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Editor's Choice

Rupture of Uterus – the Changing Trends

Rupture of uterus is a life threatening obstetric complication. It carries a high rate of maternal and perinatal morbidity and mortality. Overall incidence of rupture of uterus has been currently quoted ranging between 1 in 1146 (0.07%) and 1 in 200 (0.5%) pregnancies depending upon the quality of maternal care during the antenatal and the intrapartum period.

In low and middle income countries uterine rupture following prolonged and obstructed labor, multiparity are common. Author had the experience of managing such cases years back, while working as a registrar in one rural medical college in this state of West Bengal. One such study covering the period 1977-1986 revealed, 13.7% of all maternal deaths were due to rupture uterus.[1] Another such subsequent study, revealed a significant reduction the incidence of rupture uterus to 2.2%.[2]

In India, intervention with Janani Sishu Suraksha Karyakram (JSSK), for safe motherhood under the umbrella of National Health Mission (NHM), has brought a significant change. The presence of a skilled birth attendant at every labour, early referral, Nischay Jan (free transport), institutional delivery and partographic monitoring of labor are the important measures to lower the incidence of rupture uterus drastically. Impact of this combined approach in comprehensive routine and emergency obstetric care, is reflected by the significant reduction in maternal deaths in India.

Spontaneous uterine rupture is extremely rare. The estimated incidence is 1 in 8000 to 1 in 15000,[3] Lower segment cesarean scar rupture is observed in ≤1% of cases. Uterine rupture following prior uterine surgery (myomectomy), uterine malformation are relatively uncommon. Rupture of a gravid uterine horn of a bicornuate uterus with multiple pregnancies is extremely uncommon. Such type of uterine rupture has not been observed in the literature. Author's experience of managing such a case has been reported.[4]

Spontaneous rupture is usually complete and it involves the upper segment. Spontaneous disruption of the uterus in the midline during pregnancy has been reported. It is thought to be due to the weakness of the area of lateral fusion of the Mullerian ducts. Rupture of a gravid unicornuate uterus, has been reported, it is uncommon though. Iatrogenic rupture is rare again, in these days of obstetric practice.

Pregnancy in the cesarean scar and its rupture in the first trimester of pregnancy is a new entrant to the obstetric complications. Cesarean scar pregnancy comprises upto 6% of such cases in women with prior history of cesarean delivery. It was once thought to be the rarest complication. With rising incidence of cesarean delivery, cesarean scar pregnancy is quoted to be about 0.15% in women with a history of prior cesarean delivery. Different studies have quoted the incidence with a range from 1 in 1800 pregnancies to 1 in 2226 of all pregnancies.[5]

The mechanism of cesarean scar pregnancy is thought to be due to invasion of the trophoblasts through the microtubular tract of the scar tissue of prior cesarean section. The decidua over the scar tissue is either partially developed or may be absent at places. The trophoblasts invade the scar as well as the myometrial tissues, if it is present.

Presentation of a patient with cesarean scar pregnancy vary widely depending upon the degree of trophoblast invasion to the myomectum or the scar. There may be painless bleeding in the first trimester of pregnancy mimicking a miscarriage problem. In a worse situation, invasion may go up to the bladder. Pregnancy may grow towards the uterine cavity. In that situation, patient may remain asymptomatic and continue the pregnancy till term and deliver a live born baby.

Severe pain with or without profuse bleeding is due to scar rupture of the uterus. Cesarean scar pregnancy is often referred to as cesarean scar ectopic pregnancy. Interestingly, implantation occurs inside the uterus and above the internal os and it is not an ectopic pregnancy. Diagnosis of cesarean scar pregnancy is made by the way of exclusion of intrauterine, endocervical and a tubal pregnancy. Diagnosis is made mostly by using transvaginal sonography (TVS). Sensitivity of TVS to the diagnosis is of 86%.[5]

The myometrium between the gestation sac and the urinary bladder may be very thin or even absent. Invasion of the trophoblasts outwards often lead to rupture of the uterus. The bleeding may be life threatening. Color Doppler USG study is useful to diagnose the invasion of the bladder. MRI is an adjunct to sonography to confirm the diagnosis of bladder invasion. MRI can show the thinning out of myometrium between the gestational sac and the bladder.

Complications of cesarean scar pregnancy are many and at times may be life threatening. When gestational sac has no fetal pole, it may end in missed abortion or pregnancy failure. When the pregnancy continues, risk of placenta previa and its complications are to be kept in mind. Morbid adherent placenta is a known complication. Invasion and stretching of the scar, ends in rupture of the uterus with massive hemorrhage.

Majority of the patients that present with pain and hemorrhage are treated with laparotomy or laparoscopy. Hysterectomy may be a life-saving surgery. It is done in cases with uterine rupture, or placenta accreta for control of hemorrhage.

Uterine preserving surgery could be done with termination of the pregnancy safely. Medical management includes ultrasound guided local injection of methotrexate (MTX) or KCL. Laparoscopic or hysteroscopic resection of the gestational sac has been reported.[6] Hysteroscopic surgical excision were associated with less complications. Uterine artery embolization has been done to reduce the risk of bleeding. Patients with medical or conservative surgical management need to be followed up with serum βhCG for complete regression of trophoblastic activities.

