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EDITORIAL

As we all know September is World PCOS Awareness Month as it is one of the most common causes of infertility in women since Polycystic Ovarian Syndrome is a hormonal imbalance that interferes with a woman's reproductive system. When you have PCOS, your ovaries are larger than normal and can have many tiny cysts that contain immature eggs which create hindrances in conceiving.

"High levels of insulin have also been linked to PCOS. Insulin is a hormone that controls the change of sugar, starches and other food into energy for the body to use or store.

Many women with PCOS have too much insulin in their bodies because they have problems using it. Excess insulin appears to increase the production of androgen."

"Being overweight or obese is another major and contributory cause of PCOS. However, if you are overweight or obese, excess fat can make insulin resistance worse. This may then cause the level of insulin to rise even further. High levels of insulin can contribute to further weight gain. Since obesity, central obesity and insulin resistance are strongly implicated in its etiology, reduction of these risk factors should be a central treatment focus."

"The first-line treatment is to educate the patient about weight loss. Weight loss has been consistently successful in reducing insulin resistance and restoring ovulation and fertility. Current conservative treatment should emphasize sustainable weight loss through dietary modification and exercise. Modifying additional lifestyle factors, including alcohol consumption, psycho-social stress, and smoking, are also crucial in the long-term treatment of PCOS."

Having PCOS does not mean you can't get pregnant because even though it is one of the most common causes of infertility in women, PCOS is treatable. The good news is that fertility treatment is available for PCOS. There are some treatments that can help women with PCOS have healthy pregnancies. If you have PCOS and you want to get pregnant, you should work with a competent fertility specialist. The specialist will help make sure you get the right dose of medicines, help with any problems you have, and schedule regular checkups and ultrasounds to see how you're doing. In vitro fertilization or IVF is a great option for getting pregnant with PCOS and many women have had great success with IVF.



Dr K Jayakrishnan, Managing Director and Chief Consultant
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A CASE OF CAESAREAN SCAR DEFECT : LAPAROSCOPIC ISTHMOCOELE REPAIR



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36 year old lady (P1L1) came to our OPD in view of secondary infertility. She had history of one previous caesarean section 7 years back, in view of failed induction and has a male child of 7 yrs age. She consulted nearby hospital for secondary infertility, did 2-3 ovulation induction cycles. Repeat USG done there showed caesarean scar defect and was referred to our hospital for surgical correction before infertility management. She had no symptoms of abnormal uterine bleed, dysmenorrhea or chronic pelvic pain. Transvaginal USG was done which showed isthmal region dilated 1.6 cm close to myometrium 3 mm with echogenic areas? clots inside. Cavity distended 8.8 mm, with echogenic areas seen in cavity.? Clots in 3D Cavity

Basic investigation done for patient were normal. After cardiology and anaesthesia clearance, patient was taken up for laparoscopic isthmocoele repair. Hysteroscopy done using Bettochis hysteroscope with cervical dilatation, Isthmocele was noted in anterior myometrium above the level of internal os. Then proceeded laparoscopically, UV fold was dissected and bladder pushed down. Hysteroscopic guidance and transillumination was done which revealed the edges of the defect. The fibrotic edges of isthmocoele were cut and edges freshened, and then resutured in 2 layers using V - Loc sutures. Then UV fold sutured using 1-0 vicryl. Post operative period was uneventful and patient was discharged on postoperative day 3.



DISCUSSION :

Over recent decades, delivery by cesarean section (CS) has become increasingly common in both developed and developing countries. In addition to short-term risks of CS, there are long-term risks several years after the current delivery that can affect women's health and their future pregnancy outcomes.

Isthmocele is not a rare sequel of CS. An isthmocoele is defined as a triangular hypoechoic area at the site of the previous caesarean scar and represents an inadequate healing of the myometrium. It's reported incidence varies greatly, being comprised between 6.9 and 69%.



In the majority of cases, it is asymptomatic and is diagnosed incidentally on transvaginal ultrasound scan (TVUS). In some cases, it can cause abnormal uterine bleeding, dysmenorrhea, dyspareunia, chronic pelvic pain or secondary infertility. Its presence is associated with an increased risk of obstetrical complications including morbidly adherent placenta, caesarean scar ectopic pregnancy, or uterine rupture.



