the mesh.
- The most important factor is the adequate training and learning of the right technique.

Conclusion and recommendations:

Laparoscopic hernia repair is safe and provide less post-operative morbidity in experienced hands and definitely have many advantages over open repair. For bilateral and recurrent inguinal hernias laparoscopic approach is recommended. Nowadays for primary inguinal hernia also it is recommended.

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ORIGINAL ARTICLE

Comparision of intramuscular ketamine and combination of ketamine and dexmedetomidine for paediatric sedation in MRI studies

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KEY WORDS : Paediatric sedation, intramuscular, dexmedetamidine.

ABSTRACT

AIM : To compare the efficacy of intramuscular ketamine and mixture of ketamine and dexmedetomidine for paediatric sedation in MRI studies.

METHOD : 60 Children with ASA 1 and 2 were included in our randomised control study and they are divided into two groups. Group K received intramuscular (IM) ketamine 4 mg/kg and Group DK received IM ketamine 3 mg/kg and dexmedetomidine 1.5 ug/kg. We have observed onset of sedation, duration of sedation, recovery time,and vital signs like HR, SPO2, RR and adverse events. Quality assurance data included identification of adverse events which included bradycardia,tachycardia,and oxygen desaturation (< 92%) and need for cardiopulmonary resuscitation.

RESULTS : Sixty children received consecutive IM injections of two drugs and successfully completed imagingstudy. There was no statistical difference between two groups in terms of demographic data.The onset of sedation (6.27±0.87 min) and recovery time(72±5.35min) in Group K were more than Group DK (4.8±0.66 min) and (55.17±5.8min) respectively.Duration of sedation was more in Group K(51.73±4.96 min) as compared to group DK(34.17±6.03 min).After onset of effect , group DK had significantly lower HR as compared to patients in group K ($p<0.01$).No significant change in RR was seen in both the groups. Total incidence of side effects in Group K was 32.9% while in Group DK was 0%.

CONCLUSION : In paediatric sedation for MRI studies, combination of dexmedetomidine and ketamine is more favourable than ketamine alone regarding time of onset, duration of sedation, side effect and hemodynamic stability.

INTRODUCTION

MRI is a non invasive, radiation free diagnostic procedure. MRI scan takes about 10 – 30 min, depending on diagnostic procedure. For accurate diagnosis, patient has to stay motionless. It is challenging for anaesthesiologist to give sedation without compromising airway and hemodynamic instability. Remote location of MRI centre requires more vigilance especially in paediatric patients. The ideal sedative should be administered by simple nonsophisticated technique and produce adequate sedation conditions while minimizing the incidence of adverse events. Benzodiazepines, ketamine and Propofol are commonly use drug during MRI sedation.Dexmeditomidine (dexmed) a highly selective α2 adrenoceptor agonist, may be useful for paediatric sedation in variety of clinical situations.1

Intravenous (IV)Dexmed has fast onset and rapid recovery but patients are easily aroused by minor stimulation which leads to movement during scanning 2.Apart from that most common and serious side effect of IV Dexmed is bradycardia3.Intramuscular administration of Dexmed might avoid this complication. Ketamine also has fast onset but recovery period is longer and tachycardia more common with ketamine. Combination of both drug might counterbalance the disadvantage of eachother.4So the study was design to compare the sedative effect of Ketamine and Dexmed- ketamine combination given intramuscularly in paediatric patients for MRI.

METHOD AND MATERIALS

Sixty patients betweenage groups of 1 – 5 yr, ASA grade I and II planned for elective MRI studies are included in our study after taking written informed consent from parents / guardians of childrens. Patient with active respiratory tract diseases, known allergy or hypersensitivity reaction to dexmed, general contraindication for MRI (like cardiac pacemaker, ferro-magnetic implants etc.) renal or hepatic

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disease, cardiovascular diseases and history of taking cardiac drugs, increased ICP, increased IOP, Head injury,Seizures were excluded from study. Patient were randomly allocated into two groups .Group K receiving IM ketamine 4 mg/kg and Group DK receiving combination of IM ketamine 3mg/kg and Dexmed 1.5 ug/kg

Parents were instructed to keep NBM for 6 hrs for solids and for 2 hrs for clear liquids upto 2 hrs prior to appointment of MRI. Preprocedure detail history, examination & routine blood investigation checked. Measurement of HR, RR & SPO2 was done. All sedatives were mix with 0.9% NS in syringe to make total volume of 2 ml, then injection was given. IM injection given in lateral upper thigh muscle using 25G needle. Sedation level were assess with Ramsay sedation scale (RSS). Time of onset of sedation,duration ofsedation, recovery time & adverse effect were noted. When RSS was 5, pt was taken for MRI study. IC was inserted and inj glycopyrolate and inj ondansetron were given intravenously (iv). We have observed HR, RR &SPO2 atbaseline, 5min interval throughout the procedure (upto 30 min) thenat 45min, 60 min & 75 min.

The time to sedation is defined as the time in minutes from administration of sedative to achievement of adequate sedation (RSS 5) of pt.

Duration of sedation is defined as time from onset of sedation to offset of sedation (RSS 2).

Time to discharge is time from introduction of sedation to fulfilment of the discharge criteria (Alderte score 8 or greater).

Bradycardia and Tachycardia aredefined as 20% change in HR from baseline value.

SPO2 < 92%,emergencereaction, vomiting & other side effect were recorded.

MRI image quality of each examination was assessed using the following five grade scale: Grade 0 or 1: was applied if the examination was of no or very little diagnostic usefulness because of extensive motion artefacts. Grade 2: allowed us to make the diagnosis, but some motion artefact were still present. Grade 3 and 4: include a good or excellent image quality, with no or almost absent motion artefact.

STATISTICAL ANALYSIS

Data is collected and entered into SPSS V20. Continuos data are expressed as mean ±SD form while non continuous data are expressed in frequency (or in %). Independent t test and Mann whitney test are applied for the calculation of p value for continuosdata. Chi square test is applied for the categorical data for the calculation of p value.

OBSERVATION AND RESULTS

This is randomized control study of 60 paediatric patients of age group 1-4 years planned for elective MRI.Effect of intramuscular Ketamine or Ketamine Dexmedetomidine combination for sedation was observed. All 60 patients had successfully completed the study.

There was no statistical difference between two groups in terms of demographic data including age, sex, weight. [TABLE1]. Average imagines time in both the group is21±2.9 minutes.

The onset of satisfactory sedation (RSS 5)was significantly earlier in group DK (4.8±0.66 min) compared to group K (6.27±0.87 min[TABLE 2].While duration of sedation was less in group DK(34.17±6.03) then group K(51.73±4.96) [TABLE 2].Radiologist were significantly very satisfied with group DK regarding quality of MRI & continuity of procedure. Time of discharge was also earlier (55.17±5.8min) in combination group compared to ketamine alone (72±5.35min). [TABLE 2].

In 5th to 60th minutes patients of group DK had significantly lower HR compared to patients in group K ($p<0.01$).On the contrary patients of group K experienced tachycardia after 15 min. [TABLE 4].None of the patients in both the group observed bradycardia.

No significant change in RR was seen in both the group after 5 min.

None of the patients of group DK observed any side effects while incidence of 13% of vomiting,10% nausea and 9.9% of emergence phenomenon seen in group Kunene of the patient in both the groups observed oxygen desideration(SPO2 <92%) and no one required hospital admission.

DISCUSSION

In recent years a growing number of paediatrics with complex medical conditioning has proposed the MRI for diagnosis. To ensure motionless conditions during imaging paediatric patients commonly requires sedation. Main goal of paediatric sedation is anxiety relief; pain control and control of excessive motion.Midazolam, Ketamine and Propofol are routinely used in paediatricsedation. Dexmed a highly selective α2 adrenoceptor agonist, may be useful for paediatric sedation in variety of clinical situations.[1]

Intravenous Dexmed requires repeated administration &hemodynamic instability.[2] On using high dose of dexmed as a sole sedative cardiovascular side effects were noted[3] and movement was noted with low dose of dexmed. Ketamine as a sole agent for sedation also has its own side effects like tachycardia and emergence, nausea, vomiting, secretions.

The studies regarding IM dexmed and use of IM dexmed with other agents are less.IM dexmed administered with ketamine, which is routine agent may counterbalance the side effects of each other.[4] In this study doses used for ketamine were 3mg/kg in group DK and 4mg/kg in group K.[4]

All 60 patients in this study had successfully completed the study. A case series described in 3 mechanically ventilated children with trisomy 21 and obstructive sleep apnoea the use of IV dexmed with Ketamine for MRI.The regimen was successful and achieving sedation to complete MRI without significant effect on hemodynamics and respiratory rate.

In this study patients given IM dexmed ketamine combination showed less adverse effect, high radiological satisfaction compared to IM ketamine used alone.

Onset of desire sedation was earlier with combination of dexmed-ketamine than ketamine alone. This might be due to less dose required in combination group.

Dexmed has an α2 agonist effect on sympathetic ganglia and produces dose dependent decrease in heart rate.[7]In this study hemodynamic status was stable without significant changes in the DK group & tachycardia is noted with ketamine alone. The hemodynamic side effects of dexmed and ketamine are opposite to each other. Dexmed provide counterbalance to sympathetic stimulation associated with ketamine.[6,7],Also it helps to attenuate post anaesthetic delirium with ketamine.[8]

Tarek F. Tamman also reported that combination of IM dexmed and ketamine with regard to sedation, onset& hemodynamic stability.[4] In this study none of the patient had respiratory depression or oxygen desideration.

13.3% of incidence of vomiting & 10% of nausea is seen with ketamine group while none with combination group. Vomiting is common in patients receiving ketamine.[12] Green et al. reported the incidence of vomiting to be 3.5% in children aged < 5years.On other hand nausea and vomiting are rare side effect of dexmed. Also incidence of emergence phenomenon which is more with ketamine, counteracted by dexmed-ketamine combination.

IM dexmedetomidine in combination with ketamine might have useful application for paediatric sedation in terms of quality of sedation in short non invasive,non painful procedure as they counterbalance disadvantages of each other,6different doses combinations should be compared.

Table : I DEMOGRAPHIC DATA

	GROUP DK	GROUP K	SIGNIFICANCE
Age	2.81±1.46	3.04±1.51	0.55 (NS)
Sex: Male	24 (80%)	23 (76.66%)	1.00 (NS)
Female	6 (20%)	7 (23.33%)	
Weight	14.20±3.52	13.27±3.06	0.28 (NS)

Table : II CLINICAL OUTCOME CHARACTERISTICS

PARAMETERS	GROUP DK	GROUP K	SIGNIFICANCE
Onset	4.80±0.66	6.27±0.87	<0.01*
Duration	34.17±6.03	51.73±4.96	<0.01*
Recovery	55.17±5.80	72±5.35	<0.01*

Table : III TYPE OF MRI EXAMINATION

TYPE	GROUP DK	GROUP K
MRI BRAIN	16	19
MRI COCHLEA	06	06
MRI SPINE	07	04
MRI ORBIT	01	01

Table : IV MEAN VALUES OF INTRA PROCEDURAL MONITORING OF HEART RATE

TIME	GROUP DK	GROUP K	SIGNIFICANCE
0 min	102.53±11.36	107.47±14.33	0.15 (NS)
5 min	97.10±10.57	114.87±15.19	<0.01*
10 min	93.93±10.65	112.43±14.99	<0.01*
15 min	92.20±10.40	123.50±12.72	<0.01*
20 min	91.37±9.79	126.07±12.35	<0.01*
25 min	91.1±10.50	123.97±12.30	<0.01*
30 min	91.10±10.45	123.97±12.17	<0.01*
45 min	90.2±8.75	121.77±11.15	<0.01*
60 min	89.13±8.20	119.77±11.38	<0.01*

Table : V MEAN VALUES OF INTRA PROCEDURAL MONITORING OF RESPIRATORY RATE

TIME	GROUP DK	GROUP K	SIGNIFICANCE
0 min	21.47±2.24	21.17±2.26	0.56 (NS)
5 min	20.53±2.0	21±4.7	0.19(NS)
10 min	20.00±2.03	20.40±3	0.49(NS)
15 min	19.97±1.81	20.90±2.44	0.17 (NS)
20 min	19.97±1.81	20.73±2.3	0.21(NS)
25 min	19.90±1.78	20.73±2.3	0.2(NS)
30 min	19.93±1.78	820.73±2.3	0.20 (NS)
45 min	19.40±1.07	220.07±1.9	0.23(NS)
60 min	19.60±1.22	919.87±2.03	0.89 (NS)

Table : VI INCIDENCE OF SIDE EFFECTS

	GROUP DK	GROUP K
Nausea	0(0.0%)	03(10%)
Vomitting	0(0.0%)	04(13.33%)
Emergence	0(0.0%)	02(6.66%)

