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Motto : Knowledge is Power - Unity is Strength
Theme : Health & Happiness for Her

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President
Dr. Sunil Shah
+91 90999 77077
sunilshah0501@gmail.com

Hon. Secretary
Dr. Akshay C. Shah
+91 94264 85805
drshahakshay1974@gmail.com

HEALTH & HAPPINESS FOR HER

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Dr. Nita Thakre
+91 98250 42238
drthakre@gmail.com

Vice President
Dr. Parth Shah
+91 94296 17556
parthpjs@yahoo.com

Hon. Treasurer
Dr. Munjal Pandya
+91 97129 11784
munjal171184@gmail.com

Hon. Jt. Secretary
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+91 98254 40292
mehulsukhadiya@yahoo.com

Clinical Secretary
Dr. Arati Gupte
+91 96620 28127
arati2811@gmail.com

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TEAM AOGS MESSAGE

Dr. Sunil Shah
President

Dr. Akshay C. Shah
Hon. Secretary

Dearest AOGS colleagues,

We are in the middle of the biggest and longest festival in the World **Navratri**. We pray and take blessings of Ma amba, Durga and Kali. People worship, do fast and play dandia- Garba these days. Soon after it will be one of the biggest and most important festivals of Hindustan Dipawali will come and we celebrate the homecoming of lord Ram after winning and triumphing over Ravan. So from me and team AOGS wishing you all happy navratri and happy dipawali and new year in advance.

Dear friends we have one of the biggest conferences of this year that will be held by AOGS that's the ICOG FOGSI conference on 18-19-20th October at Karnavati club. Many renowned local, national and international speakers are going to come and address the conference for spreading their knowledge. I request each and every one to take advantage of it and register for it.

I thank each and every contributors for this and previous bulletin. I also thank all advertisers also.

Please keep on suggesting innovative and useful ideas to team for betterment of AOGS and AOGS members.

Looking forward....

Dr. Sunil Shah I Dr. Akshay Shah

RHABDOMYOMA

Dr. Swati Trivedi
ICOG-Fellow Fetal medicine

Dr. Parth Jay Shah
Consultant and Fetal Medicine expert

Cardiac tumors are rare mesenchymal or hamartomatous nodules arising from the cardiac layers.

Classification of cardiac tumors

1. Rhabdomyomas - encapsulated tumors and often multiple in number. They arecharacteristically of late onset, and often shrink significantly in the neonatal period.

2. Teratomas - encapsulated tumors and usually single. They tend to grow within the pericardial cavity, frequently leading to large pericardial effusions.

3. Other occasional tumors—fibroma, haemangiomas, and myxomas - usually single tumors.

Rhabdomyomas are the most common benign tumors of striated muscle origin. They account for two-thirds of the cardiac masses detected in the fetus and neonate. Rhabdomyomas account for 1% of all cardiac problems diagnosed before birth.

Pathophysiology

Cardiac rhabdomyoma belongs to the hamartomatous subtype of neoplasms. It is most often identifiable in fetus at 20 - 30 weeks of gestational age. With the advent of advancements in imaging technology, tumors are identified more in the fetal series than in the postnatal series.

A rhabdomyoma grows in the myocardium involving the lower chambers of heart - the left and right ventricles. More rarely tumor grows to involve the interventricular septum and the atrioventricular valves. It can be usually sporadic. A strong clinical association often exists with an autosomal dominant condition i.e. Tuberous sclerosis syndrome, which is characterized by benign hamartomas in multiple organ systems- brain, kidneys, eyes, skin, and lungs. Diagnosis of tuberous sclerosis is usually clinical. Confirmation can be done by genetic testing (looking for TSC1 and TSC2 gene mutations).

Ultrasound diagnosis

Rhabdomyomas are usually detected on the four-chamber view. They appear as hyperechoic, well defined and round or oval masses. These tumors may arise anywhere in the myocardium. In a few cases, they may completely replace some areas of the myocardium, appearing as non-vascular, homogeneous, hyperechoic regional myocardial thickening. Figure 1,2 and 3