A shallow incision through the cervical tissue, inadequate suturing, or incomplete closure of the uterine wall due to a single-layer endometrial-saving closure technique or use of locking sutures are various suggested causes for the development of isthmocoele. Primarily, isthmocoele located in the higher part of the cervix correlated with previous cesarean section, whereas, in the patients with the defect in the lower part of the cervical canal, the cesarean section had been performed after cervical dilatation.



Discontinuation of the myometrium at the site of a previous CS is seen as a hypoechoic area described as a "niche". A "filling defect," anechoic and triangular shape, is observed on SIS under the bladder recess, in the region between the uterine body and the cervix. This is the typical site where low-transverse cesarean deliveries are performed.

The management of isthmocoele should be decided based on the patient's symptoms and plans for future childbearing. It can be surgically treated using laparoscopy, hysteroscopy, vaginal repair or by combining hysteroscopy and laparoscopy. In clinical practise, two factors are used in selecting the optimal route of repair, isthmocoele size and the patients desire for fertility. According to



Marotta et al, an isthmocele can be classified as a large defect if the residual myometrium is >3 mm and as a small defect if the residual myometrium is <3 mm. For small defects, hysteroscopic resection has been reported as a safe, fast, and efficient method in controlling symptoms for the patients who did not desire fertility. For larger defects, hysteroscopy has been associated with an increased risk of uterine perforation and bladder injuries. Thus, for defect >3 mm and for the patients desiring a future pregnancy, laparoscopy is considered the optimal approach.

In our case the patient was asymptomatic, the isthmocele being diagnosed as an incidental finding during an ultrasound examination during secondary infertility evaluation. The management of asymptomatic patient is still debatable. However, in patients desiring a future pregnancy the benefits of a surgical treatment should not be questionable. One of the most crucial technical aspect of an isthmocele is the correct identification of the defect. Several techniques have been used, including illuminating the defect with the hysteroscope, which we used in our case for identifying the edges of isthmocele.

Hysteroscopic simultaneous assistance during laparoscopic isthmocele repair can be of great help in identifying the edges of the defect, especially in large cavities in which edges might not be clear otherwise. Resecting all of the fibrotic tissue while respecting healthy myometrium is essential. Excessive cauterization and ischemic suturing could prevent proper healing of the myometrium.

CONCLUSION:

Laparoscopic isthmocele excision and repair seem to be an appropriate approach for the treatment of isthmocele-related symptoms when done by skilled laparoscopic surgeons. Surgical management in asymptomatic patient is still debatable. However, in patients desiring a future pregnancy the benefits of a surgical treatment should not be questionable. The management of isthmocele should be decided based on the patient's symptoms and plans for future childbearing. The benefit of this new surgical approach seems to persist even after a subsequent CS. Further investigations and prospective studies are required to define the long-term gynecological and obstetrical outcomes of this procedure.

MORAL DILEMMA CONCERNING PROPHYLACTIC GONAECTOMY IN AN UNMARRIED FEMALE - A PROFESSIONAL PERSPECTIVE



Dr. KOKILA B.T. MD (OBG)
Fellow in Reproductive Medicine

INTRODUCTION

Turnersyndrome (TS) is one of the most common chromosomal aneuploidy and is present in 1:2000 to 1:2500 live births. [1] Approximately 40-60% of patients with TS have 45XO (45, X/46, XY) karyotype, while some can have severe mosaicism like 45, X/46, XY mosaicism, which is rare with estimated detection rates of 1.7 per 10,000 new-borns. [2] In Turner syndrome, individuals may present with a wide spectrum of manifestations ranging from phenotypic females without virilization, ambiguous genitalia or Turner features to phenotypic males and are typically short or of normal height. [3] This may go undetected into adulthood unless there are gross features of Turner syndrome, growth retardation, pubertal delay or sexual ambiguity. Adult males may present during investigation for infertility. Traditionally, gonadectomy has been recommended in individuals with Y chromosomal material [4], as there is a considerable risk of gonadal malignancy. Bilateral gonadectomy is recommended as soon as the diagnosis is established. [5]

CASE REPORT

A 22-year-old, unmarried female, presented with secondary amenorrhoea for 7 years. She got her menarche spontaneously at the age of 15 yrs., accompanied by two menstrual cycles, followed by amenorrhoea. On investigating the same, she was diagnosed with left ovarian dysgerminoma (Germ cell tumour). She underwent Staging Laparotomy, left adnexal tumour excision with omentectomy and peritoneal biopsy, on March 2016, from RCC, Thiruvananthapuram. Following which she took four cycles of chemotherapy with cisplatin, bleomycin, and etoposide. Still, she remained amenorrhoeic and was started on progynova and meprate cycles (Hormonal therapy) for 5 years, to no avail.