Figure I – Age Distribution

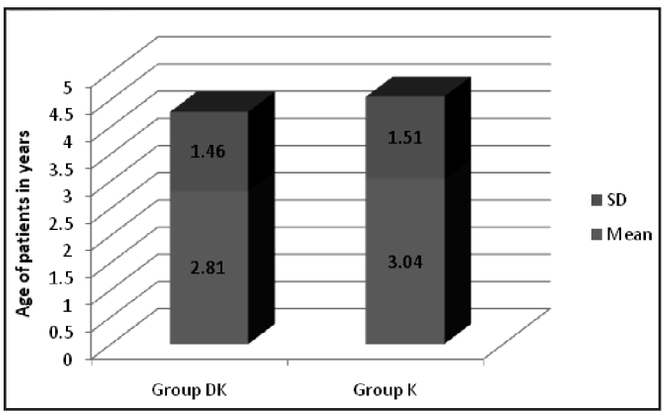


Figure II – Sex Distribution

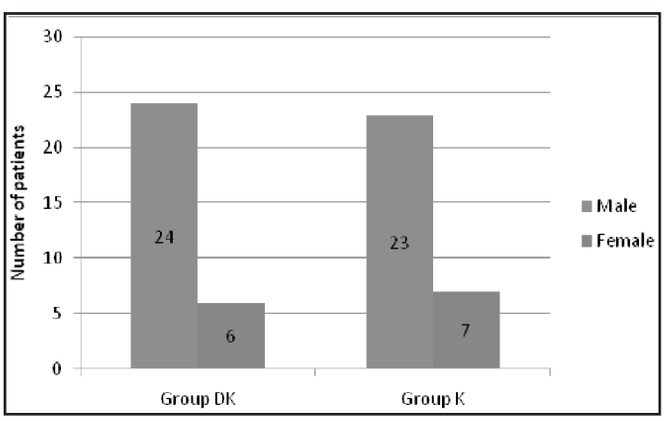


Figure III – Weight Distribution

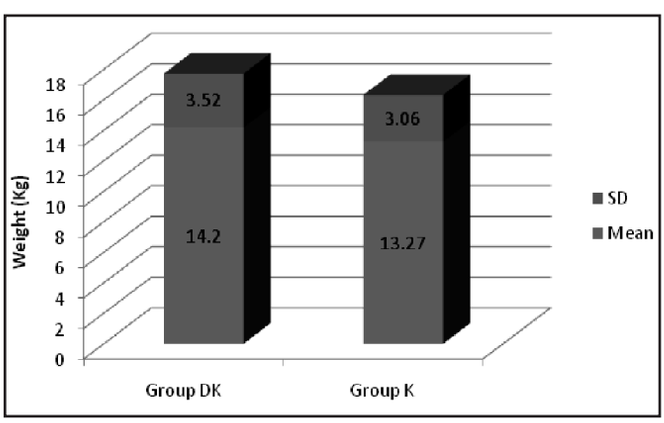


Figure IV – Clinical Outcome

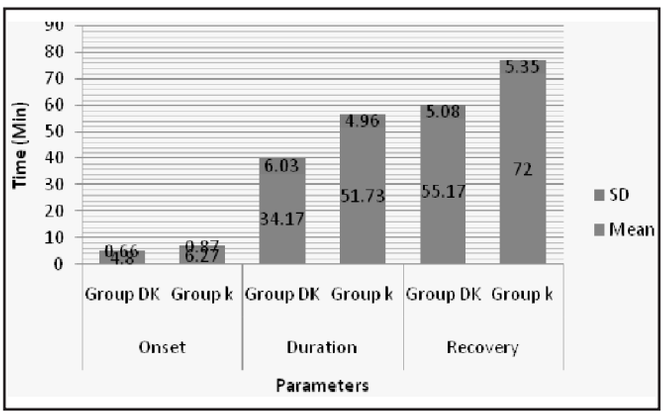


Figure V – Mean values of intra procedural monitoring of heart rate

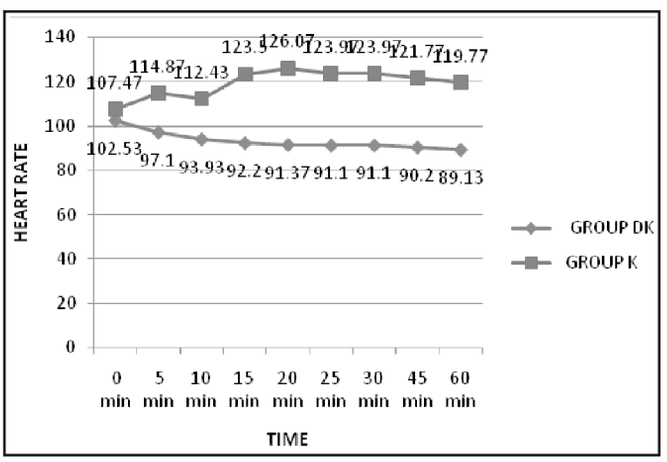
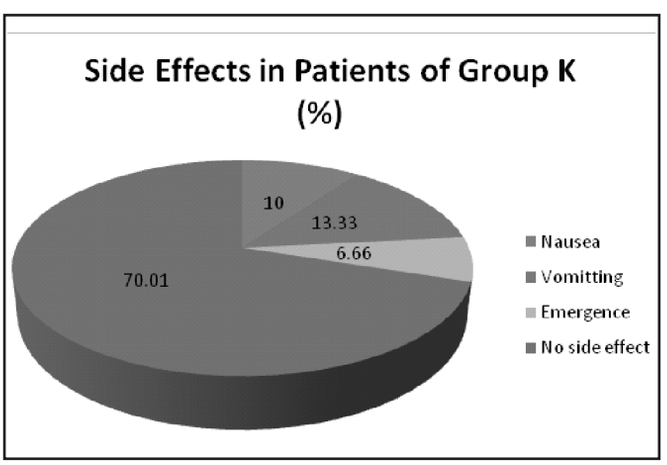


Figure V – Side Effects in Patients of Group K



CONCLUSION

Combination of intramuscular dexmedetomidine & ketamine was superior to ketamine alone in terms of onset of sedation, hemodynamic stability & less side effects.

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REVIEW ARTICLE

Ambulatory blood pressure measurement

Dr. Urman Dhruv*

"The measurement of blood pressure is likely the clinical procedure of greatest importance that is performed in the sloppiest manner"1

KEY WORDS : : ambulatory blood pressure monitoring, office blood pressure monitoring, masked hypertension, white coat hypertension

ABSTRACT

Hypertension remains the most common modifiable cardiovascular risk factor, yet hypertension control rates remain dismal. Office measurement of blood pressure has many limitations which led to development of newer modalities of measuring blood pressure. Ambulatory blood pressure measurement is widely accepted tool to diagnose hypertension and also to rule out white coat hypertension which is very prevalent cause of unnecessary initiation of treatment. Ambulatory blood pressure monitoring is also helpful in determining masked hypertension and nocturnal blood pressure records. Just as complications of diabetes are closely associated with glycated hemoglobin rather than fasting or post prandial blood sugar levels, cardiovascular outcome and target organ damage are more closely related to ambulatory blood pressure monitoring rather than office measurement of blood pressure.

INTRODUCTION

Blood pressure (BP) is an extremely labile hemodynamic parameter; it varies from heartbeat to heartbeat, from morning to evening, from winter to summer, from sleeping to awake, and from sitting to standing2. The last decade has seen emergence of two important techniques of blood pressure measurement outside the office of the doctor:Home Blood Pressure monitoring (HBPM) and Ambulatory Blood Pressure monitoring (ABPM). Hypertension guidelines propose home or ambulatory blood pressure monitoring as indispensable after office measurement. However, whether preference should be given to home or ambulatory monitoring remains undetermined. Of these two, ambulatory blood pressure monitoring (ABPM) is becoming widely accepted as a clinically useful tool not only for assessing cardiovascular risk in hypertensive patients but also to define presence of hypertension and to exclude white collar hypertension.The wide acceptance of this method can be attributed to improvements in technique, proper understanding of its data analysis and also due to recognition of unacceptably high limitations of office blood pressure measurements. Office BP is in fact characterized by a random error affecting casual BP readings and by a systematic error related to the patient's alerting reaction to the measurement procedure and setting, known as "white coat effect".

Advantages of ABPM

ABPM seeks readings in patients' natural environment leaving out the stress and alarm reactions encountered in office set up. As it is an automated, validated oscillometric device, it excludes operator mediated errors. Apart from obesity and arrhythmias especially atrial fibrillation, ABPM is of utmost importance and useful to all sets of patients and persons. Additionally the device can measure multiple readings at convenient time and does not require any skilled help once started. That makes it all the more reliable and efficacious to have more stable estimates of the prevailing BP in a given subject, reflecting the actual BP burden on cardiac and vascular targets more precisely than office readings. This is not only a methodological advantage but all major studies now suggest that the readings are more closely associated with and related to cardiovascular outcome and target organ damage as compared with office records. It has therefore arguably become the reference standard for the diagnosis of hypertension.

Thresholds for Hypertension Diagnosis Based on ABPM[7]

24-hour average	More than or equal to 130/80 mm of Hg
Awake (daytime) average	More than or equal to 135/85 mm og Hg
Asleep (night-time) average	More than or equal to 120/70 mm of Hg

Table 1 showing threshold of Hypertension based on ABPM as per The Working Group on Blood Pressure Monitoring of the European Society of Hypertension (ESH)

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Number of Measurements for a Satisfactory ABPM

The ABPM should be repeated if the following criteria are not met

- 24-h Recording with e"70% of expected measurements
- 20 Valid awake (0900–2100 h)
- 7 Valid asleep (0100–0600 h)
- Blood pressure measurements at 30 min intervals throughout 24 hours
- For research purposes e"2 valid daytime and 1 valid night-time measurement per h

Table 2 showing minimum necessary criteria to define a valid ABPM data[7]

INDICATIONS OF ABPM

Most of the guidelines have widened the horizons of indications of late. NICE guideline[8] published in 2011 states unequivocally that ABPM should be offered to anyone suspected of having hypertension by virtue of having had an elevated conventional BP measurement. The ESH 2013 guidelines[9] for hypertension took a more conservative approach by recommending that all subjects with grade I hypertension in the office at low or moderate total cardiovascular risk should be evaluated with out-of-office BP monitoring (ambulatory or home) to exclude white-coat hypertension, as well as all subjects with high-normal office BP or normal office BP with asymptomatic organ damage or at high total cardiovascular risk, to exclude masked hypertension.

METHOD

1. Performed on a working day
2. Cuff on non dominant arm
3. Frequency of measurement every15-30 minutes
4. Diary to be maintained for symptoms and events
5. Keep arm still while measurement

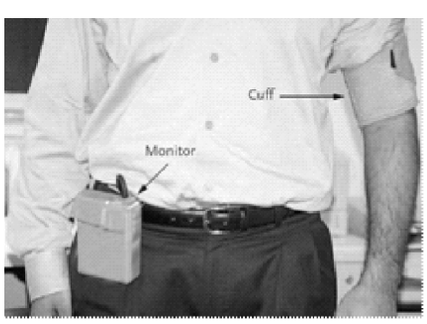

Figure 1: Method depicting ABPM measurement

CLASSIFICATION

Cross-classification of patients according to office and out-of the-office BP measurement delineates 4 groups as seen in figure 2.

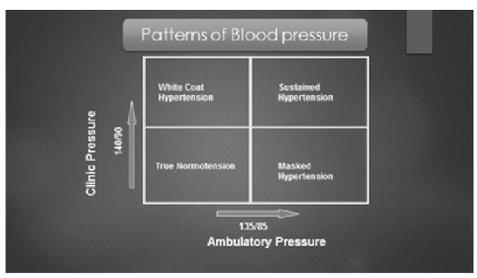

Figure 2: Classification based on ABPM15

White coat hypertension[16]

In clinical practice, the most well-established indication for performing ABPM, as recommended in all international guidelines, is to identify untreated patients who have high BP readings in the office but normal readings during usual daily activities outside of this setting, that is, white-coat hypertension, and to identify varying 24-hour BP profiles

White-coat hypertension

- Untreated subjects with elevated office blood pressure e"140/90 mm Hg† and
- 24-h ABPM <130/80 mm Hg and
- Awake ABPM <135/85 mm Hg and
- Sleep <120/70 mm Hg or
- Home blood pressure <135/85 mm Hg

Table 3: Diagnosis of white coat hypertension[7]

With the prevalence of white-coat hypertension in the community being as high as 20% to 25%, it is important to make an accurate diagnosis,10 which can best be achieved by performing 24-hour ABPM and home BP monitoring before prescribing antihypertensive drug therapy.

Masked hypertension [7,16]

As compared with white coat hypertension which is seen only in untreated patients, masked hypertension can be seen in treated and untreated patients where office records are acceptable but home BP records or ABPM records are high. However, as those patients who are on treatment already, cannot be classified as "masked Hypertension" and so are placed in the category of masked uncontrolled hypertension.

Masked hypertension : Untreated subjects with office blood pressure <140/90 mm□Hg and24-h ABPM ⩾ 130/80 mm□Hg andAwake ABPM ⩾ 135/85 mm□Hg and Sleep ⩾ 120/70 mm□Hg orHome blood pressure ⩾135/85 mm□Hg.

Masked uncontrolled hypertension : Treated subjects with office blood pressure <140/90 mm□Hg and24-h ABPM ⩾130/80 mm□Hg and/orAwake ABPM ⩾135/85 mm□Hg and/orSleep ⩾ 120/70 mm□Hg orHome blood pressure ⩾135/85 mm□Hg

Table 4: Diagnosis of masked hypertension and masked uncontrolled hypertension[10]

Diagnoses of masked hypertension require confirmation by repeating ABPM or Home BP monitoring within 3–6 months, depending on the individual's total cardiovascular risk. Recent trials indicate that masked uncontrolled hypertension is directly related to excessive cardiovascular risk and it is mainly attributed to isolated nocturnal hypertension making ABPM an important tool to assess high risk individuals[16.]

Nocturnal blood pressure measurement[7,16]

ABPM is best suited to measure blood pressure during sleep. Phenomenon of "dipping" is related to a physiological fall in blood pressure readings during sleep. The day time and night-time are decided by patient's diary or his traditional hours of sleep. There is compelling evidence that nocturnal BP is superior to daytime pressure in predicting outcome.[11,12] This has led investigators to suggest that the most important parameter for predicting outcome is the level of night-time BP, rather than any measure of day-night BP difference. Isolated nocturnal hypertension, which may be present in 7% of hypertensive subjects, can only be diagnosed with ABPM[13]. Nocturnal hypertension in patients participating in antihypertensive drug trials could have an important influence on the evaluation of the 24-hour efficacy of BP-lowering drugs. Although the degree of night-time dipping (defined as the difference between daytime and night-time BP) has a normal distribution in a population setting, it is generally agreed that a nocturnal BP fall >10% of daytime values, corresponding to a night/day BP ratio >0.9 serves as an arbitrary cutoff to define subjects as dippers. Recently, the American Diabetes Association recommended the administration of ⩾1 antihypertensive medications at bedtime[14]

Is ABPM cost effective[7]?