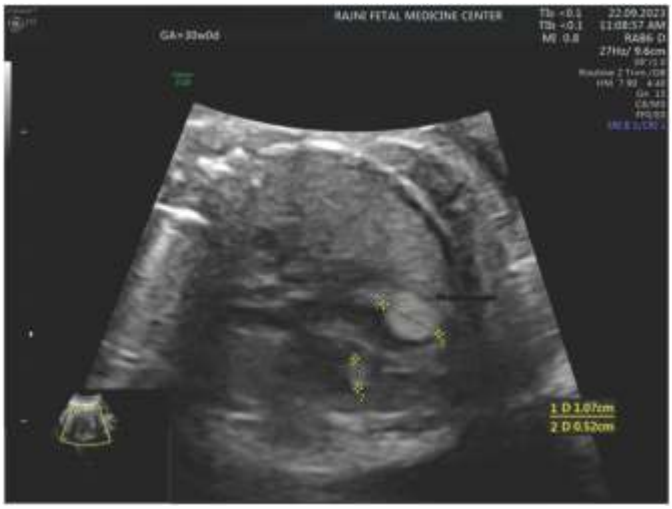

Figure 1. Transverse four chamber view of heart demonstrating Rhabdomyoma cardiac tumors measuring 10.7 mm and 5.2 mm

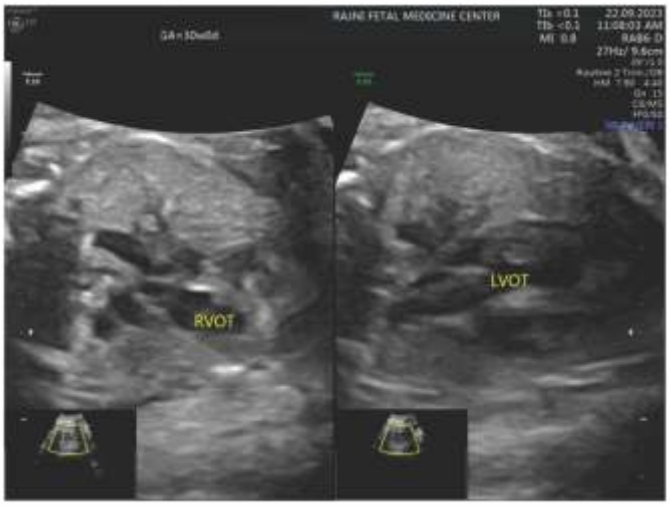

Figure 2. Outflow tracts LVOT and RVOT- intact and no obstruction seen

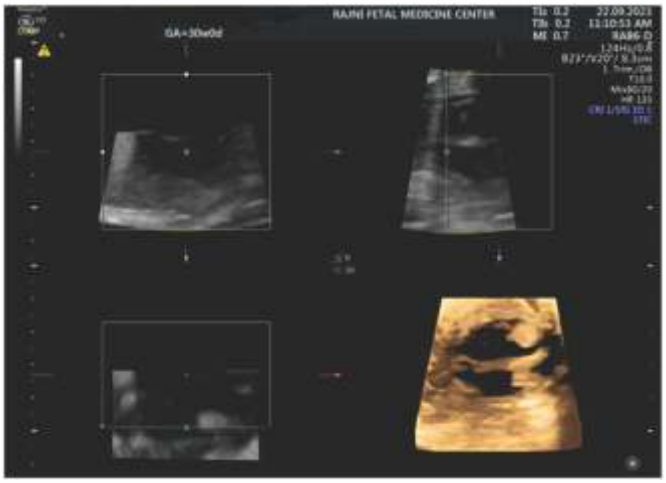

Figure 3. Multiplanar view with Rhabdomyoma

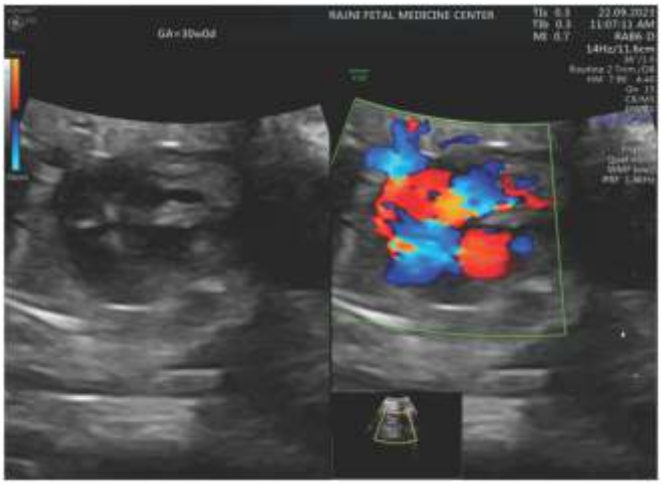

Figure 4. Using colour doppler to demonstrate patency of atrioventricular valve, no obstruction here.

Rhabdomyomas evolve during the course of pregnancy. They appear in the late second trimester, growing steadily until 32–35 weeks, when they reach a plateau. After birth, rhabdomyomas shrink significantly or even disappear macroscopically. This biphasic behaviour may be related to the maternal hormonal milieu, which may stimulate tumor growth in the third trimester. After birth, the neonate is no longer in contact with maternal estrogens, and this allows the tumor to shrink as the mitotic hormonal stimulus is withdrawn.