Physical and mental milestones and developmental history throughout the infancy and childhood were normal. She is 141 cm tall with a mid-parental height of 152 cm. Her weight is 52.5 kg with a body mass index of 26.4. She has no acne, hirsutism or deepening of voice. There was no dysmorphic Turner features. External genitalia appeared female. Breast development was Tanner 3 and pubic hair was Tanner 2. There was no clitoromegaly or palpable gonads. According to her parents, her developmental milestones were normal and academic performance was also average. She did not have any issues with her social interactions. Systemic check-up including abdominal and cardiovascular examination was normal. Her karyotyping with GTG banding revealed to be



14/30-XO, 16/30-Iso X - Isochromosome mosaic Turner Syndrome (IMST), with two cell lines, one with one X chromosome found in 14 cells, and another with one X chromosome and an isochromosome for the long arm of one chromosome found in 16 cells (Mosaic Turners syndrome). Her FISH with CEP X/Y probe showed mos.46, X, I (Xq) [58%] /45, X [42%]. Her hormonal workup showed, FSH - 110.57, LH-32.3, TSH -2.47 and Estradiol - 2.37. Ultrasonography revealed a hypoplastic uterus (5.7x 2.0 cm) with thin endometrium and small right ovary (1.2 x 1.3 cm) with no follicles.

The case was morally challenging in terms of future prospective for the patient and risk associated with the remaining ovary. After discussing the options with the patient and considering endocrinologist's and surgery oncologist's opinion regarding the same, in view of dysgenetic ovary, she was planned for Prophylactic salpingo oophorectomy. Complete pre anaesthetic investigation including cardiologist opinion and check-up was done.

Intra operative findings revealed to be small uterus, with absent left adnexa. Right ovary appeared streak, right tube was normal with flimsy adhesion between the omentum and right adnexa. Bluish nodules seen over the mesenteric and omentum, all over the peritoneal cavity.

Omental adhesiolysis was done. Right salpingo oophorectomy done using Ligasure. Nodules were taken for biopsy from the omentum. Overall, time taken for the surgery was 90 min. There were no major intraoperative or post operative complications. Patient was discharged on 2nd post operative day on oral antibiotics and analgesics. Histopathological report was within normal limits with suture granuloma and no tumour deposits.



1. Right sided streak ovary



2. Right salpingo oophorectomy done



3. Specimen retrieved through umbilical port and sent for HPE

DISCUSSION

In our patient typical dysmorphic features of TS were not found. Except for short stature, all other findings are inconstant, which is the possible explanation for undetected mosaicism. Hormonal evaluation also revealed elevated gonadotropins with low estrogen levels.

Dysgenetic gonad dysfunction as the risk factor for origin of germ cell tumours, its precursor lesion is gonadoblastoma, which could progress toward invasive germ cell tumours, particularly dysgerminoma or less frequently embryonic carcinoma, teratoma, yolk sac tumour, and choriocarcinoma. [6]. GBY (gonadoblastoma locus on the Y chromosome) is assumed to be related to the origin of tumours located in the pericentromeric region of Yp. SRY is located proximally while GBY is located distally. Gonadal dysgenesis is due to Y-chromosome without the testis determining region (SRY). SRY is functional in spermatogenesis and is the structural gene for H-Y antigen which is an oncogene, which is associated with development of GCTs.

Recommendation for the search for Y-chromosome fragments in TS in two circumstances: signs of virilization and/or when there is a marker chromosome not identified by classical cytogenetics. To rule out the same for our patient, FISH with CEP X/Y probe was done, which showed mos.46, X, i(Xq) [58%]/45, X [42%]

CONCLUSION

We present a rare disorder of 14/30-XO, 16/30-Iso X - Isochromosome mosaic Turner Syndrome (IMST) with heterogeneous manifestations. Work-up revealed a diagnosis of mosaic Turner syndrome. The mean age of gonadal tumour diagnosis as per previous reports is 18 years. Our patient was 15 years old when diagnosed with GCT. The presence of Y cell line, mosaicism is indicated in patients with virilisation in TS, even if Y cell is not identified in the peripheral blood. But our patient had no virilizing features. In few studies, the gonadal tumour has been reported without evidence of Y-chromosome, its basis was dysgenetic gonad itself giving rise to GCT. So, laparoscopic and ovarian biopsy might be considered in any patient suspected of dysgenetic or dysplastic ovaries. However, as most of the studies have found higher incidence of GCT among dysgenetic gonads, up to 30-35%, [7] we thought it was suitable to recommend prophylactic gonadectomy to our patient.