As the investigation easily identifies and defines those having white coat hypertension, it prevents unnecessary use of medicines in approximately 20% of patients. The investigation also provides compliance as the patient has more faith once he sees that so many readings are high despite taken by an automated machine. The patients stop associating symptoms with BP records and thereby do not take self- medication. It also identifies patients at high risk of cardiovascular disease. The technique can achieve potential savings of 3% to 14% for cost of care for hypertension and 10% to 23% reduction in treatment days when ABPM was incorporated into the diagnostic process at an annual cost that would be <10% of treatment costs.

Comparing 3 modalities of blood pressure measurement :

Home blood pressure record could provide an appropriate alternative to ambulatory monitoring in terms of diagnosis, particularly in primary care where it might not be immediately available or deemed too costly or when patients find it inconvenientor uncomfortable. Homemonitoringhasasmallerevidencebase than ambulatory monitoring but has gained acceptance over recent years as data accumulate and accurate equipment becomes more widely available. Both HBPM and ABPM are extremely useful in hypertension management, with a partial overlap of their clinical indications. Given that the clinical information they provide is not identical, both of these methods are likely to remain in use by physicians in daily practice.

Variable	ABPM	Office BP measurement	HBPM
True or mean BP	Yes	?	Yes
Diurnal BP rhythm	Yes	No	No
Dipping status	Yes	No	No
Morning surge	Yes	No	?
BP variability	Yes	No	?
Duration of drug effect	Yes	No	Yes

Table 5: Comparing features of 3 different modes of blood pressure measurement

Perhaps an approach using clinic (or home) measurements as a screening test followed by ambulatory blood pressure monitoring for blood pressures that are within10mmHg of threshold might be appropriate before definitive treatment but arguably a wider use of ambulatory monitoring would be needed to avoid overtreatment of white coat hypertension as well as detection of masked cases.

CONCLUSION

Ambulatory blood pressure monitoring estimates “true” mean blood pressure more accurately than clinic measurement. AMBP is more closely associated with cardiovascular outcome and target organ damage.Initially elevated BP measured by office-based methods is best confirmed by ABPM to avoid potential over diagnosis of isolated clinic hypertension and the potential harms of unnecessary treatment .Looking to the large number of patients having white coat hypertension, implications of AMBP will be widened to cover the definition of hypertension. Within near future, ABPM will become the reference standard to diagnose, treat and to prognosticate hypertension.

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REVIEW ARTICLE

Role of lifestyle modification in the management of pregnancy induced Hypertension.

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KEY WORDS : : hypertension, anti-hypertensive agents, preeclampsia, pregnancy, gestational hypertension, lifestyle modification

ABSTRACT

Hypertensive pregnancy disorders results in 6–8% of complicated pregnancies and cause significant maternal and fetal morbidity and mortality. The goal of lifestyle modification is to prevent significant cerebrovascular and cardiovascular events in the mother, without compromising fetal well-being. Current guidelines differentiate between the treatment of women with acute hypertensive syndromes of pregnancy and women with preexisting chronic hypertension in pregnancy. This review will address the management of hypertension in pregnancy through lifestyle modification, review the various pharmacologic therapies, and discuss the future directions in this field.

INTRODUCTION

Hypertensive pregnancy disorders cover a spectrum of conditions, including preeclampsia/eclampsia, gestational hypertension, chronic hypertension, and preeclampsia superimposed on chronic hypertension. According to the National High Blood Pressure Education Program (NHBPEP) Working Group Report on High Blood Pressure (BP) in Pregnancy, hypertension occurs in 6–8% of pregnancies in the United States.[1] Hypertensive pregnancy disorders represent the most significant complications of pregnancy and contribute significantly to maternal and perinatal morbidity and mortality. [2] Most of the current recommendations for the treatment of these disorders are based on expert opinion and observational studies, with a lack of evidence from randomized controlled trials. The overall strategy in the treatment of hypertension in pregnancy is to prevent maternal cerebrovascular and cardiac complications, while preserving the uteroplacental and fetal circulation and limiting medication toxicity to the fetus.

Hypertensive Pregnancy Disorder classification

Treatment strategies fall into two general categories – the management of acute hypertensive syndromes of pregnancy, such as preeclampsia/eclampsia, and the management of chronic hypertension. While the definitive treatment for acute hypertensive syndromes of pregnancy is delivery, expectant management with close observation may be appropriate in carefully selected patients, especially before 32 weeks gestation. Women with chronic hypertension should ideally be evaluated prior to pregnancy, with a focus on the presence of end-organ damage, evidence of secondary causes of hypertension (such as renal artery stenosis due to fibromuscular dysplasia, primary hyperaldosteronism and pheochromocytoma), medication adjustments, and counseling regarding the risks of preeclampsia and adverse fetal events.

Women with hypertensive pregnancy disorders should have a comprehensive plan of care, which includes prenatal counseling, frequent visits during pregnancy, timely delivery, appropriate intrapartum monitoring and care, and postpartum follow up. Care of these patients involves counseling at every step of the pregnancy to ensure that the woman is aware of the risks to her and her fetus such that she can make informed decisions.

Blood Pressure Management in Pregnancy

Hypertension in Preeclamptic Patients

The NHBPEP Working Group Report on High BP in Pregnancy and the American College of Obstetrics and Gynecology (ACOG) guidelines recommend treatment in preeclampsia when the diastolic BP (DBP) is persistently above 105–110 mm Hg, but there is no official recommendation regarding a systolic BP threshold for treatment. Most experts agree that pharmacologic therapy should be initiated when the BP approaches 150/100 mm Hg, [3] with the goal of preventing cerebral and cardiovascular events in the mother. If a woman has mild preeclampsia (DBP<100 mm Hg) with normal laboratory tests, other than low-level proteinuria, management as an outpatient can be appropriate,

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provided that there are frequent outpatient visits and that fetal nonstress testing (NST) is favorable. The frequency of formal ultrasound testing depends on the clinical condition and is at the discretion of an obstetrician. In the setting of severe preeclampsia that is being managed expectantly in the hospital, daily ultrasounds for fetal well-being may be indicated.

While treatment of hypertension may improve the risk profile of the mother, and therefore delay delivery, it does not cure preeclampsia, nor does it delay the progression to preeclampsia. [4] The diagnosis of severe preeclampsia includes greater than 1 of the following criteria – severe hypertension (defined as DBP>100 mm Hg), proteinuria > 5 g/24 hours or > 3+ on 2 random urine samples 4 hours apart, oliguria, cerebral or visual disturbances, pulmonary edema, epigastric or right upper quadrant pain, impaired liver function, thrombocytopenia or fetal growth restriction. The only definitive therapy for preeclampsia is delivery. When urgent control of BP is necessary, or when delivery is expected within the next 48 hours, intravenous agents, such as labetalol or hydralazine, are the drugs of choice. Oral agents may be considered if delivery is not imminent, and the choices of medications will be discussed further below.

Eclampsia may occur in the absence of gestational hypertension or preeclampsia in up to 20% of cases.[5] Magnesium sulfate has been shown to decrease the risk of eclampsia and maternal death without evidence of significant harm to the mother or baby.[6] Therefore, intravenous magnesium sulfate should be administered for seizure prophylaxis both during delivery and for 24 hours after delivery. The rate of continuous infusion, but not the loading dose, should be decreased for women with renal failure (as magnesium is renally excreted), and serum magnesium should be checked every 1–2 hours, as compared to women with normal renal function, for whom the level can be checked every 4–6 hours.

Timing of Delivery

The decision regarding the timing of delivery should be made after a careful assessment of the risks to the fetus and the mother. In appropriately selected patients, especially those before 32 weeks gestation, delivery can be postponed to allow for fetal maturation, particularly of the respiratory system. Women with mild preeclampsia, i. e., those with a diastolic BP < 100 mm Hg, without evidence of cerebral involvement, hemolysis with elevated liver enzymes and low platelets (HELLP) syndrome, or significant proteinuria (> 1 g/24 hours), may be candidates for this approach.

Treatment of hypertensive pregnancy disorders

Non-pharmacological therapeutic approach

Lifestyle interventions, such as weight loss and a reduction in salt intake, are of proven benefit in non-pregnant hypertensive patients. There is currently no evidence from prospective, randomized trials that instituting an exercise program during pregnancy is effective in preventing preeclampsia in at risk individuals,[7] , although some benefit has been seen in an animal model. Similarly, there is no current evidence that instituting a weight loss program in pregnancy can prevent preeclampsia,[8] although obesity is a risk factor for gestational hypertension and preeclampsia.[9] In 2009, the Institute of Medicine revised their guidelines for gestational weight gain and recommended that women who are overweight prior to pregnancy (body-mass index (BMI) 25–29.9) gain only 15–25 lbs during pregnancy, as opposed those with normal weight (BMI 18.5–24.9) prior to pregnancy, who should gain 25–35 lbs.[10] Obese women with a BMI > 30 should gain only 11–20 lbs, according to the new recommendations.

As volume contraction is common in preeclampsia, salt restriction is not routinely recommended. However, bed rest is frequently advised, and has been shown to lower blood pressure, promote diuresis, and reduce premature labor[11, 12.]

Some references regarding lifestyle modification in pregnancy induced hypertension are available in Ayurved literatures which can provide future direction for research for prevention of pregnancy induced hypertension, which are as below[13]

- Proper sleep at night, minimum 8-10 hours sleep at night, which calm down the mind and reduces stress and hypertension
- Early wake up before sunrise,
- Yogasana and Pranayam in early morning useful to reduce weight which provides relief in hypertension
- Light breakfast- ripen green gram or ½ cup of boiled cow milk
- Following of code and conducts of dietetics, like to take food whenever good appetite, proper chewing of food, not eat with full stomach etc.
- Avoidance of daytime sleep after taking food
- Meditation provides relief in hypertension by its tranquilizing effect
- Cheerfulness

- Patient should be advised to take ¼ reduced boiled water whenever thirst in little quantity
- Avoidance of late night awakening, anger, jealous, grid, worry etc.
- Cow milk and ghee should be taken daily
- Intake of green and leafy vegetables

CONCLUSION

Hypertension in pregnancy is a common complication of pregnancy and one associated with significant maternal and fetal morbidity and mortality. The central issue in the management of hypertension in pregnancy is achieving a balance between the maternal benefits derived from improved BP control, and the fetal risks resulting from intrauterine medication toxicity and possible uteroplacental hypoperfusion. Women with mild preeclampsia prior to 32 weeks gestation may be candidates for expectant management, but after 37 weeks, current evidence supports induction of labor to prevent adverse maternal and fetal outcomes. Unhealthy life style is of the most important cause for pregnancy induced hypertension. The lifestyle modification mentioned in Ayurved classics is useful to maintain the physiology of that specific system and thus provides strength to that system to fight against disease affecting that system. So healthy lifestyle mentioned in Ayurved can be useful to prevent from pregnancy induced hypertension and also felicitate the natural process of labor and may be beneficial to fetus too. Further systemic research on above mentioned lifestyle can bring a new preventive modality for the betterment of society, suffering from pregnancy induced hypertension.

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A-11/HFC/LM/2016-2017 Date: 9-12-2016

Dear Branch Secretary,

I hope that this circular finds you in the best of health and spirit. In continuation of my circular **A- 11/HFC/LM/2016-2017,** further tabulated information is given below for the revision of fees effective from immediate effect. Herewith I am sending the copy of I.M.A. H/Q fee schedule regarding revised fees

ORDINARY MEMBERSHIP FEES

CATEGORY	HFC	GMJ	GSB	ADM.FEE	TOTAL TO BE SENT TO GSB.IM
Annual Single :	**489-00**	**25-00**	**10-00**	**20-00**	**544-00**
Annual Couple :	**733-00**	**38-00**	**20-00**	**30-00**	**821-00**

LIFE MEMBERSHIP FEES

CATEGORY	TOTAL FEES	BR. SHARE	ADM. FEES INCLUDING GSB. IMA	TO BE SENT TO GSB. IMA
Single	**9565-00**	**760-00**	**{20-00 }**	**₹. 8805-00**
Couple	**14255-00**	**1200-00**	**{30.00 }**	**₹. 13055-00**

Local branch share to be collected extra as per individual branch decision/resolution Kindly note that fees at old Rates will be accepted up to **9-12-2016** only at State Office. Thereafter the new revised rates will be applicable.

Yours Sincerely

(Dr. Kamlesh B. Saini)
Hon. State Secretary

CASE REPORT

Lymphedema of lid –A rare entity

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KEY WORDS : Lymphedema , Blepharoplasty , Debulking

ABSTRACT

Here by we report two cases of lower lid lymphoedema. One was due to ulcer on the temporal side of the face and other due to radical neck surgery on the same side. Both were resistant to medical treatment. Surgical line of treatment in the form of blepharoplasty with debulking was done with excellent results.

INTRODUCTION

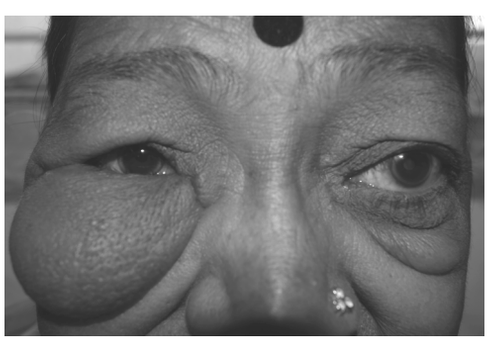
Lymphedema of lower eyelid is a rare condition. It is due to blockage in lymphatic drainage which in its recurrent or chronic forms leads to the development of the pitting type of swelling associated with epidermal thickening and rich subepidermal accumulation, particularly of lymphocytes and plasma cells. Eventually permanent hypertrophy of skin or elephantiasis may result. This sequence is due to stagnation of extracellular fluid rich in proteins which leads to the cellular infiltration and stimulation of fibroblasts. Lymphedema may be of primary or secondary type. Primary lymphedema may appear after birth or in later life due to defect in development of the lymphatic vessels in the embryonic life, also known as essential lymphedema.This was primary established by Noone and Milroy. Delayed type was established by Meige so it is also called Noone-Milroy-Meige disease(9).It is commonly transmitted as an autosomal dominant trait sometimes as a X chromosomal character. This condition usually involves lower lid and genitalia but may affect face and lids or the conjunctiva. Secondary lymphedema is more common and may be due to several factors like, malignant disease , irradiation and skin disease like psoriasis and acne rosecea . Aetiology of many cases are unknown. If lymphedema persists and if there is superadded bacterial infection then bacterial cellulitis may follow. Only few cases of lid lymphedema are identified .Here we report two cases of secondary type of lymphedema .