Colour Doppler assessment may help in:

- detecting dynamic outflow obstruction (if the tumor is located in the ventricular outflow), and
- checking the patency of atrioventricular valve (if the nodule is located just below the atrioventricular plane) figure 4.

Spectral Doppler may be used to quantify the outflow obstruction. Four-dimensional echocardiography is of little use in cases of cardiac tumors.

Differential diagnosis.

1. Hyperechogenic foci and the rare dystrophic calcifications- hyperechogenic foci do not have any continuity with the myocardium as they are located on the chordae tendinea of the atrioventricular valves, and are very small.
2. Cardiomyopathy- rare form of diffuse rhabdomyomatosis can be mistaken for cardiomyopathy.
3. Inflammatory myofibroblastic tumor

Association with other malformations

1.Risk of chromosomal anomalies.

This is virtually absent.

2.Risk of nonchromosomal syndromes.

It is extremely high for multiple rhabdomyomas.

Lesser association with single rhabdomyomas which form the majority (90%).

Occasional association with tuberous sclerosis (TS) has been described which is an autosomal dominant inheritance disorder.TS can be diagnosed at birth, with recognition of the typical café-au-lait spots

Obstetric management

Karyotyping is not mandatory, as the risk of chromosomal anomalies is low. However, prenatal genetic counselling and genetic assessment of the parents are indicated in order to disclose a possible, previously unknown presence of Tuberous Sclerosis in one of them.

Obstetric management requires follow-up of the tumors:

- If active growth is seen, and threaten to cause complete valve obstruction early delivery.
- If tachyarrhythmia is associated transplacental antiarrhythmic therapy with digoxin (rhythm disturbances secondary to cardiac tumors are often unresponsive).

Delivery should be organized in a tertiary referral center, in order to provide cardiologic and respiratory assistance if needed.

Complications

The complications of cardiac rhabdomyoma include the following:

- Infections
- Arrhythmias (bradycardia and tachycardia)
- Congestive heart failure
- Hemodynamic compromise

Prognosis

The surgical mortality rate for cardiac tumors is extremely low.

The tumor volume is the main prognostic factor.

Poor prognostic factors:

1. Large tumors obstructing the atrioventricular inlets or the outflows.

2. Tachyarrhythmias resulting from mechanical stimulation of the atrioventricular conduction system by tumors located at that level (cardiac failure → hydrops).

The patients who underwent surgery for the removal of rhabdomyoma have a fair to good prognosis.

Survival rates are high; and deaths (due to refractory cardiac failure and tachyarrhythmias) are usually confined to the first months of life.

The long-term prognosis in the case of TS is guarded because of:

- 30% risk of mental retardation,
- possible sequelae requiring repeated surgery.

Postnatal course

Postnatally85% cases of cardiac tumors regress significantly, hence do not require any medical or surgical treatment. The remaining 15% of cases are symptomatic at birth, and may need medical and/or surgical intervention.

In the case of multiple rhabdomyomas, tuberous sclerosis must be ruled out by serial examinations including cerebral MRI,electroencephalography, renal US, and an ocular fundus examination. ECHO is preferred for cardiac evaluation. mTOR inhibitors are a consideration in cases associated with tuberous sclerosis.

Surgical resection is not indicated unless the tumor is symptomatic. Indications for surgical resection of tumor mass are:

1. Tumors causing severe pleuro-pericardial effusions (which are responsible for cardiac tamponade and low cardiac output)

2. Tumor-induced tachyarrhythmias (unresponsive to medical antiarrhythmic treatment)

3. Tumors causing hemodynamically significant valve obstruction

Orthotopic heart transplantation is considered in extremely rare events when the tumor is so large that it has replaced the normal myocardial tissue

Fertility, ART & Reproductive Outcomes in Adenomyosis

Dr. Jwal Banker
Banker IVF & Women's Hospital

Dr. Manish Banker
Banker IVF & Women's Hospital

Introduction

Adenomyosis is best defined by Bird in 1972 as “the benign invasion of endometrium into the myometrium, producing a diffusely enlarged uterus which microscopically exhibits ectopic non-neoplastic, endometrial glands and stroma surrounded by the hypertrophic and hyperplastic myometrium”. In the past, adenomyosis was thought to be present only in parous women. However, it is frequently encountered in nulliparous infertile patients also.Puente et al found that the prevalence of adenomyosis was 24.4% and 22% in women aged ≥40 years ≤40 years, respectively. This was found to be higher in women with recurrent pregnancy loss (38.2%) and previous ART failure (34.7%) when compared with the rest.