Complete Uterine Septum - Case Report

Dr. Srilatha Dhulipudi MBBS, DGO
Fellow in Repro Medicine



28y/0 Mrs. X was referred to our hospital with history of primary infertility for 1.5 yrs. She was evaluated and was found to have a incomplete uterine septum on MRI(AFS Class Vb), P/S - Showed single cervix

USG was done which showed Uterus with 2 widely separated cavities

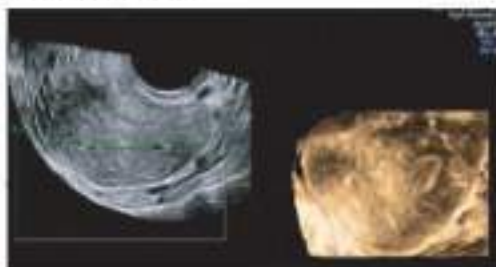
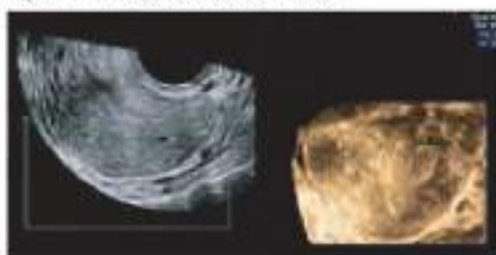


Figure 1 Widely separated 2 cavities



Proceeded with Operative laparoscopy and Operative Hysteroscopy. Intra-operatively complete Uterine septum seen upto the level of internal os, Uterine septum cut with 26Fr Resectoscope using Colin's knife, bipolar cutting current, with Saline as distension medium. Endometriotic deposits seen over the both uterosacrals and pelvic sidewalls were fulgurated using bipolar cautery. Chromotubation done and bilateral free tubal spill seen.



Figure 2
Complete Uterine septum seen upto the level of internal os



Figure 3
Septum resection done with 26Fr resectoscope



Figure 4
Bilateral ostia visualised after the septal resection

Post-operatively the patient was given HRT for 3

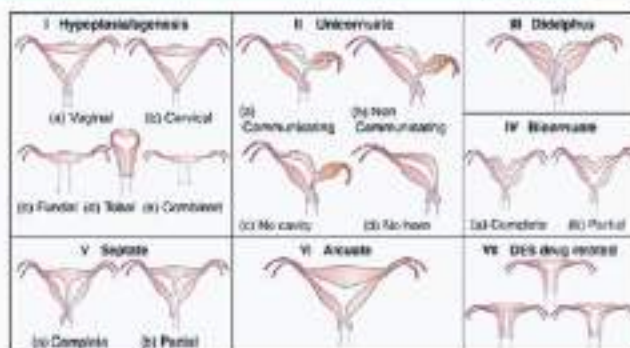
Discussion : The uterus originates from the paramesonephric, or Müllerian ducts. In a septate uterus, the Müllerian ducts have not fused during the period of embryologic development. A septate uterus is defined as a uterus with a division of the uterine cavity (septum) without any restrictions to the length of the septum, according to the new ESHRE (European Society of Human Reproduction and Embryology)/ESGE (European Society for Gynaecological Endoscopy) classification system for female genital tract congenital anomalies. The external contour of the uterus should not have an indentation. Septate uterus is the most common uterine anomaly, accounting for 35% of all identified uterine anomalies.

Traditionally, the uterine septum was resected by a laparotomic hysterotomy, but since the introduction of hysteroscopic septum resection in 1970, the latter approach is considered first-line therapy. Possible complications of a hysteroscopic septum resection are bleeding, perforation of the uterus, postoperative intrauterine adhesions, and uterine rupture in subsequent pregnancies. Even so, hysteroscopic septum resection is still common practice in many countries

The pathophysiology behind poor reproductive outcomes in women with a septate uterus is unknown. Earlier studies asserted that the septum is avascular and mainly consists of fibrous tissue. The main cause of impaired fertility in women with a septate uterus was considered to be a disturbed implantation. More recent studies suggest that the septum consists of normal endometrium and myometrium and resembles the uterine wall. It is unclear whether restoring normal anatomy also restores normal function, and thereby improves fertility outcomes in women who wish to conceive.