CASE REPORTS: Case 1

A 55 year old hindu female developed ulcer temporally on the left side of the face. It gradually increased in size. She took medical treatment in the form of antibiotic and anti-inflammatory for ulcer. After two months she developed swelling of the lower eyelid of the same side. Which was gradually increasing. Swelling was reddish brown in color, 35 * 25 mm in size , oval in shape ,solid, compressible, pitting and non tender in nature. There was a scar in the left pre-auricular region. Vision of the patient was 6/12. Other torch light examinations and slit lamp examinations were normal in left eye

CASE 2

A 68 year hindu female had developed squamous cell carcinoma of the neck 3 years back. She took radiotherapy and chemotherapy as a primary line of treatment. Then she was operated for right hemi mendibulectomy with radical neck dissection before two years. She has developed swelling over right side of lower lid since last 4 months. Swelling was 35 * 25 mm in size, reddish brown in color , oval in shape ,solid, compressible, pitting and non tender in nature. Patient's vision was 6/18 and lower lid cilia were turning inside. (fig 1)



In both the cases clinical diagnosis was lymphedema of lower lid. Blepharoplasty with mass debulking was performed with excellent results without any serious complications.

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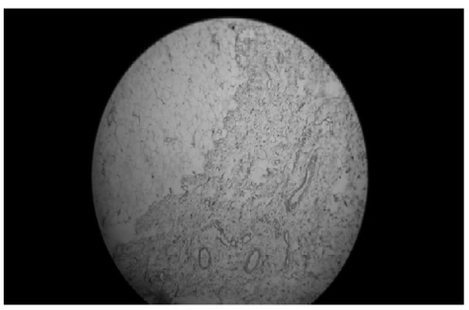
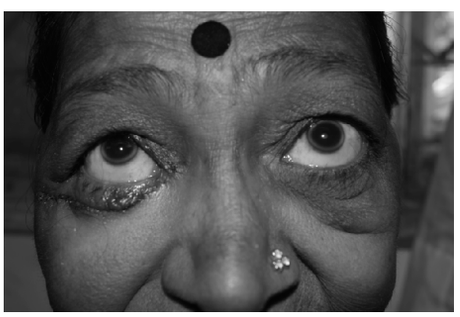
Histopathology :-

Excised tissues were sent for histopathological examination. Microscopic examination showed: significant capillary proliferation, dermal fibrosis, reactive fibroblasts, collagen breakdown, and multiple foci of chronic perivascular inflammation. These changes are consistent with chronic lymphedema.(fig 2)





RESULT

After doing blepharoplasty ,edema disappeared after 1st week and there was no scar after 1 month.We followed 1st patient for next 6 months and no recurrence was noted(fig 3). Second patient was followed for next 1 month with satisfactory result.

DISCUSSION

Lymphedema of the lids are uncommon. The possible etiologies are inflammatory (acne vulgaris, systemic lupus erythematosus, sarcoidosis, allergic dermatitis, angioedema, dermatomyositis), infectious (erysipelas, mononucleosis, herpes zoster), congenital (facial hemiatrophy, Apert's syndrome, McCuneAlbright syndrome), malignant (angiosarcoma, lymphoma, mycosis fungoides, lymphosarcoma, myeloma, Kaposi's sarcoma), trauma, trichinosis, hypothyroidism and nephrotic syndrome(8)(9). According to a study(1) ,the first order sentinel node for lymphatic drainage of the eyelids is pre-auricular lymph node.In our patient, the pre auricular site was affected by ulcer . In this situation lymphatic drainage was obstructed and no compensation was possible so patient developed lymphedema of the lower eyelid. In world literature(2) (3)it shows that radical neck dissection and irradiation are more common with associated complication of lymphedema of the face and lids. Venous disruption may also be a contributing factor to the lymphedema in the patient presented here, as internal jugular veins had been invaded with tumor.(4)

CONCLUSION

Here we report a rare cases of lower lid lymphedema which generally occur secondary to head and neck surgery (6)(7) . and blepharoplasty with debulking is treatment of choice. Patient were followed for few months with excellent result.

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CASE REPORT

Seronegative Celiac Disease : a case report

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KEY WORDS : Celiac disease, seronegative, intestinal biopsy.

ABSTRACT

In this article we report a rare case of 17 years old male with short stature, severe anemia and delayed puberty.After extensive workup andinspiteofbeing seronegative for celiac antibodies , he was diagnosed as an atypical case of celiac disease based on doudenal biopsy which showed Marsh grade 2 lesions. The patient is well on follow-up with gluten free diet.So, it can be concluded that negative serology should not rule out celiac disease, intestinal biopsy should be performed if there is strong clinical suspicion.The relevant literature has been reviewed and discussed in brief.

INTRODUCTION

Celiac disease(CD) is an immune mediated systemic disorder elicited by gluten and related prolamines in genetically susceptible individuals and is characterised by presence of variable combination of gluten dependent clinical manifestations, CD- specific antibodies, HLA-DQ2 or HLA-DQ8 haplotypes and enteropathy. It is one of the most common causes of chronic malabsorption[1]. The availability of screening test has resulted in picking up of many cases of CD.However many a times lack of gastrointestinal symptoms results in delayed diagnosis. Hereby we present a case of such delayed diagnosis.

CASE REPORT

A 17 years old male patient was admitted with complaintsofbilateralpedal edema and puffiness of eyelids for 15 days. On examination his weight was 18 kg(< 3rd centile) and height was 123 cm (< 3rd centile), BMI 11.89 kg/m2(severe undernutrition) , US/LS ratio 0.8. Height age was 8 years andbone age was7 years. Patient had severe pallor, bilateral pedal edemaupto knees with periorbital puffiness and mild ascites.His SMR staging was 1 (preadolescent), stretched penile length was 4 cm.Onasking leading questions patient gave history of poor appetite,abdominal fullness (bloating) and infrequent history of diarrhoea. Father was concerned for child having short stature and having no signs of puberty. Routine investigations showed Hb 5.5 gm% ,TLC 5600/ c mm, platelet 2.17 lacs/c mm, MCV 65fl, MCH 15pg,MCHC 24 g/dL, RDW 18%. Liver function tests showed normal liver enzymes,total serum proteins 4.2 g/dl ,serum albumin 1.8 g/dl ,serum globulin 2.4 g/dl. His lipid profile showed total lipid 243.91mg%,TG 52 mg%, HDL 16mg%, LDL 11mg%,cholesterol 37mg%. serum vitamin D3 <3ng/ml , serum vitamin B12 – 578pg/ml, serum T3- 1.12 ng/ml, T4- 9.54ng/ml, TSH 4.09microIU/ml. His USG abdomen showed fatty liver grade 1,USG scrotum showedtesticular volume of 0.5 ml,serum FSH 3.38mIU/ml (N 1.27-19.3mIU/ml), serum LH 0.456 mIU/ml (N 1.24-8.6mIU/ml) suggestive of hypogonadotropic hypogonadism. His HIV, HBsAg, HCV tests were negative, MT was negative. Serum amylase level was 49 U/L(N 10-90 U/L).Stool examination was negative for ova/cyst. In the view of severe malnutrition, short stature and delayed puberty, screening test for CD was done which showed serum tTGIgA 4.9IU/ml (N less than 20 IU/ml) and serum IgA 4.01g/L(N 0.61 - 3.48 g/l).Inspite of seronegativity, on the ground of strong clinical suspicion of CD, intestinal biopsy was done from duodenum which showed Marsh grade 2 changes. After biopsy report, patient was put on gluten free diet andnutritional supplements were started. During hospital stay his edemasubsided and now the patient is on regular follow-up for his nutritional status. Owing to seronegativity for CD, genetic testing for HLA DQ2 & DQ8 was planned, but due to the unavailability of this investigation at our centre it couldnot be done.

Our patient presented at 17 years age with severe malnutrition, short stature and delayed puberty. Lack of gastrointestinal symptoms could be the reason for not suspecting CD by treating physician earlier. Extensive diagnostic workup to rule out other causes and Marsh grade 2 lesions on intestinal biopsy helped to confirm the diagnosis in our case.

DISCUSSION

CD is T-cell mediated chronic inflammatory disorder with autoimmune component.[2] The current global prevalence is 0.5 to 1.26 % and Makharia et al. reported the prevalence of 1.04% in India.[3]

The classic CD presents with gastrointestinal symptoms.While the individuals with atypical CDcan also

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have gastrointestinal symptoms, approximately 70% of them are diagnosed based on extraintestinal manifestations like iron-deficiency anemia, dermatitis herpetiformis, unexplained short stature , neurologic symptoms and delayed puberty [Telega et al 2008][4].

The age of diagnosis is extremely variable. In India 80% of children with CD have classical presentation.Nonclassic CD usually presents in later childhood or adulthood.[1]

A diagnostic test for CD specific antibody detection is the first tool used to identify patient with signs and symptoms suggestive of CD for further workup. Initial testing is IgA class anti-tTG or EMA antibody for IgA competent subjects[5].

In a seronegative patient with CD, other tests which help to confirm diagnosis are

I. measurement of anti-DGP titres in patient <2 years age OR

ii. measurement of IgG class antibodies in IgA deficient subjects OR

iii. duodenal biopsy OR

iv. genetic testing for HLA DQ2 and HLA DQ8 typing by DNA test, which offers higher sensitivity (91% to 96%). The main role of HLA DQ typing is to exclude the diagnosis of CD [6]

According to ESPGHAN criteria, the 2 requirements mandatory for the diagnosis of CD are the finding of villous atrophy with hyperplasia of the crypts and abnormal surface epithelium, while the patient is eating adequate amount of gluten and a full remission after withdrawal of gluten from the diet[2].

As per study by Salmi et al. [7] out of 177, 22 were found to be seronegative. The seronegative group differed significantly from the seropositive group by being significantly older ,more likely to be male and having significantly more abdominal symptoms.The authors suggest that seronegativity may result in cases in which a prolonged, severe immunological reaction at the intestinal mucosal surface increases antibody avidity reducing blood stream overspill from intestine.

Carroccio et al. [8] analysed 191 patients with biopsy proven celiac disease with 15% being seronegative for either anti EMA or anti tTG.Carroccio et al. demonstrated that the culture medium of intestinal biopsy provides a higher sensitivity and diagnostic accuracy than serum assay for anti EMA [10]. This may be useful diagnostic tool for additional confirmation for CD,particularly for the patients in whom the serology is negative.

All patients with confirmed diagnosis of CD should be put on gluten free diet i.e.,foods that contain ≤ 20 ppm of gluten. Oats are considered safe, however 5% of patient may be sensitive to oats[5]. Celiac U.K. guidelines have also recommended pneumococcal vaccine for patients with CD.

CONCLUSION

Atypical CD with negative serology is a diagnostic dilemma. Intestinalbiopsy still should be carried out with strong clinical suspicion.Genetic testing if available offers additional support to the diagnosis.

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ABBREVIATION NOTES

CD=celiac disease, anti-tTg= anti-tissue Trans glutaminase antibody, EMA= endomysial antibody, HLA=human leukocyte antigen, DGP= deamidated forms of gliadin peptides, N= normal range.

CASE REPORT

Rasmussen's Encephalitis in a 15 month old female child: an unusually early presentation

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KEY WORDS : Focal seizures, hemiplegia, hemiatrophy.

ABSTRACT

Rasmussen encephalitis is a chronic, progressive inflammation of the brain of unknown origin. Early diagnosis and treatment with immunoactive agents and/or hemispherectomy are sought to prevent the progressive cognitive decline that accompanies this disease. Clinical features and neuroimaging help in early diagnosis. We report an usually early presentation (probably the earliest ever reported!) with the first partial seizure occurring at seven months age and diagnosis clinched at fifteen months age.

INTRODUCTION

Rasmussen's encephalitis (RE) is an inflammatory disease of the brain of unknown origin, usually unilateral. It clinically presents with intractable focal seizures, epilepsia partialis continua, and progressive deterioration of neurological function of the affected hemisphere leading to hemiplegia, hemianopia and aphasia (dominant hemisphere) 1.

CASE REPORT

We present a case of a fifteen month old female child who presented with a left sided focal seizure(fourth episode) which occurred while awake and afebrile , lasted for nearly forty five minutes, relieved by injection midazolam followed by referral to our tertiary care hospital. The first attack had occurred at seven months age, CT scan and CSF studies were normal. EEG was advised but the patient did not turn up at the given date. MRI was advised but due to economical reason, could not be done. The patient had an uneventful birth history with no family history of epilepsy. She was developmentally normal previously, with a regression of milestones atb present. In the current admission, the patient presented as conscious, vitally stable, with pupils reacting to light, left sided hemiparesis and hypotonia, with preferential use of right upper limb to grasp objects. Routine investigations and metabolic workup were normal. On visual examination, left hemianopia was detected. MRI brain showed abnormal hyperintensity in cerebral white matter; no cerebral edema. T2/FLAIR images were suggestive of prominent subarachnoid space with hemiatrophy involving right parieto-occipito-temporal region.(Figure) These findings clinched the diagnosis of Rasmussen's Encephalitis. EEG was suggestive of epileptiform discharge. Patient was started on syrup carbamazepine, sodium valproate and tablet clobazam alongwith physiotherapy. Plan was to start steroids on follow up if seizures were not controlled. Prognosis was explained to the relative.