Infertility/ subfertility due to adenomyosis

It is hypothesised that adenomyosis might cause infertility by:

- Altering the normal myometrial architecture and function by affecting the uterine peristalsis and/or by interfering with sperm transport.
- Altering the normal architecture of the fallopian tubes and this results in abnormal peristalsis with increased intrauterine pressure.
- Less rapid intra-uterine sperm transport due to dysperistalsis caused by adenomyosis.
- Altering the shape of the uterine cavity itself, in severe cases causing a ‘T-shaped’ uterus.
- Altering signalling at a molecular and genetic level causing alteration of estrogen and progesterone receptors in these women which can reduce the β-3 integrin secretion and hinder receptivity as well as dysregulation of other factors like HOXA10, LIF, IL-6, cytochrome P450.

Effect on pregnancy outcomes

Given the fact that adenomyosis causes abnormal uterine shape, vascularity and peristalsis, it has been thought that this can affect the implantation of the embryo. A recent meta-analysis by Younes et alfound that implantation was significantly lower in women with adenomyosis undergoing ART. He evaluated 12 different studies and also found significantly reduced clinical pregnancy rates(CPR) per cycle. An earlier meta-analysis also showed that women with adenomyosis had a 28% reduction of CPR in IVF/ICSI cycles as compared to controls .

Adenomyosis causes changes in the myometrial architecture and thus affects the vascularity by distorting the spiral arteries. This vascular change is assumed to cause increased miscarriages in these women.In the study performed using donor oocytes on women with and without adenomyosis with similar age and reason for infertility, there was a significantly increase in miscarriage rates in the affected women (13.1% vs 7.2%).

By reducing the CPR and increasing the abortions, it is safe to assume that the overall live birth rate (LBR) will

also drop.Younes et al. made it quite evident in their meta-analysis that there is a 41% drop in LBR in patients with adenomyosis compared to healthy women and more recent studies also agree to that.

Surgical Management of Adenomyosis related Infertility

The surgical management of women with associated subfertility is highly controversial, and there remains an overall lack of consensus regarding the value of conservative surgery with or without medical management to improve reproductive outcomes.Factors like the method of removal of adenomyotic tissue, the degree of residual adenomyosis, the method of reconstructing the uterine wall, postoperative complications, and the interval between the procedure and conception are of immense importance.

From excision of adenomyotic tissue after longitudinal incision of the uterus, to the wedge resection and using classical V-shaped resection technique, multiple newer techniques have been experimented with, including a uterine 'muscle flap' method that stresses fertility preservation.

Another approach using hysteroscopy can be useful in cases with cavity modifications. Regarding such cavity abnormalities induced by adenomyosis, prospective study by Mikos et al evaluating the role of hysteroscopic enlarging metroplasty in women with a T-shaped uterus and infertility demonstrated better live birth and reduced miscarriage rates after the procedure.

Medical Management of Adenomyosis related Infertility

Most of the literature is focused mainly on medical management for adenomyosis associated dysmenorrhoea, abnormal uterine bleeding and pelvic pain, with less focus on associated subfertility. It is still assumed that by reducing the volume of the uterus and the defect, it will help improve fertility outcomes. Treatment options include GnRH-a, oral contraceptives (Ocs), progestins, danazol, and, more recently, selective oestrogen receptor modulators (SERMs), selective progesterone receptor modulators (SPRMs), and aromatase inhibitors (AIs) along with some other experimental drugs.

Along with causing a hypoestrogenic state, agents like GnRH agonists also reduce the expression of aromatase cytochrome P450 in the eutopic endometrium.To assess this on IVF outcomes, Xiaoni Hou et al. recently compared outcomes using long agonist protocols with and without prior suppression by GnRH agonist depots. They found that the suppressed patients needed a significantly longer and higher dose of gonadotropins for stimulation and number of oocytes retrieved were also less. But on the other hand, these women had a significantly higher implantation and live birth rate.

Overall, it seems that all these factors work by reducing the size of the lesion and the uterus and ultimately creating a favourable environment. This can then help improve pregnancy and live birth rates.

Conclusion

Adenomyosis still continues to mystify patients and gynaecologists.A lack of standardization in diagnoses makes this disease challenging to study. Although pain and bleeding problems are common symptoms, sometimes subfertility or even infertility is the only sign of this uterine disease.Existing literature suggests early IVF and embryo culture with or without the use of suppression using GnRH agonists or even aromatase inhibitors prior to embryo transfer can give good and early results. Surgical management, in spite of being helpful in some instances, is still not recommended for all and patient selection guidelines are pending.All cases must be evaluated individually and a personalized treatment plan must be suggested depending on the patients' need and the extent of the disease.