ESHRE/ESGE classification Female genital tract anomalies	
Uterine anomaly	Cervical/vaginal anomaly
Abbreviation	Abbreviation
U0 Normal uterus	C0 Normal cervix
U1 Dysmorphic uterus	C1 Single cervix
U2 Septate uterus	C2 Double 'lower' cervix
U3 Bicornuate uterus	C3 Unilateral cervical anomaly
U4 Horn uterus	C4 Cervical agenesia
U5 Agenesis	C5 Normal vagina
U6 Unclassified malformation	C6 Local uterine anomaly to the vaginal opening
	C7 Congenital obstructive vaginal anomaly
	C8 Prolapsed vaginal opening
	C9 Vaginal agenesis
	C9b Vaginal malformation

Figure 5 ESHRE classification of Müllerian anomalies



RECURRENT HYDROPS IN PREGNANCY

Mrs.S 32 year old, G4P1L1A2 at 11 weeks 2 days pregnancy was diagnosed with hydrops fetalis during NT scan. Her first pregnancy was a spontaneous conception within a year of marriage. It was a missed miscarriage at 10weeks and medically managed. After 4 months she conceived spontaneously and delivered a full term male baby by normal vaginal delivery. After 4 years, by the age of 30, she had her 3rd pregnancy. During the NT scan there was diffuse body and scalp edema in the fetus suggestive of hydrops fetalis. The pregnancy was terminated by medical method. At that time, Karyotyping of the abortus was not done. After 2 years, she again conceived spontaneously. 1st trimester scan showed raised NT (3.7mm) with absent nasal bone and diffuse body edema and scalp edema. There are no genetic disorders in her family. She had a non consanguineous marriage. Her blood group is A positive and she has not had any blood transfusion in the past. In view of fetal hydrops, she was given the option of termination of this pregnancy and the fetus was sent for genetic testing. Chromosomal microarray analysis (CMA) of the abortus showed duplication of chromosome 21 suggestive of Down syndrome. Now parental karyotyping has been suggested.

Non immune fetal hydrops (NIFH) refers to cases not caused by red cell isoimmunization. There are various causes of NIFH like cardiovascular, genetic, hematological, urogenital, gastrointestinal, infections, metabolic, monochorionic twin pregnancy, idiopathic where genetic causes constitutes almost 13.4% of NIFH. The aetiology of NIFH varies significantly depending on the gestational age at the time of diagnosis. The most common causes of NIFH in early gestation are chromosomal aneuploidy or a complex developmental anomaly. Aneuploidies account for 7% to 16% of NIFH cases. The most common aneuploidy associated with hydrops fetalis is Monosomy X (42% - 67%) and others are Trisomy 21-Down syndrome (23%-30%), trisomy 13, 18 and 12 (10%) and triploidies and tetraploidies.

As the number of potential genetic causes of NIFH is significant, genetic evaluation is crucial. Also, the recurrence risk of NIFH in each subsequent pregnancy may be as high as 25%. So it is important to make an accurate diagnosis that also allows targeted molecular prenatal testing in following pregnancies. This can be done using classical karyotyping or CMA (to exclude chromosomal aberrations) and/or gene sequencing (to diagnose monogenic syndromes).

Structural aberrations occur due to a loss/gain or rearrangement of genetic material within a particular chromosome. These aberrations include deletions, duplications, inversions, ring formations and translocations. Depending on the size of the deleted or duplicated

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fragment, these aberrations may or may not be identified in standard karyotyping. Whereas, a chromosomal microarray analysis (CMA) can detect even small ones (<5Mb, sub microscopic chromosomal imbalances) called copy number variants. CMA does not detect balanced chromosome rearrangements that may have clinical significance, alterations in the ploidy level (such as triploidy or tetraploidy), may not detect a clinically significant mosaicism, so a concurrent chromosome analysis should still be performed.