DISCUSSION

Rasmussen's Encephalitis is a sporadic chronic inflammatory disease of the central nervous system, first reported by Theodore Rasmussen in 19581. Investigators in a German study[2] estimated the countrywide incidence at [2-4] cases per 10 million people aged 18 years and younger per year. The mean age of presentation is between 6 to 8 years, rarely at adolescent and adult age(roughly 10%)[4,5,6.] The existence of bilateral disease is very rare. Only two out of the roughly 200–300 published cases of Rasmussen's encephalitis had evidence of bilateral disease on histopathology.[8,16]

The etiology of Rasmussen's Encephalitis is unknown, theories include role of viral infections, autoimmune phenomenon against a protein of glutamate receptor.[9,10,] immunopathological mechanisms responsible for CNS degeneration include antibody-mediated, T-cell cytotoxicity and microglia-induced degeneration.[11]

Diagnosis of Rasmussen's Encephalitis is based on characteristic clinical, radiological and pathological features with more emphasis on clinico-radiological features, as brain biopsy, due to its invasive nature, is not done in all the cases. Bien et al. proposed a three stage natural history of Rasmussen's Encephalitis based on long term observation of 13 patients.[12,13] The first prodromal stage, manifests with a relatively low seizure frequency and, rarely, mild hemiparesis. The second

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acute stage is characterized by an augmentation in the seizure frequency, often as EPC, and an increase in the degree of hemiparesis. The final residual stage presents with permanent and stable neurological deficits, mostly severe hemiparesis, and a decreased frequency of seizures. Our patient presented in acute stage. Bien et al. also proposed a five stage MRI model of RE based on a retrospective study of 39 MRI scans of 10 patients (Table).Functional studies using 18F-FDG PET show diffuse unilateral cerebral hypometabolism that might manifest when MRI atrophy is still at a minimum. [3]

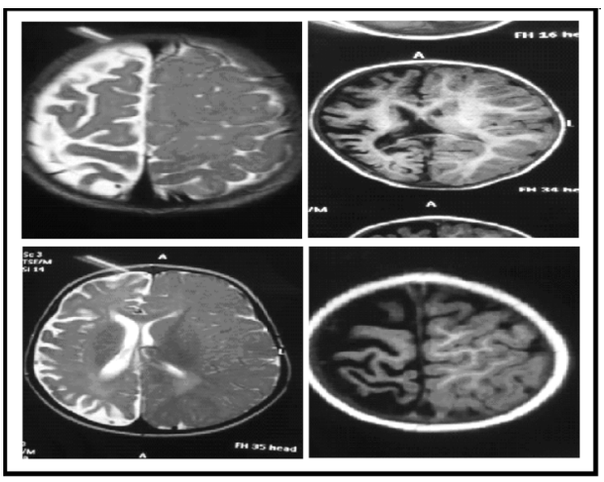

Figure: the MRI findings of our patient

MRI STAGES:

Stage	Volume	T2/FLAIR signal
0	Normal	Normal
1	Swelling	Increased
2	Normal	Increased
3	Atrophy	Increased
4	Atrophy	Normal

CSF examination in most cases is normal. Histopathological examination of biopsy material and resected specimens reveal a characteristic triad of findings: perivascular lymphocytic cuffing of round cells, gliosis and microglial nodules in the cortical layers of the brain and white matter. Resected specimens in the more advanced clinical stages have demonstrated diffuse cortical atrophy with neuronal loss and a lack of inflammatory cells. [15]

Radiologically, Rasmussen's Encephalitis should be differentiated from other causes of cerebral hemiatrophy like Sturge-Weber syndrome, hemispheric infarction (Dyke-Davidoff-Masson), and MELAS (mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes).[21]

Immunomodulatory treatment with either high-dose steroids or intravenous immunoglobulin is a treatment option for patients with Rasmussen's Encephalitis, and this can achieve more than 50% reduction in the seizure frequency [18]. Strategies have also been proposed that start with steroid pulse treatment and change to tacrolimus.[3] Despite the controversy surrounding the introduction of functional hemispherectomy, it is still the only curative treatment for Rasmussen's Encephalitis [19,18,20]. Homonymous hemianopia and hemiplegia are inevitable, although both might be present before surgery. Hemispherectomy offers one of the best chances of making patients with Rasmussen's encephalitis seizure free (>70–80% long-term seizure-free outcome). Most results show cognitive stabilization after hemispherectomy,[17,14] with better cognitive outcome in non-dominant-hemisphere surgery and poorer outcome in dominant-hemisphere surgery and in individuals with refractory seizures after surgery.[7] Early diagnosis of Rasmussen's Encephalitis is crucial for selecting patients for aggressive medical therapy or major surgical interventions .

CONCLUSION

As diagnosis was delayed due to economical reasons and poor follow-up, Rasmussen's encephalitis could be detected in this case at a later stage when atrophy had already occurred. Rasmussen's Encephalitis can present as early as at seven months age. Thorough investigation must be carried out in every case to allow early diagnosis and timely intervention with immunomodulatory treatment before cerebral atrophy, cognitive decline and developmental regression sets in.

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CASE REPORT

Ruptured Spinal Dermoid with fat dissemination in intracranial subarachnoid space

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KEY WORDS : Intraspinaldermoid, disseminated fat, subarachnoid space

ABSTRACT

Intraspinaldermoid tumours are rare benign, slow growing tumours and tend to extend to the subarachnoid space. Several causes including spontaneous, iatrogenic or traumatic rupture have been reported to result in dissemination of lipid material from the dermoid tumours into the subarachnoid space or ventricles. We report such case of a 26 years old male patient with spontaneous rupture of spinal dermoid causing dissemination of fat droplets in intracranial subarachnoid space.

INTRODUCTION

TIntraspinal dermoid tumours are rare, benign, slowgrowing tumours and tend to extend to the subarachnoid space [1].Dermoid cysts arise from the inclusion of embryonic ectoderm into the neural tube during the 5th to 6th weeks of fetal life and typically occur in the midline [3]. We report a case of a 26 years old male patient with spontaneously ruptured spinal dermoid with disseminated intracranial fat droplets.

CASE REPORT

A 26 years old male patient presented with headache and vomiting since 2 weeks. Laboratory findings were normal. There was no history of lumbar puncture or major operative procedure.

NCCT Brain shows well defined hypodense lesions (HU - 57) involving the subarachnoid spaces.

MRI Brain T1W images show multiple tiny hyperintense foci involving bilateral fronto-temporal lobe sulci, intraventricular regions and basal cistern. The lesions appear hyperintense on T2W images. On DWI images, the lesions do not show diffusion restriction. On GRE, the lesions show blooming.

MRI Spine T1W, T2W and Fat-suppressedT1W sagittal images shows an altered signal intensity intramedullary lesion (shown by arrow) at the level of T11-T12 vertebral bodies which appears hyperintense on T1W and T2W images and shows suppression on Fat-saturated T1W images. Another large lobulated heterogeneous altered signal intensity intramedullary lesion (shown by dotted arrow)involving the spinal cord at L1-L2 vertebral body level. It appears heterogeneously hyperintense on T1W images and hypointense on T2W images. An epidural collection is seen anterior to the lesion at the same level showing hyperintense signal on T2W and hypointense signal on T1W images. The lesion also causes posterior scalloping of the vertebral bodies. These imaging findings suggest an intramedullarydermoid with rupture causing hematoma and disseminated intracranial fat droplets.

DISCUSSION

Spinal dermoids are dysontogenetic tumours arisingfrom inclusion of ectopic embryonic rests of theectoderm within the spinal canal at the time of neuraltube closure between the third and the fifth week ofembryonic development. They have a thick wallcovered with stratified squamous epithelium containingdermal appendages such as hair, sebaceous glands,sweat glands and hair follicle and less commonly, teethand nails [1].

Dermoid tumours comprise 1.1% of intraspinal tumours. There is no communication between the cyst and the subarachnoid space. Several causes including spontaneous, iatrogenic or traumatic rupture have been reported to result in dissemination of lipid material from the dermoid tumours into the subarachnoid space or ventricles.Dermoid tumours show aslight male predominance, and most dermoid tumoursare revealed during the second and third decades.The lumbosacral region is mostcommon site (60%) involving the caudaequina and thecornusmedullaris followed by the upper thoracic (10%)and cervical (5%) regions [1].

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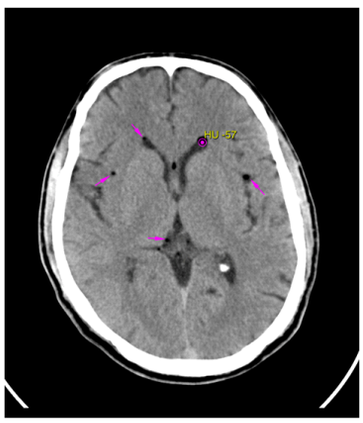
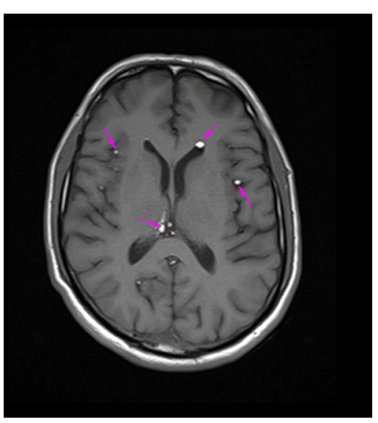
NCCT BRAIN AXIAL



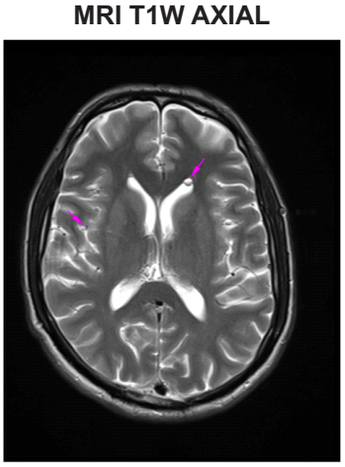
NCCT Brain shows well defined multiple hypodense lesions (HU -57) involving the subarachnoid spaces.

MRI T1W AXIAL



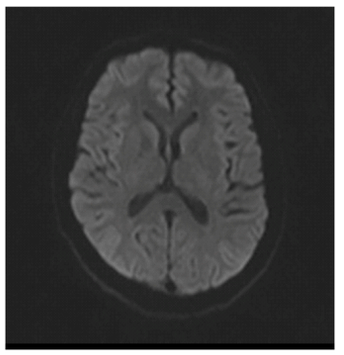
MRI Brain T1W axial image shows multiple tiny hyperintense foci involving bilateral fronto-temporal lobe sulci, intraventricular regions and basal cistern.

MRI T1W AXIAL



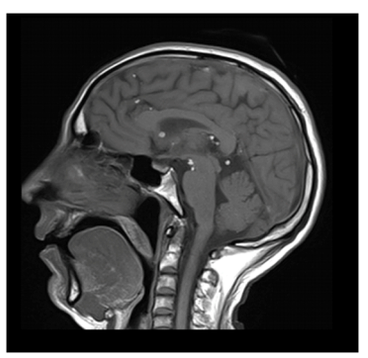
MRI T2W axial image shows multiple hyperintense lesions in subarachnoid space.

MRI DWI



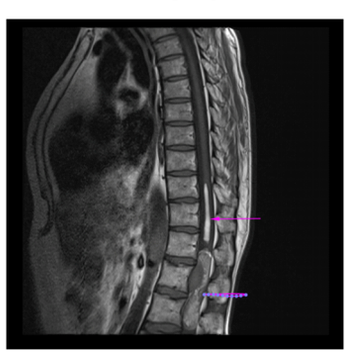
MRI T2W axial image shows multiple hyperintense lesions in subarachnoid space.

MRI T1W SAGITTAL



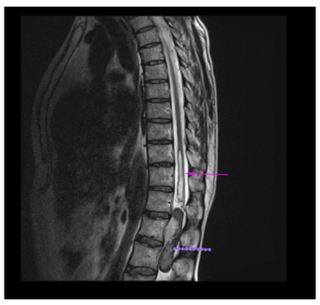
MRI Brain T1W Sagittal image showsmultiple tiny hyperintense foci involving frontal and temporal lobe sulci, intraventricular regions and subarachnoid spaces. The above imaging findings suggest fat droplets in the subarachnoid space.

MRI T1W SAGITTAL



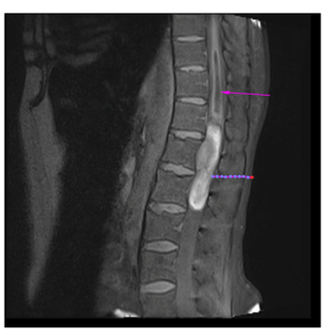
MRI Spine T1W sagittal image shows an altered signal intensity intramedullary lesion (shown by arrow) at the level of T11-T12 vertebral bodies which appears hyperintense on T1W. Another large lobulated heterogeneous altered signal intensity intramedullary lesion (shown by dotted arrow) involving the spinal cord at L1-L2 vertebral body level. It appears heterogeneously hyperintense on T1W. An epidural collection is seen anterior to the lesion at the same level showing hypointense signal on T1W images. The lesion also causes posterior scalloping of the vertebral bodies.

MRI SPINE T2W SAGITTAL



MRI Spine T2W sagittal image shows an altered signal intensity intramedullary lesion (shown by arrow) at the level of T11-T12 vertebral bodies which appears hyperintense on T2W images. Another large lobulated heterogeneous altered signal intensity intramedullary lesion (shown by dotted arrow) involving the spinal cord at L1-L2 vertebral body level. It appears heterogeneously hypointense on T2W images. An epidural collection is seen anterior to the lesion at the same level showing hyperintense signal on T2W images.