LAPAROSCOPIC REPAIR OF VERY LARGE VESICO VAGINAL FISTULA WITH BILATERAL URETERIC REIMPLANTATION

Dr Pragnesh Shah MD (Gyn),
Visiting endoscopic surgeon, SVIMSR, Smt NHLMMC,
Heli woman's clinic, Motera, Ahmedabad
Apollo Hospital, Bhat, Ahmedabad

Introduction:

"A Vesico Vaginal Fistula(VVF) with its constant odorous, scalding, unimpeded leakage of urine is one of the most devastating surgical complication that occur in women." It can occur after abdominal, vaginal, laparoscopic hysterectomy, obstructed labor, after obstetric hysterectomy, after anterior colporrhaphy, long standing forgotten or neglected intravaginal pessary, malignancy: ca cervix, after radium insertion. It affects physical, mental, social and sexual life of the patient.

There are multiple approaches to manage VVF ranging from conservative management to open surgical repair. The choice of operation for VVF is predominantly a matter of surgeon's preference. Laparoscopy has evolved to be an efficient surgical modality as it has the advantages of less post-operative pain, blood loss, shorter convalescence and minimal scar. CT IVP and Cystoscopy are the two important investigations to confirm diagnosis.

A woman having long standing large vesico vaginal fistula for 40 years which is treated by laparoscopic VVF repair with bilateral ureteric reimplantation.

Case Report:

Patient aged 80 years having c/o leaking of urine p/v since 40 years since after her last delivery may be due to obstructed labor. Her complaints of leaking of urine was exaggerated for 2 years, despite having indwelling catheter. Due to urinary incontinence due to fistula, there was severe excoriation of vulva and perineal skin. On P/S examination, there was large vesico vaginal fistula. CT IVP showed 5.5 cm VVF near ureteric orifices.

Management plan:

The primary objective was to stop leaking even at cost of severe compromised bladder capacity and life-long catheterization (As patient was 80 years old, augmentation cystoplasty was not possible due to her general condition). Patient was planned for laparoscopic repair of fistula with bilateral ureteric reimplantation along with laparoscopic hysterectomy.

Operative steps:

Cystoscopy was performed. Fistulous bladder was dissected from uterus and hysterectomy was completed. Fistula was so large that whole supratrigonal portion of bladder was compromised with bilateral ureteric orifices.

Both the ureters are dissected. Bladder mobilization and wide excision of fistulous tract was carried out. There was only trigonal portion of bladder remaining which was sutured and bilateral ureters were reimplanted.

Post-operative management:

Patient was given anticholinerics to prevent bladder spasm and better healing and broad spectrum antibiotics. Indwelling catheter was kept for 3 months. Cystoscopy carried out after 6 weeks, there was good healing of bladder, ureteric stents removed. Trial of catheter removal was planned along with bladder training in postoperative phase. Surprisingly, fistula did not recur. However, patient has to go for urination every hour.

Discussion:

Laparoscopic repair of Urogenital Fistula though technically challenging, has multiple advantages. This modality is particularly well-suited to work in deep pelvic cavity, less Blood loss may due to tamponade effect of the pneumoperitoneum, which limits bleeding from perivesical and perivaginal venous plexus. During VVF repair, it provides excellent magnified view of the bloodless surgical field whereby accurate dissection in anatomical planes can be done. It permits wide mobilization of the bladder flaps to achieve precise closure of the fistula and tensionless suturing of the bladder. During ureteroneocystostomy repair, it aids wide mobilisation of the bladder to perform vesico-psoas hitch.1

If fistula is near ureteric orifice, better not to compromise adequate dissection and excision of fibrous margin of fistula and proper suturing bladder. Ureteric reimplantation is preferred in such cases rather than preserving ureteric orifice with compromised VVF repair. If fistula is chronic, wide excision of margin is must to prevent recurrence.

References:

Sharma S, Rizvi SJ, Bethur SS, Bansal J, Qadri SJ, Modi P. Laparoscopic repair of urogenital fistulae: A single centre experience. J Minim Access Surg. 2014 Oct;10(4):180-4. doi: 10.4103/0972-9941.141508.