If a woman has had a previous pregnancy with Down's syndrome and the additional chromosome 21 was non inherited there is still an increased risk of recurrence. The increase has been estimated at three points in pregnancy. In an unpublished study of more than 2,500 women who had first trimester invasive prenatal diagnosis because of a previous affected pregnancy, the Down's syndrome incidence was 0.75% higher than that expected from the maternal-age distribution. Similarly, a meta-analysis of four second trimester amniocentesis series totalling 4,953 pregnancies found an excess of 0.54%. A meta-analysis of 433 livebirths had 5 recurrences, an excess risk of 0.52%. The weighted average of these rates, allowing for fetal losses is 0.77% in the first trimester, 0.54% in the second and 0.42% at term. Examination of the data suggests that the excess is similar at different ages so the excess can be added to the age-specific risk expressed as a probability. The recurrence risk is relatively large for young women but by the age of about 40 it is not materially different from the risk in women without a family history.

There are primary and secondary prevention strategies for couples who are at high risk of Down syndrome. Women with a high a priori Down syndrome risk because of an inherited translocation or a previous pregnancy with a non-inherited form of Down Syndrome, should have access to preimplantation genetic diagnosis (PGD). PGD is also an option for couples undergoing in vitro fertilization who are at an increased risk of passing along certain genetic conditions. The embryo is tested for genetic abnormalities before it is implanted in the womb. The effectiveness of this technique is limited by the availability of normal embryos in such families but reasonably high pregnancy rates are achievable with an extremely low risk of a Down Syndrome birth.

Secondary prevention of Down Syndrome can be done through antenatal screening followed by invasive prenatal diagnosis and termination of affected pregnancies. The screening test include the first trimester combined screening test and the integrated screening test. PAPP-A, HCG, nuchal translucency are measured in first trimester and alpha fetoprotein, estriol, HCG and inhibin A are measured in second trimester. If the screening test results are positive, diagnostic tests like chorionic villus sampling, amniocentesis are done to diagnose Down syndrome.

CASE REPORT

SELECTIVE FETAL REDUCTION IN MULTIPLE PREGNANCY

Dr SHARDA SHARMA MBBS, DNB
Fellow in Reproductive Medicine



31-year-old, Primi, 16 weeks 1 day, FET conception, diagnosed with DCDA twins in first scan, came for routine NT scan diagnosed with Twin B having increased NT (4.7 mm) and absent nasal bone. Patient and relatives were explained in detail about chromosomal abnormalities and possibilities of down syndrome in affected baby. Repeat scan was advised at 14 weeks and need for amniocentesis explained to rule out if baby is affected. Opinions were given -

1. About continuation of both (acknowledging that the one baby is Downs).
2. Selective termination of affected baby.
3. Termination of the pregnancy.

Scan was done at 14 weeks by the same Fetal medicine specialist and findings were confirmed.

Suggestion was given -

1. Amniocentesis at 16 weeks for both fetuses in continuation of both pregnancies.
2. If patient opts for fetal reduction of fetus B, then planning amniocentesis of fetus A and fetal reduction of fetus B in one sitting at 16 weeks.

Patient and her relatives opted for selective fetal reduction of twin B and amniocentesis of fetal A.



Discussion

Selective Fetal Reduction has been developed as a solution to try and minimise these risks for both mother and baby.

Selective fetal reduction can be carried out on a fetus diagnosed with a severe abnormality in utero, whilst the remaining fetuses are healthy. This is particularly relevant in the face of a diagnosis of a severe congenital abnormality-the affected fetus could be terminated to improve the viability of the unaffected fetus. This scenario is often referred to specifically as selective fetal reduction or selective termination.

Fetal anomaly is a key deciding factor in Selective fetal reduction. The majority of selective fetal reduction is legal and conducted under statutory ground E, which states, 'if the child

were born, there is a substantial risk that it would suffer from serious physical or mental abnormalities.

Selective Fetal Reduction should only be done with in tertiary level where facility of fetal medicine specialist is available. The number of fetuses and their chorionicity should be documented before 14 weeks of gestation. Detailed documentation and great care of each fetus to enable labelling their position, size, chorionicity, placental location and any anatomic feature that may help in their correct identification such as major anatomic abnormalities and markers of aneuploidy. The same operator should perform both procedure and the time interval between diagnosis and selective termination should be minimum.

The most common method of fetal reduction is trans abdominal because it is usually easiest. For this procedure, the fetal medicine specialist uses real-time ultrasound as a guide and inserts a 20 G or 22 G spinal needle through the mother's abdomen and into the amniotic cavity and then fetal heart, through which desired amount of 15% of potassium chloride solution is administered, which stops the fetal heart.