MRI SPINE T1W FAT SUPPRESSED IMAGE SAGITTAL



MRI Spine Fat-suppressed T1W sagittal images shows an altered signal intensity intramedullary lesion (shown by arrow) at the level of T11-T12 vertebral bodies which shows suppression on Fat-saturated T1W images. Another large lobulated heterogeneous altered signal intensity intramedullary lesion (shown by dotted arrow)involving the spinal cord at L1-L2 vertebral body level. It appears heterogeneously hyperintense. An epidural collection is seen anterior to the lesion at the same level showing hypointense signal. The above imaging findings suggest intramedullary spinal dermoid with rupture causing epidural hematoma.

Although dermoid tumours develop from the embryonic period, symptoms may not occur until adulthood due to their slow growth, symptoms and signs secondary to the space-occupying lesion are location- dependent and are due to the irritative effect on and/or compression of the adjacent structures [1] .Once rupture of the cyst occurs acute symptoms relateto chemical or aseptic meningitis, headache orseizures may be developed due to dissemination of lipiddroplets in the cerebrospinal fluid (CSF) pathways.Lumbar arachnoiditis may be developed as a result ofleakage of fat and proteinaceous material into thesubarachnoid space. The highly irritativelipid contentof dermoid tumours may cause severe inflammatory response, though spread of fat into the CSF may also beclinically silent. After rupture of dermoid tumour occurs, lipiddroplets float in the CSF and are passively conveyedby CSF movement. It can therefore spread throughoutthe subarachnoid space and ventricular system [1].

These lesions will have internal density characteristics consistent with fat (negative Hounsfield units), although density values greater than fat may be encountered depending on the nature of an individual tumor's contents. The dermoid wall is typically seen and can calcify. Occasionally the wall will at least partially enhance following the administration of CT-iodinated contrast material. On MRI scans, dermoids will be hyperintense (bright) on T1-weighted imaging and heterogenous on T2-weighted imaging. If the internal fat content is relatively low, the lesion will reveal cerebrospinal fluid–like signal intensity. In such cases, fluid attenuation inversion recovery (FLAIR) is useful, in that the fat will appear hyperintense (bright) on a background of suppressed fluid signal (dark). On MRI, fat constituents create a so-called “chemical shift” artifact due to misregistration of the signal in the frequency-encoded direction. This can be particularly useful in diagnosing these lesions preoperatively. When a dermoidtumor ruptures, fat droplets—appearing hypodense on CT or T1 hyperintense on MRI—may be seen scattered and floating within the nondependent portions of the ventricular system and/or subarachnoid space. This is considered a classic imaging feature of these lesions. In the setting of complicating chemical meningitis, intense pial and ventricular ependymal enhancement may be detected after the administration of MRI gadolinium contrast [2].

Although the imaging appearance of dermoidtumors is characteristic, several other intracranial lesions must be considered in the differential diagnosis, such as epidermoids, teratomas, lipomas, craniopharyngiomas, and occasionally arachnoid cysts[2].

Surgical care focuses on complete microsurgical resection of the mass and wall. If the tumor has not ruptured preoperatively, great care is made to avoid spilling the contents in the surgical bed. Patients typically do well after operative intervention. Recurrence is rare but is more common if there are retained portions of the tumor wall. Rare reports describe the development of squamous cell carcinoma in retained remnants of a dermoid cystic tumor wall [2].

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CASE REPORT

Unusual cause of Colonic Obstruction in Adult - Colonic Intussuception due to Giant Pedunculated Lipoma: A case report and brief review of Literature

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KEY WORDS : Colonic Lipoma; Intussusception; Adult Bowel obstruction

ABSTRACT

Intussusception is less common in adults, and if present, is usually associated with an identifiable etiology in 90% of cases. Colonic lipomas are uncommon nonepithelial neoplasms that are typically sessile, asymptomatic and incidentally found during endoscopy, surgery, or autopsy. Small Lipomas are usually asymptomatic, but Lipomas of size more than 5 cm often present as abdominal pain, vomiting, diarrhea, bleeding and Intussusception. We present a case of 45-year-old female admitted in surgical ward with intermittent abdominal cramps for eight days and signs of sub acute intestinal obstruction. Physical examination showed tenderness in the left lower abdominal quadrant. A CT scan showed a coil spring appearance highly suspicious for Intussusception of the descending-colon splenic flexure and subsequently a laparotomy was performed. The Intussusception was found in the splenic flexure of colon and limited colonic resection was performed with end-to-end anastomosis. After surgical resection, the histopathologic examination of the specimen showed the configuration of pedunculated lipoma with tip ulceration, measuring 8x5x 6 cm in diameter. Patient was discharged on day five post-operative with good condition. The present case highlights the possibility of a benign cause of large bowel obstruction in Adults and its subsequent management.

INTRODUCTION

Intussusception, the telescoping of proximal segment of gut into the distal segment, is a rare cause of bowel obstruction in adults, accounting only 1 % of the confirmed cases and the most common site of bowel involved is the ileocolic valve. Colo colonic intussusceptions are even less common (1, 2). The most common cause for intussuception leading point are Lymphoma and adenocarcinoma (63%) (3) and intussusception due to a gastrointestinal lipoma constitutes an infrequent clinical entity (4). Colonic lipoma typically presents as a sessile polypoid mass. If complications of colonic lipoma such as obstruction, intussusceptions, perforation or massive hemorrhage present in patients, surgical removal of tumor is indicated. Computerised tomography (CT) for abdominal imaging has led to increased detection of transient intussusceptions with or without any underlying disease. Here we present a 45 years old female who presented with bowel obstruction secondary to colo-colonic Intussusception due to a colonic lipoma at the splenic flexure of colon.

CASE REPORT

A 45 year old female patient presented with complaint of intermittent left lower quadrant abdominal pain accompanied with nausea and vomiting. She had no history of medical or surgical problem. Computed tomography (CT) of the abdomen showed a coil spring appearance mass about 8cm in diameter in the left side of abdomen region (Figure 1). Colo-colic Intussusception was highly suspected. Midline laparatomy was performed and an Intussusception mass from splenic flexures to sigmoid colon was found (Figure 2). The mass was firm and had a smooth surface. A segmental colonic resection with 5 cm margins on either side was done. No regional lymph node enlargement or invasion of surrounding structure was observed. The specimen was cut open and revealed a lipoma (Figure 3). The histopathologic examination confirmed a pedunculated submucosal Lipoma measuring 8x5x 6 cm in diameter with ischemic ulceration of the overlying mucosa (Figure 4). After operation, patient was discharged on fifth postoperative day with no complications.

DISCUSSION

Intussusception is a common condition in children but in adults, Intussusception accounts for only 5 % of all cases and 1 % of all bowel obstructions (1). The most common sites of intussusceptions are entero-enteric and ileocolic

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Fig. 1 : Computerized Tomography image showing coil spring appearance in the descending colon

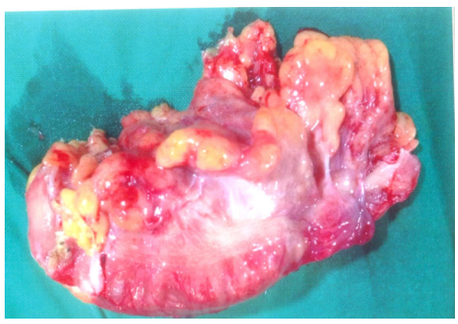


Fig. 2 : Resected Specimen

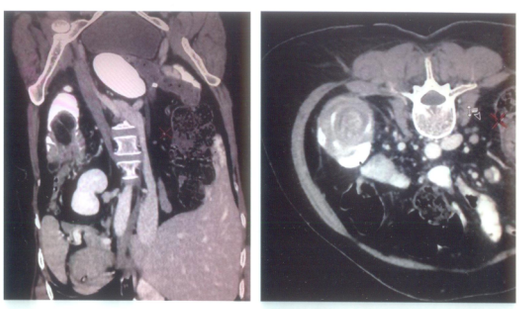


Fig. 3 : Cut section of the colonic Lipoma

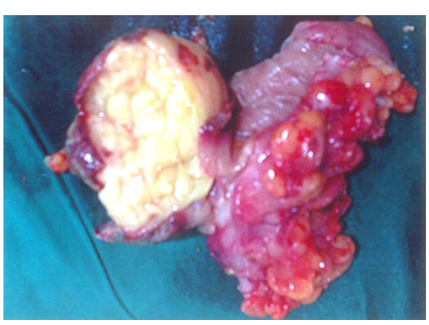
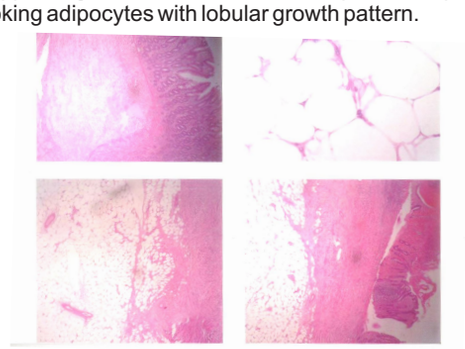


Fig. 4: Pathology: Sections show colonic wall tissue reveals a benign appearing neo formed lesion composed of proliferated bland-looking adipocytes with lobular growth pattern.



(2). Intraluminal polypoid lesions have a greater tendency to cause invagination of the bowel as peristalsis drags the lesion forward (5). The exact mechanism precipitating intussusception, especially intussusception without a lead point, is not well under-stood (6,7).In adult, Intussusception is usually due to pathological intra luminal lesions and in two thirds of adult colo-colonic Intussusception, the eitiology is colonic malignancy.

Colonic lipoma as leading cause is uncommon (8) and if found they are located in the cecum, submucosally. Colonic Lipomas are seen more commonly in women between 50 and 60 years old (9). Most typically, they are asymptomatic and incidentally found during endoscopy, surgery, or autopsy. Only 25% of them develop symptoms, especially when their diameter exceeds 2 cm. Colonic lipoma typically presents as a sessile polypoid mass, arising from the submucosa with an intact mucosa. Infrequently, lipomas of the colon are pedunculated, with ulcerated or necrotic overlying mucosa. Colicky abdominal pain, rectal bleeding and bowel obstructions are the most common symptoms and palpable mass can found in 24-42% of patients.

Recent reports in the literature have suggested that abdominal CT scanning is the preferred radiologic modality for diagnosing intussusception from colonic lipomas (10). The sensitivity of CT scan to correctly diagnose intussusceptions has been reported from 71.4%- 87.5% while its specificity in adults has been reported to be 100% as verified by the subsequent surgery. For patients with features typical of colonic lipoma, CT reliably confirms the diagnosis. In colonoscopy visualization, characteristic mark of Lipoma is elevated normal mucosa over the tumor and fat extrusion occurs after taking a biopsy. Colonic Lipoma can be treated with different options; endoscopically, surgically or with minimally invasive laparoscopically. Endoscopic resection of Lipoma concerned for tumors less than 2 cm, however perforation and hemorrhage can occurred during this procedure. Surgery has been recommended by many of the authors as the standard method of treatment for Lipoma greater than 2 cm in size. Surgical treatment includes resection, colostomy with local excision, limited colon resection, segmental resection, hemicolectomy, or subtotal colectomy; depends on the tumor size, location and presence of definitive diagnosis before operation. The choice procedure of colonic Intussusception is resection without reduction due to the presence of underlying malignancy. During last few years, a few selected cases of successful laparoscopic resection under colonoscopic guidance of symptomatic colonic lipomas have been reported (11).

In our patient, there was an intraluminal pedunculated lipoma as a leading point for colocolic intussusceptions which was diagnosed by CT abdomen and operated electively. Postoperative event was uneventful. The patient is following up since six months without any bowel symptoms.

CONCLUSION

Lipomas of the gastrointestinal tract are rare conditions and are usually localized at submucous level. CT is the examination of choice. Surgical approach in form of segmental resection remains the treatment of choice for

large colonic lipoma. The present case highlights the possibility of intussusception with an unusually benign cause, such as lipoma, when adult patients present with abdominal symptoms and obstruction. In conclusion; high index of suspicion is required to diagnose a case of colocolic intussusception in adults.

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CASE REPORT

Sino-nasal Hemangiopericytoma - a rare case report with review of literature.

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KEY WORDS : Hemangiopericytoma, Hemangiopericytoma of nose and paranasal sinuses, Vascular tumours of nose and paranasal sinuses.

INTRODUCTION

Hemangiopericytoma is a very rare angiogenic tumour first described by Stout and Murray in 1942 (1). It may occur in different parts of the body but occurrence in nose and paranasal sinus is very rare. Approximately 250 cases have been reported worldwide involving the nose and paranasal sinuses suggesting its rarity (2, 3). Due to its rarity a proper line of management has not been established to tackle this tumour. We are describing case of Left Sino-nasal Hemangiopericytoma with Clinical, Histo-pathological and Radiological features.

CASE REPORT

A female patient, 22 years age was admitted in the department of E. N. T. in emergency with epistaxis for 2 days with anterior nasal packing done outside. She has history of recurrent episodes of Left Nasal bleeding for 3-4 months for which she has taken some medications from general practitioner. She also has associated history of progressive increase in nasal obstruction. Haematological investigations including Bleeding profile was normal except anaemia with Hb 8.1 gm%. Patient was taken for pack removal and nasal endoscopy. Pack removed; there was smooth whitish mass present in the left side middle meatus extending into nasal cavity. Patient was subjected to CT scan with contrast examination that revealed 35*13*20 mm3 enhancing soft tissue mass lesion in Left middle meatus with extension to Posterior ethmoid and Nasopharynx with sinusitis of Left maxillary, ethmoid and sphenoid sinuses.

Patient was planned for surgical excision endoscopically. There was smooth surface whitish polypoidal mass arising from lateral surface of the middle turbinate extending into middle meatus, going posteriorly into naso-pharynx. The mass was dissected from the lateral surface, removed in Toto and subjected to histo-pathological examination. Drainage of all sinuses on Left side achieved. On H & E staining there was presence of ramifying vascular proliferation with compression and obscuration from surrounding cellular proliferation of round to ovoid cells, giving “Stag horn” or “Antler like” configuration with hyalinization suggestive of Hemangiopericytoma. Post op period was unremarkable. After 24 months follow up the cavity is well healed.