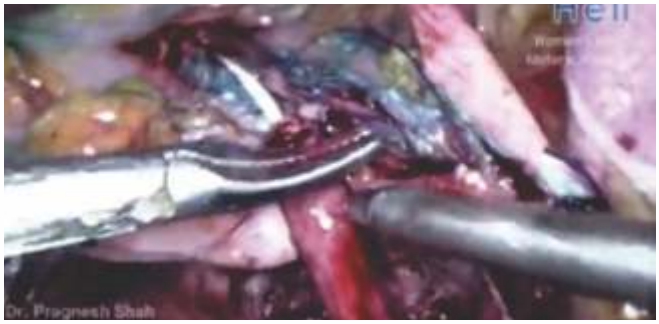

Fig 2: Right ureteric implantation

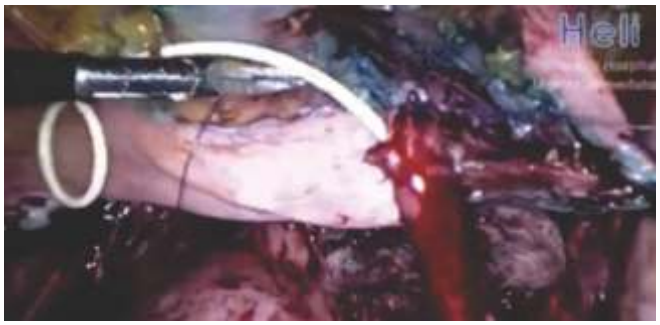

Fig 3: Ureteric stenting

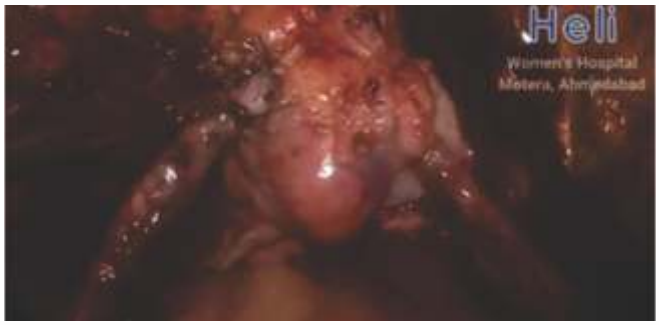

Fig 4: Bilateral ureteric implantation

RETAINED PLACENTA: CASE SERIES

Dr. Purvi M. Parikh

Asst Professor, Department of OBGY, SVPIMSR, Smt NHLMMC, Ahmedabad

Dr. Ayushi H. Vyas

2nd Year Resident, Department of OBGY, SVPIMSR, Smt NHLMMC, Ahmedabad

INTRODUCTION:

Third stage of labour starts after delivery of baby and ends with delivery of placenta. The average duration is 15 minutes in primigravida and 5 minutes in multigravida women, not exceeding more than 30 minutes with active management of third stage and 60 minutes with expectant physiological management.[1]

Retained placenta is diagnosed when placenta is not delivered spontaneously after one hour of delivery of baby. The incidence is estimated between 0.1%- 3%.[2] Risk factors include preterm delivery, history of prior uterine surgery, high parity, abnormally adherent placenta as in Placenta Accreta Spectrum Disorders (PAS), prolonged oxytocin usage, angular pregnancy and IVF conception. [3]

It is one of the leading causes of post-partum haemorrhage (PPH).

CASE 1:

A 28 years old woman, G2P1A1 with previous caesarean section was referred for retained placenta following second trimester abortion before 1 month. She had history of failed Manual Removal of Placenta (MRP) on the same day following abortion. USG was suggestive of adherent placenta inside uterine cavity with increased vascularity on Power Doppler (Fig 1). Patient consulted at our hospital for further management as placenta was inside uterine cavity for one month. On examination, vitals were normal, P/A: soft abdomen and presence of Pfannenstiel scar of caesarean section, P/S: No foul smelling vaginal discharge or bleeding, vaginal swab for C/S was taken, P/V: Uterus anteverted, bulky, reaching up to 2 cm above symphysis pubis.

We counselled patient for conservative management and monitored with serial USG with Colour Doppler study and β hCG titre weekly for one month and then bi-weekly.

Her USG and Colour Doppler showed gradual decrease in vascularity and placental size (Fig 2A,B). Serum β hCG titre was 800 mIU/ML after 30 days of abortion, which was gradually declined and became normal (10 mIU/ML) on day 76.

On post-abortion D98, she was admitted with severe pain in lower abdomen and placenta expelled spontaneously. USG showed empty cavity (Fig2c).