Because the fetus is very small during the first trimester, the reduced fetus is usually absorbed by the mother's body and as gestational age advances it results in fetus papyraceus or compressus. Some patient may manifest vaginal bleeding.

Procedure

Counselling of the Patient and bystanders done regarding the procedure (Fetal reduction of B and Amniocentesis of fetus A). Again explained about risk and outcome. Informed consent was taken. Antibiotic and progesterone support was given in pre-op and post op period.

Under all aseptic precaution, Fetal reduction is done in short general anaesthesia for fetus B (fetus with increased nuchal translucency and absent nasal bone) using intra-cardiac potassium chloride 15% - 4 ml solution.

Amniocentesis of fetus A done - 20 ml of amniotic fluid withdrawn and sent for microarray testing. Post procedure FHR of fetus A normal and absent in reduced fetus. Amniocentesis of fetus A has come as normal and patient is continuing pregnancy at 19 weeks gestation now.

Complications

Fetal- The risks to the remaining fetuses include, the possibility of miscarriage (abortion) of the pregnancy as a whole (2.1% - 5.8%), the possibility of rupture of the membranes, death of the fetus or additional fetuses, premature labour that may end in the birth of a premature infant with all the associated complications, such as motor, mental and nervous defects and prolonged hospitalization.

STATISTICS

May - Aug. 2022

TOTAL CASE	410
LAPAROSCOPY	134
HYSTEROSCOPY	108
MINOR CASE	56
MALE CASES	18
OBSTETRIC CASE	113
OPEN CASES	1
LAPAROSCOPY	114
TLH+BSO	15
LAVH	1
MYOMECTOMY	19
ENDOMETRIOTIC CYSTECTOMY	10
ADENOMYOMECTOMY	5
SALPINGECTOMY	3
SALPINGOSTOMY	4
DERMOID CYST EXCISION	1
LAP STERILISATION	1
TLH+SALPINGECTOMY	20
PCO PUNCTURING	5
FULGURATION OF ENDOMETRIOTIC DEPOSIT	16
TUBAL RECANALISATION	2
LAP ENORCLAGE	4
LAP PRP INSTILLATION	3
LAP SACROCOLOPEXY	2
ISTHMOCELE REPAIR	1

HYSTEROSCOPY	108
PRE IVF	53
POLYPECTOMY	11
SEPTAL RESECTION	4
ENDOMETRIAL BIOPSY	6
SMF RESECTION	31
TUBAL RECANALISATION	3
MINOR CASE	56
FRACTIONAL CURETTAGE	5
SUCTION EVACUATION	14
CERVICAL ENCRCLAGE	8
ENDOMETRIAL SAMPLING	2
MIRENA INSERTION	2
CVS SAMPLING	2
ERA	6
SSG	1
VAGINAL BOTOX	3
FETAL REDUCTION	2
AMNIOCENTESIS	4
PPS	4
EUA	1
TRU CUT BIOPSY LIPOMA	1
MARSUPIALISATION	1
MALE SURGERY	18

NAB	1
TESA	10
PESA	6
TESE	1
OPEN CASE	
TAH+LIPOMA EXCISION +	1
OBSTETRIC CASE	
LSCS	76
ELECTIVE LSCS	39
EMERGENCY LSCS	37
VACULUM DELIVERY	9
FTND	28
Conception + IUI statistics	
Total conceptions	147
Total IUI conception	39
IUI conception rate	9.98%
Spontaneous	54
COH only	11
IVF/ICSI Statistics	
Total No of cases	190
Total Conception Rate	39.09%
Frozen ET cycles	97
Conception rate after Frozen ET	32.98
Fresh cycles	87
Conception rate after fresh cycle	50%

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Dr JAYAPRAKASH D. PhD

Mr ABUL SALAM

Mr SANALKUMAR

PATHOLOGY

Dr JAYASREE P.V. MD

UROLOGIST

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SURGEON

Dr SUBHASH MS

VISITING CONSULTANTS (ALLIED SPECIALITIES)

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Dr KALA S.N. MBBS, DNB
(Prof. of Medicine GMC, Triv)

PULMONOLOGY

Dr ANN MARY JACOB MBBS, MD, DNB (Pulm)

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