DICUSSION

Hemangiopericytoma is a very rare angiogenic tumour first described by Stout and Murray in 1942 (1). It may occur in different parts of the body but occurrence in nose and paranasal sinus is very rare. Approximately 250 cases have been reported worldwide involving the nose and paranasal sinuses suggesting its rarity. The youngest patient having Hemangiopericytoma was two and a half years old. There is no any gender or racial predominance (2, 3). Involvement of Nose and Paranasal sinuses with Hemangiopericytoma is less than 5%. Multiple etiologies like hypertension, trauma, steroids etc. have been proposed but none proved (1, 3). Study done by Kumar R, Corbally M. revealed genetic involvement. The most Hemangiopericytoma are diploid and have t(12;19)(q13;q13) translocation (4). Due to its rarity a proper line of management has not been established to tackle this tumour as some considered extensive excision opposite to some suggesting regular follow up with serial CT/MRI as it is a slow growing tumour (1, 4, 5).

The tumour arises from pericytes that lines the capillaries. Diagnosis is based on histopathology so it requires differentiation from glomus, angiofibroma, angiosarcoma, lymphoma, hemangioendothelioma, fibrous histioma etc. Immunohistochemistry reveals strong positive with vimentin (6). The course of Hemangiopericytoma of nose and sinuses is benign compared to other sites with low rates of recurrence, metastasis and mortality. (5). It may cause dacryocystitis due to involvement of Inferior turbinate (7). Endoscopic excision is the treatment of choice for local accessible lesions. Endoscopic approach allows direct access to the lesion, so accurate assessment can be made preserving the physiological function of nose. Wide excision or craniofacial resection will be required for extensive lesion having involvement of ethmoid, cribriform plate or having intracranial extension. Use of radiation as adjuvant or palliation therapy is not

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clearly demonstrated (1, 2, & 8). Chemotherapeutic agents i.e. Adriamycin, Actinomycin D, Cyclophosphamide, Methotrexate, Vincristine may achieve partial or complete response (9). Use of pre-operative embolization was documented in some studies but was not done in our case and tumour was removed without much bleeding (2). High recurrence rate is correlated with incomplete removal which varies from 7-20% (7, 10). Poor prognosis is associated with a tumour > 6.5 cm and histology suggesting necrosis, atypia, and higher number of mitosis (11). Due to its histopathological similarity with Glomus tumor, Sino-nasal Hemangiopericytoma may be referred as Glomangio-pericytoma according to WHO (12).

CONCLUSION

Due to its rarity and varied picture from other sites, hemangio-pericytoma of nose and paranasal sinuses requires lifelong follow up. Endoscopic approach gives direct access and completeness of excision for small tumour.

Note: Permission from Institutional Ethics Committee was taken.

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CASE REPORT

Rare presentation of retroperitoneal Schwanomma

Dr. M. Sadiq Kazi, Dr. Tripathi U.B.

KEY WORDS : Schwanomma, retroperitoneal

INTRODUCTION

A schwanoma is a benign nerve sheath tumor composed of Schwann cell, which normally produce the insulating myeline sheath covering peripheral nerves. They are reported most commonly in the head and neck, also flexor aspect of upper and lower limbs, which account for at least 90% of cases. All other sites, including the retroperitoneum, are rare, accounting for less than 10% of cases [1, 2]. 3-3.2 % of benign Schwannomas occur in retro peritoneum[3-5]. Schwannomas are relatively slow-growing. Schwannomas are mostly benign and less than 1% become malignant, degenerating into a form of cancer known as neurofibrosarcoma. Malignent transformation occurs in 1.7 % of retroperitoneal tumors..

Here we present the clinical feature and therapeutic implication of the patient affected by retroperitoneal schwannoma who was operated for retro peritoneal lump in abdoman.

CASE REPORT

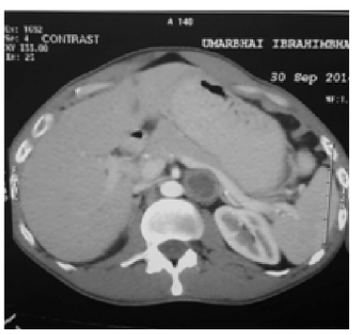
A 34-year old man, presented with chronic abdominal pain and weight loss during last few months..

He described constant, moderate pain in epigastric region associated with fever occasionally ; other constitutional symptoms were 9 kg (15% of his total weight) weightloss.

He was investigated for cause of above mentioned symtomps... ultra sound suggestive of hypoechoic lesion with central liquefication in upper abdoman posterior to pancreas suggestive of ? liquified node of 34-37 mm size and other investigation sr.widal positive ,WBC count 2400/cmm and platelet count 111,000/cmm. He was given anibiotic trial for one month.

After one month patient had no relief in symptomps and repeat sonography suggestive of same findings hence CT scan done... which s/o 35-34-28 size well defined peripheral enhancing lesion with central non enhancing necrotic area in left para aortic region as described above findings are suggestive of necrotic lymph node. (Figure 1)

Figure 1

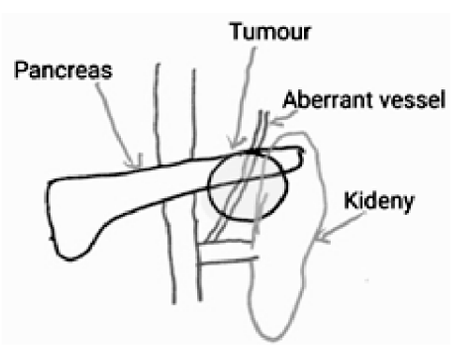


He was then referred for further evaluaiton and mangement. He was further investigated which suggestive of little increse (but still under normal value) in WBC count and platelet count. Furhter investigation suggest TB gold positive and Pet scan suggestive of increse FDG uptake in necrotic left para aortic lymph node lesion at the level of renal hilum. Size of lesion was 31-26 mm size, with no evidence of metabolically active disease seen elsewhere in the body...He was started four drug AKT.

Repeat evaluation after three months s/o no change in nodal lesion. CT guided biopsy done from lesion s/o ganglioneuroma. CA-19.9 was 54.9 U/ml (normal up to 37 U/ml), IHC markers (S-100, Synaptofysin, Chromogranin) also confirmatory for ganglioneuroma.

He was taken for exporatory laparotomy. Lesion was present at renal hilum juxta duodenum and superior to renal artery Figure 2. Lesion was not adherent ot surrounding and was excised completely and sent for biopsy.

Figure 2



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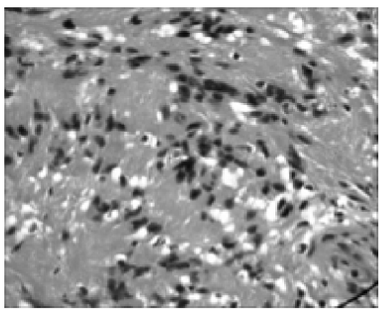
Patient was doing nicely in post operairve period and recovered uneventfylly. Post operative blood investigation suggestive of WBC and Platelets counts were in normal range. He was discharged on 5th postoperative day. We could not explain preoperative neutopenia

Figure 3



The pathological microscopic description shows an encapsulated tumour composed of interesting fascicles of spindel cells with verrocay bodies, hyalinised blood vessels, cystic and myxoid changes and mild inflamation.

DISCUSSION

Schwannoma arising from Schwann cells are among Schwannoma-neurofibromas entity [6,7].

A review of literature indicates most Gastro Intestinal involvement among 20-50 year old patients.

However, females outnumbered males, with a ratio of 2:1[8, 9].The occurrence of isolated Schwannoma in a retroperitoneal organ such as the pancreas is extremely rare. Otherwise in many cases affects occurrence as a component of Von-Recklinghausen's disease [10]. Schwannomas are encapsulated, single lesions with distinct borders. So so complete excision is possible surgically. It merely shows adhesion to surrounding stryctures. But if it is there has to sacrifice surrounding structure if possible surgically to avoid recurrance.

Histologically it contains two discrete components Cellular component (Antoni A) arising from spindle cells Hypocellular component (Antoni B) accompanied with degenerative changes [8, 11].

Delayed Diagnosis may be attributed to vast retroperitoneal cavity and Variety of symptoms ranging from vague abdominal pain, distention and secondary hypertension, renal colic may be present[12].

Pre-operative diagnosis modalities; varying from ultrasound and ct to MRI may facilitate the diagnosis [11, 12], but no specific imaging exists [13].

The features suggesting schwannoma in MRI T1, T2 signal imagings include hypo intense, hyper intense mass respectively[13, 14].

The treatment of choice for retroperitoneal schwannoma is complete surgical resection [15]; particularly in those without expected response to chemo radiotherapy. Some authors believe that complete resection is ideal, an extensive operation sacrificing of vital structures may bewarranted in order to achieve negative margins [16, 17].

In patients who underwent partial resection, some investigators have even reported recurrencerates of up to 10-20% even in benign circumstances, thus emphasizing the importance of a complete resection [18].

In our patient, classical complete surgical excision done without damaging any vital structure nearby.

CONCLUSION

- Schwanoma is tumor arising from nerve tissue.
- It is difficult to diagnose preoperatively by imagimg or other markers.
- Normally it is not adherant to surrounding so radical resection would be the treatment of choice.
- Even if adherant to surrouding structure wide radical excision with sacrificing such structure is advisable.
- Because even in benign case if not completely excised recurrance rate is high.
- It may be part of generlised neurofiberomatosis type-1(Von-Recklinghausen's disease) which should diagnose and treated accordingly.

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CASE REPORT

Extra Ovarian Primary Peritoneal Carcinoma

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KEY WORDS : Extra Ovarian Primary Peritoneal Carcinoma (EOPPC)

ABSTRACT

Extra Ovarian Primary Peritoneal Carcinoma a relatively newly defined disease that develops only in women accounts for approximately 10% of cases with a presumed diagnosis of ovarian cancer. EOPPC is an adenocarcinoma that develops form the peritoneum lining of the pelvis and abdomen and is characterised by abdominal carcinomatosis, uninvolved or minimally involved ovaries. EOPPC is similar in clinical presentation, histologic appearance and response to serous ovarian carcinoma.study in Denmark in our setup.

CASE REPORT

A 47 years old female presented with distention of abdomen and pain in abdomen since 2.5 months her sonography reports shows moderate ascites. She had history of weight loss on examination per abdomen moderate amount of ascites found otherwise all the systems are normal. Ascetic fluid tapping shows positive for malignant cells suggestive of metastatic adenocarcinoma. Her CA-125 report was very high. Her CT abdomen shows diffuse omental thickening with mix solid cystic lesion in left ovary moderate ascites was noted. Considering primary in left ovary neo adjuvant chemotherapy in form of carboplatin plus paclitaxol was given. After 15 days repeat sonography shows gross reduction of ascitic fluid. With one cycle of chemotherapy in form of carboplatin plus paclitaxol patient was responded dramatically so, neoadjuvant chemotherapy will vary from patient to patient and cycles of the neoadjuvant chemotherapy varies individually. Considering the gross reduction of the disease, decision for the surgery was taken and remaining chemo was planed postoperatively. All other blood investigation, X-ray chest and ECG were normal. Preoperative CT scan shows no lymphadenopathy in para-aortic or illiac regions.

Patient was operated under general anaesthesia for ovarian mass. Pan hysterectomy with omentectomy done. Omentum was very badly rolled up and adherent with transverse colon. On exploration no gross lymphadenopathy in illiac and para-aortic regions, so no lymph-node sampling was done. Tube drain kept. Closure done. Post operative recovery was uneventful. Second cycle of chemotherapy in form of carboplatin with paclitaxol was given on 6th post. Op day.

Final HPE shows tumour cells in right ovary were very superficial and majority of tumour was in omentum. Considering this final HPE was reported as a EOPPC. Post operatively remaining five cycles of chemotherapy were completed.

Patient was operated on 26-02-2016 and recent status of the patient in august 2016. Patient is absolutely fine. Patient is doing well.

DISCUSSION

EOPPC, a relatively newly defined disease is a rare adenocarcinoma that arises in the peritoneum. Most reported cases of EOPPC have seen in women, usually elderly. However rare cases have been reported in children (6) and in males (7). At laprotomy all most all cases shows diffuse peritoneal implants, which usually involved the omentum and upper abdomen. Although EOPPC always involved the full thickness of the omentum, invasion to other abdominal organ is rare. The ovaries are almost always of normal size and shape and frequently display surface tumour implants, which may be focally invasive, rarely may they be normal grossly and microscopically.

The diagnosis of the EOPPC is typically made by exclusion after both operative assessment and pathological study. If ovaries seem to be normal with wide spread disease elsewhere in the abdomen EOPPC becomes a leading diagnostic possibility. However because surface involvement of ovaries present in approximately 96% of the cases the distinction between EOPPC and epithelial ovarian carcinoma may only be made after histological examination to evaluate extend of ovarian invasion by tumour (5).

EOPPC spreads mainly transperitonealy however lymphatic and blood-borne metastases have been suggested.

The light microscopic, histochemical, immuno histochemical, and ultra structural features of EOPPC are

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similar to those of ovarian serous carcinoma. Thus EOPPC appears has a high grade, purely epithelial neoplasm with frequent mitotic figures, necrosis, slitlike glandularspaces and psammoma bodies (11).

In order to differentiate EOPPC from papillary serous adenocarcinoma of the ovary. The gynaecologic oncology group has stipulated that following criteria be met (2).

1. Histology must be predominantly serous or identical to any grade of ovarian papillary serous tumour.
2. The ovaries are of normal size or enlarged by a benign process.
3. The involvement in the extra ovarian sites must be greater than the involvement on the surface either overview.
4. The ovarian component must be non existence or confined surface epithelium.