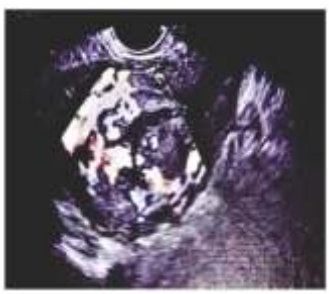

Fig 1: USG on post-abortion D7: shows 5.1×4.4 cm size retained placenta inside uterine cavity with increased vascularity on Power Doppler suggestive of adherent placenta

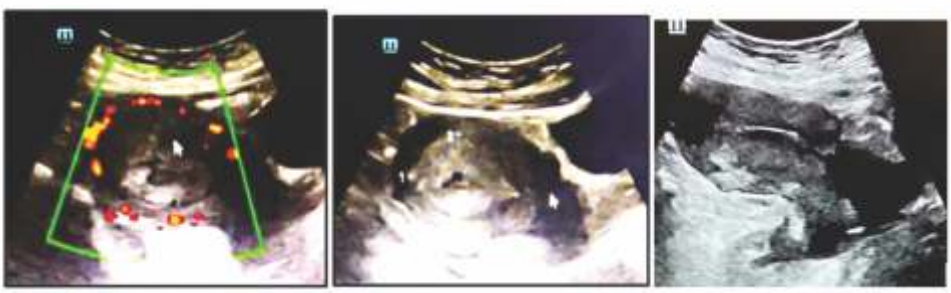

Fig 2: (A) On D21 decreased vascularity and size of the placenta (B) On D42 further decrease in size of placental tissue, (C) On D98 after the expulsion of placenta, empty uterine cavity

CASE 2:

A 25 years old woman G2P0A1 (h/o previous one spontaneous abortion at 2 months) presented at 26 weeks of gestation with severe PIH and as USG findings were suggestive of severe utero-placental insufficiency (very high resistance in umbilical artery AEDF with intermittent reversal). After counselling regarding poor perinatal outcome and high maternal morbidity, patient and relatives agreed for induction of labour with Tab Misoprostol. Patient delivered 600 gm stillbirth child after 48 hours of induction.

Placenta was not delivered after 1 hour of delivery. Patient was shifted to operative room for Manual Removal of Placenta under General Anaesthesia but it could not be separated. Intra-operative USG revealed deeply trapped placenta over right cornu with surrounding thin myometrium <2mm and also absence of retro-placental clear zone at anterior wall of myometrium (Fig 3).

Diagnosis of retained placenta due to angular pregnancy was made. There was no active bleeding, decision was taken for conservative approach and placenta was left in situ. Relatives were counselled regarding conservative approach and may need laparotomy if heavy bleeding or sepsis.

Injectable broad spectrum antibiotics were started and patient was closely monitored for vital parameters including temperature and any active vaginal bleeding, serial CBC, serum creatinine and Procalcitonin. On D1, WBC count was 29000/ mm3, procalcitonin was 5.12 ng/ml and β hCG was 10,405 mIU/ML. On D3, total WBC count was 18,810/ mm3 and procalcitonin was 3.38 ng/ml. On D5, WBC count was 12460 / mm3 and procalcitonin was 0.96 ng/ml and β hCG was 7076 mIU/ML. On post-partum D6, whole placenta was expelled spontaneously (Fig 4), USG was also done which was suggestive of empty uterine cavity.

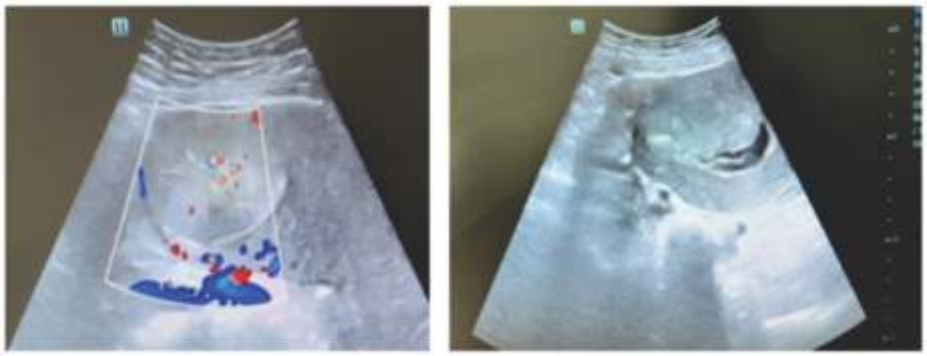

Fig 3: Deeply trapped placenta over right cornu with surrounding thin myometrium <2mm and also absence of retro-placental clear zone at anterior wall of myometrium.

Fig 4: Expelled placenta

CASE 3:

A 24-year-old primi para patient presented with lower abdominal pain in our OPD. Patient was delivered vaginally outside before one month. On clinical examination, uterus was sub-involuted and no active bleeding or foul-smelling discharge was present. USG was suggestive retained placenta of 9cm × 7cm with thin fundal myometrium and increased vascularity (Fig 5). As patient had no active vaginal bleeding or foul-smelling discharge from vagina, conservative management was opted and was monitored with serial USG, Beta hCG and CBC. Serial USG were suggestive of gradual decrease in size and decreased vascularity on Color Doppler and β hCG was decreased from 754 mUL/ML to 8 mUL/ML in one month. Oral antibiotics were also started. Vaginal swab detected no pathogenic organism.

On post-partum D 65 patient developed severe pain. USG was suggestive of placenta in lower uterine cavity and cervical canal, which was trapped in cervix. Inj Oxytocin was started. Patient was shifted to operation room and retained placenta was removed (Fig 6) with help of ovum forceps under anaesthesia. She had purulent blood stained discharge from uterine cavity, endometrial swab was taken. Post procedure USG showed empty uterine cavity with minimal fluid collection.

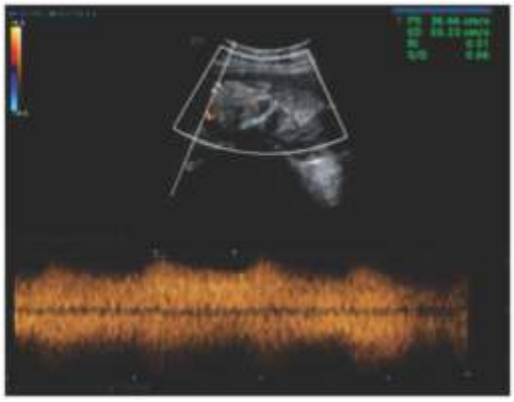

Fig 5: USG on D30 - Retained placenta of 9cm × 7cm with thin fundal myometrium and increased vascularity

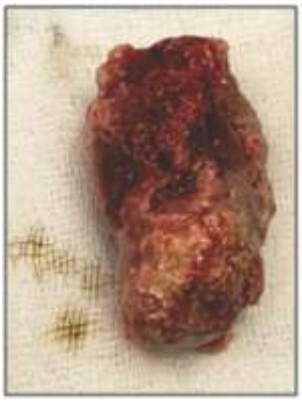

Fig 6: Placenta separated and expelled on D65

DISCUSSION:

Retained placenta can be a major cause of post-partum morbidity and mortality. Cases in which significant morbidity is associated such as haemorrhage or sepsis surgical intervention may be required.

In all 3 cases, conservative management was carried in which 2 cases were managed on outpatient basis as there was no active bleeding or any signs or symptoms of sepsis were present and admitted when they had signs of separation. All 3 cases were monitored with serial USG, CBC and β hCG and started on prophylactic antibiotics. Placenta was separated spontaneously in all three cases.

There is evidence that angular pregnancy is associated with Fetal Growth Restriction, pre-eclampsia, pre term birth,Retained placenta which reflects the possible impact of defective placentation on the fetal well being.

Inj Methotraxate (MTX)was not used in any of the cases. Currently, MTX is used as an off-label drug in conservative management of retained placenta as hypothetically, it induces placental necrosis and accelerates involution of placenta. Ellen H et al discourages the use of MTX as placenta which is left in situ is already a degenerating tissue.4 In the limited available literature, it is observed that the outcome ranges widely from expulsion of placenta after 7 days to resorption within 6 months.5 For further assessment such as development of PAS disorders or risk of retained placenta in future pregnancies patients need to be followed up.

We are highly obliged and thankful to Dr Sapana R Shah (Prof and HOU, Department of OBGY, SVPIMSR Ahmedabad, Author of the book “Placenta Accreta Spectrum Disorders: A Detailed Sight”) for her utmost support and guidance in managing all three cases and also giving insights for this article.

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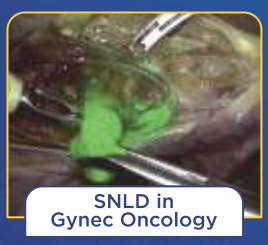
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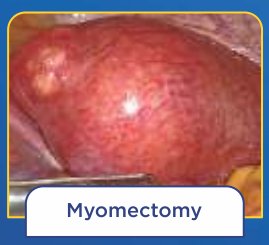
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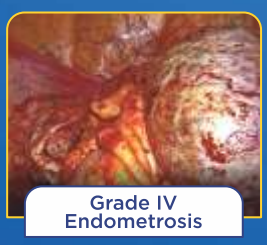
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