EOPPC must be differentiated from malignant mesothelioma, benign papillary mesothelioma, metastatic peritoneal carcinomatosis, endosalpingosis, and psammocarcinoma of the peritoneal.

Primary serous border line tumours of the peritoneal have also been reported affecting younger patients and having the microscopic features of ovarian border line serous tumour. These primary serous border line tumours have an excellent prognosis. The pathogenesis of EOPPC has been controversial. Some authors believe that embryonic germ cells rests remain along the gonadal embryonic path way and that EOPPC develops from malignant transformation of these cells.

Treatment usually includes abdominal hysterectomy, bilateral salpingo-oophrectomy and tumour de bulking followed by chemotherapy. Surgery remains important for both diagnosis and therapy of EOPPC. Cisplatin based regimes have been the most common first line chemotherapy regimens used in patient if EOPPC. These authors (14) demonstrated that patient who received cisplatin based regimes have significantly longer survival than patient who do not and them patients given combination chemotherapy (cisplatin plus taxane, cisplatin plus cyclophamide, cisplatin plus doxorubicin plus cyclophamide) survives longer than those treated with single agent regimens.

3983 URMILABEN 10X H&E OMENTAL TUMOR

Sections reveal tumor in omental tissue composed of variable sized glands lined by columnar epithelium with pleomorphic and hyperchromatic nuclei and moderate pale cytoplasm.

CONCLUSION

Recognition of EOPPC seems to be increasing. Patients with EOPPC should be reported separately from those with ovarian carcinoma but should treat in a similar fashion. Recent reports of the prognostic significance of residual tumour mandate that surgeons should make every effort to achieve maximal tumour debulking when faced with occasional patient who has abdominal carcinomatosis, normal sized ovaries and no identifiable primary tumour.

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CASE REPORT

Cesarean scar pregnancy

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KEY WORDS :scar pregnancy, ectopic pregnancy

INTRODUCTION

The implantation of a pregnancy within the scar of a previous cesarean delivery is the rarest form of ectopic pregnancy.[1] A computer Medline and bibliography search has yielded only 112 cases reported in the English language from 1966 through 2005.[2] Jurkovic et al recently described a series of 18 cesarean delivery scar pregnancies, with a prevalence of 1 per 1800 pregnancies.[1,3]

If the early diagnosis has been made, treatment options are capable of preserving the uterus and subsequent fertility. However, a delay in either diagnosis or treatment can lead to uterine rupture, hysterectomy, and significant maternal morbidity or even death. Although expectant and medical managements have been reported, termination of a cesarean scar pregnancy by laparotomy and hysterotomy, with repair of the accompanying uterine scar dehiscence, may be the best treatment option.[1]

Early sonographic diagnosis allows appropriate treatment to preserve uterine integrity and future fertility. In previously published reports, this condition was treated successfully with intramuscular methotrexate (MTX).[4-8] We describe a case of cesarean scar pregnancy in whom appearance of fetal cardiac activity and rising serum ß-hCG titre and development of acute abdominal pain after treatment with 2 doses of MTX and required laparotomy.

CASE REPORT

A 26 years old female, gravida 3 para 2 with previous history of 2 cesarean section, 1st before 3 years for oligohydroamnios, 2nd before 3months for previous cesarean section and breech presentation, was admitted with complaints of pain in hypogastric region and right iliac fossa and vaginal spotting for one month. Pain was refractory to oral analgesia. On examination her general condition was fair. Her weight was 75 Kg. Abdominal examination did not reveal any abnormality. Per speculum examination showed spotting from cervical canal. Bimanual vaginal examination, the uterus was anteverted ,bulky and external os of cervix was closed. Her urine pregnancy test was positive. Her S ß hCG level was 3082mIu/ml. Investigations showed hemoglobin 11.2g/dl, total WBC count-6000/cumm, platelet count - 2.94lac/cumm. Transvaginal ultrasonography showed empty uterine cavity and cervical canal, location of gestational sac measuring 17mm corresponding 5 weeks 5 days in anterior uterine wall at the level of isthmus embedded in right side of previous scar. There was absence of myometrium between gestational sac and urinary bladder. Yolk sac was present. Crown-rump length was 2.5mm corresponding 5 weeks 5 days fetal maturity. Fetal cardiac activity was absent. A clinical diagnosis of cesarean scar pregnancy was made. Patient was admitted and treatment started with medical therapy- First dose of methotrexate 1mg/kg body weight on day 1 followed by inj. Leucoverin 0.1 mg/kg body weight on day 2. Second dose of methotrexate on day 3 followed by inj. Leucoverin on day 4 was given. On day 4 patient complained of increased abdominal pain. Transvaginal ultrasonography showed gestational sac measuring 22mm corresponding 6 weeks 2 days and Crown-rump length was 6mm corresponding 6 weeks 1 days fetal maturity. Fetal cardiac activity was present. Her S ß hCG was 5904mIu/ml. Investigations showed hemoglobin 9.2g/dl, total WBC count-4280/cumm, platelet count -2.73 lac/cumm. Decision of laparotomy was taken, because of appearance of fetal cardiac activity and rising serum ß-hCG titre and development of acute abdominal pain after treatment with 2 doses of MTX. There was popping out of gestational sac through right side of previous scar with band of omental adhesion, minimal free blood in anterior pouch. Gestational sac was removed and uterine scar repaired. Histopathological examination showed fibrous tissue with chorionic villi, trophoblastic cells proliferation & decidual stroma with haemorrhage in it suggestive of cesarean scar pregnancy. Post operative course was uneventful.

DISCUSSION

Cesarean scar pregnancy, an extremely rare type of ectopic pregnancy, is defined as the embedding of the gestational sac in the scar of a previous cesarean

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section.[1] In 1978, the first case of cesarean scar pregnancy was reported by Larsen and Solomon, as a post-abortal haemorrhage due to what they described as uterine scar saculus.[1]

The exact cause of cesarean scar pregnancy is unknown. Several hypotheses have been proposed. Vial et al[4] proposed two different type of cesarean scar pregnancy - first is an implantation on prior cesarean scar with progression towards the cervicoisthmic space or the uterine cavity. Such cesarean scar pregnancy may progress to a viable birth but with the risk of life threatening bleeding and second is a deep implantation into a cesarean scar defect growing towards the bladder and abdominal cavity , a type more prone to rupture. Other hypothesis argues that the conceptus enters the myometrium through a microscopic dehiscent tract or defect in the cesarean section scar.[5]

In 2000, Seow et al presented the first case of cesarean scar pregnancy following in vitro fertilization-embryo transfer. Consequently, Seow recommended that in patients with a history of a cesarean section, the embryo should be transferred at least 4 cm from the cervix so as to avoid the caesarean scar and a cervical pregnancy.[8]

Ultrasonography is a useful tool for diagnosing Cesarean scar pregnancies. Because outcomes and treatments may differ, a cesarean scar pregnancy must be distinguished from other types of abnormally implanted pregnancies, including cervical, cervicoisthmic, and cervicoisthmic corporeal pregnancies. Cesarean scar pregnancy is different from intrauterine pregnancy with placenta increta or percreta in that it is more aggressive, occurs in the first trimester, and involves the complete embedding of the gestational sac in the myometrium. The myometrium between the bladder and the sac becomes thinner or disappears due to distension of the sac. Only the thin, serosal layer is apparent, portion of the sac is not covered by myometrium. Robert et al[9] described a cesarean scar pregnancy as having a “salmon red” appearance under a laparoscope. Since the advent of endovaginal echography and MRI, it has been possible to make a diagnosis earlier in the gestation, and to use a more conservative approach to treatment. Strict imaging criteria must be used in performing the diagnosis: an empty uterus, empty cervical canal, development of the sac in the anterior part of the isthmic portion, and an absence of healthy myometrium between the bladder wall and the gestational sac. On sagittal section, a discontinuity on anterior wall of uterus running through the amniotic sac, high velocity with low impedance peri-trophoblastic vascular flow clearly surrounding the sac is proposed in doppler examination . This third criteria permits the differentiation of a pregnancy implanted in a cesarean section scar from a cervical or cervicoisthmic pregnancy.[1,3]

The optimal treatment of cesarean section scar ectopic pregnancies is unknown. Presentation of the patient often dictates the mode of treatment, given that many patients present with hemoperitoneum and require hysterectomy. There are few reports of cervical pregnancies successfully treated conservatively with MTX and expectant management.[4-8] These reports referred to several protocols: systemic single-dose MTX or multiple-dose MTX with alternate-day folinic acid rescue or MTX injection into the gestational sac. The use of MTX resulted in resolution of these pregnancies without surgical intervention. Godin et al described a transvaginal injection of potassium chloride into the fetal thorax and methotrexate (MTX) to the sac and surrounding myometrium of a 9-week viable pregnancy in an existing uterine scar. Complete resolution of the pregnancy was observed. However, dehiscence of the uterine scar was noted 16 weeks later by hysterosalpingography.[5] However, in some cases, hemorrhage occurred after MTX treatment, requiring emergency treatment.[1,4,6] In the case reported by Lai et al. two weeks after endovaginal sonography-guided intralesional delivery of MTX into an ectopic gestational sac, an emergency laparotomy was performed at the onset of active vaginal bleeding from the ruptured uterine scar.[6] Haimov-Kochman R et al. suggested that non-invasive therapy should be considered in suitable cases of cesarean scar ectopic pregnancy. In cases discovered at no more than 6-8 week's gestation without fetal cardiac activity, MTX delivery and expectant management may be a safe treatment alternative.[7] Our case represents a failure of conservative treatment with MTX.

Some authors propose that dilation and curettage should not be first-line therapy due to the risk of perforation and catastrophic hemorrhage[10] Lee et al reported laparoscopic resection of a cesarean section pregnancy.[10] Graesslin et al reported the use of systemic methotrexate followed by dilation and evacuation with success.[11] Uterine artery embolization to reduce hemorrhage has also been described as adjunctive therapy.[12]

Non-surgical treatment options (which include systemic and local MTX, potassium chloride, and hyperosmolar glucose) even when successful could be expected to leave the uterine scar defect that will accompany cesarean scar implantation. The potential for an unprepared scar dehiscence that will affect future pregnancies is left to speculation. Surgical resection, in this case, offered the opportunity to remove the pregnancy and to repair the defect simultaneously. As soon as the diagnosis is confirmed, primer surgical treatment by laparotomy should be recommended.

Fig 1: Transvaginal ultrasonography showed empty uterine cavity and cervical canal, location of gestational sac(GS) in anterior uterine wall at the level of isthmus embedded in right side of previous scar.(Longitudinal section)

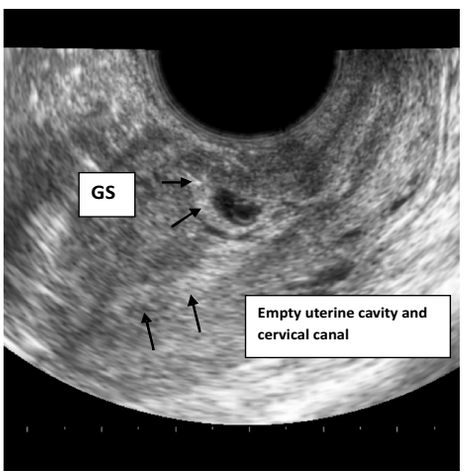


Fig 2 : Transvaginal ultrasonography showed empty uterine cavity and cervical canal, location of gestational sac(GS) in anterior uterine wall at the level of isthmus embedded in right side of previous scar.(Transverse section)

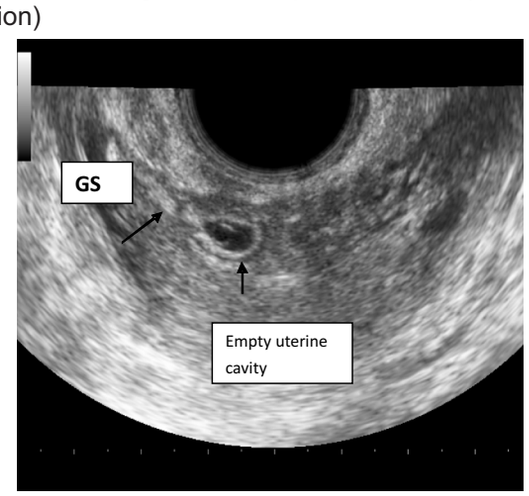


Fig 3: Color Doppler shows peri-trophoblastic vascular flow clearly surrounding the sac

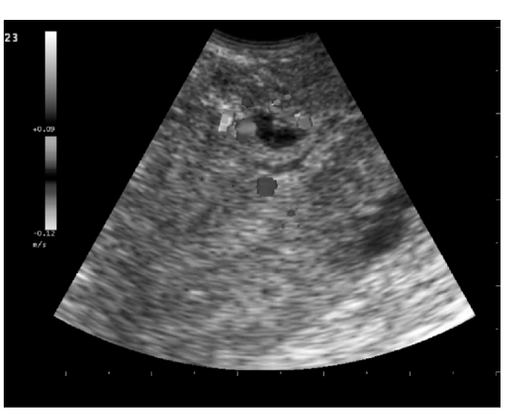
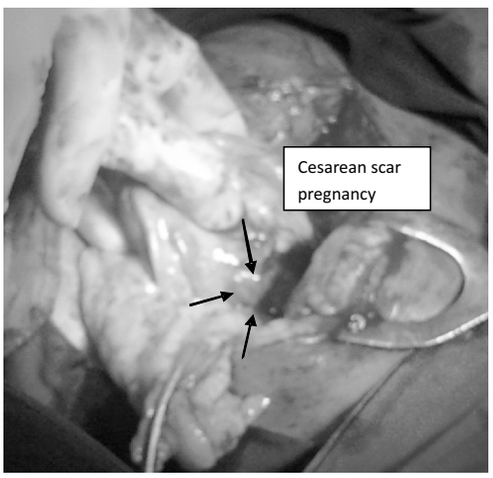


Fig 4. Popping out of gestational sac through right side of previous scar with minimal free blood in anterior pouch.



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