This issue of IJOPARB has two case reports on rupture of uterus. One in the first trimester with a scarred uterus (p.64) and the other with spontaneous rupture of the fundus, in the third trimester. (p.57) These are uncommon. Going through these two case reports, I believe all of us would be benefitted.

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Second Stage Cesarean Section

Anjali Mantri,[1] Ashok Kumar[2]

Cesarean deliveries done in second stage of labor account for 1/4th of all primary Cesarean sections.[1] The incidence of second stage Cesarean sections is more in developing countries, where babies are delivered at home by traditional birth attendants and where the mothers report to hospital late in labor, when the traditional birth attendants fail in their endeavours.

Cesarean sections done at full cervical dilatation with impacted fetal heads are technically difficult and they are associated with an increased incidence of maternal and fetal morbidities. Extraction of the impacted fetal head may be done by 'push method', i.e. pushing through the vagina or by "pull method", i.e. reverse breech technique. Various studies[2,3] have compared both these methods. However, both these methods are associated with an increased rate of maternal morbidity in the form of uterine extensions, postpartum hemorrhage, and fever.[4,5] Patwardhan technique is a unique technique which is used for delivering babies in second stage Cesarean sections.[6]

Cesarean delivery in second stage of labor:

Currently, obstetric trainers perform most of the second stage trials of instrumental delivery. A recent UK study found that decisions made by consultant obstetric staff are more important in determining whether a second stage Cesarean section is optimum method of delivery for women with delay in advanced labor.[8] According to RCOG audit figures, about 35% of Cesareans for singleton pregnancies are performed because of failure to progress in labor, of which a quarter occur at full cervical dilatation.[9]

In 55 % of these cases, no attempt was made to achieve a vaginal birth with either forceps or ventouse. In those births where instrumental delivery was attempted, the audit noted a "failed" rate of 35% for ventouse and 2% for forceps.[9] Breech and twin deliveries can also lead to second stage Cesareans. Planned delivery is safer than vaginal birth for a term breech fetus with respect to immediate neonatal outcomes.[10] The place of emergency Cesarean is less clear when a mother presents in labour and reaches full cervical dilation with an unexpected breech presentation. In the absence of an experienced and skillful obstetrician to perform assisted vaginal breech delivery, women are advised to undergo an emergency second stage Cesarean.

According to a study by Anusha et al out of 90 Cesarean sections 30 were performed in second stage and 60 in first stage.[11] : Indications of Cesarean section are depicted in [Table 1].

Table 1: Indications of Cesarean Section in Second Stage

SECOND STAGE
Arrest of descent-malposition 76.7%
Arrest of descent-CPD 20%
Failed vacuum 3.3%

Cesarean section in the second stage of labor took significantly longer and was associated with more frequent post-operative pyrexia. There were more neonatal admissions. Hypoxic ischemic

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encephalopathy was more frequent in infants following second stage Cesarean section as was sub aponeurotic hemorrhage.[12]

Despite much discussion of the increase in elective Cesarean rates over the past 20 years,[13] little attention has been paid to the rise in second stage Cesarean section rates. The maternal risks of second stage Cesarean include major hemorrhage, longer hospital stay, greater risk of bladder trauma, and extension of tears of the uterine angle leading to broad ligament hematoma.[14] Although second stage Cesarean section is sometimes appropriate, many could be prevented by the attendance of a more skilled obstetrician.

In a study done by Asicioglu et al, maternal and neonatal outcomes were compared between second stage and first stage Cesarean sections which showed results as depicted in Table 2:[15]

Table 2: Comparison of Outcomes in Second and First Stage Cesareans

	Second stage of labour	First stage of labour
Labour Characteristics		
Skin incision to delivery time (min)	7.45±2.46	4.18±1.74
Total operation time (min)	48.63±3.21	36.78±2.72
Indication for Cesarean		
Previous CS	15.1%	15.2%
Dystocia	65.1%	37.6%
Fetal distress	7%	25%
Malpresentation	7.7%	6.1%
Others	5%	16.1%
Maternal outcomes		
Hospital stay (days)	2.48±.82	2.09±0.35
Estimated blood loss (ml)	914.18±91	701.51±52
Blood loss (> 1 litre)	37.5%	5.4%
Blood transfusion	10%	2.9%
Intraoperative complications	25.5%	6%
Injury of Uterine vessels	4.3%	0.7%
Unintended extension	13%	3.4%
Bladder Injury	2.4%	0.3%
Cervical lacerations	1.6%	0.4%
Hysterectomy	0.4%	0.001%
Wound infection	5.9%	5.4%
Postpartum endometritis	6.3%	5.3%
Maternal death	0.03%	0
Neonatal outcomes		
Birth weight (g)	3272±331	3077±388
5 min Apgar <=3	3.2%	0.2%
Septicaemia	2.8%	0.4%
Fetal injury	2.3%	0.1%
NICU admission	5%	0.8%
Neonatal death	1.3%	0.003%

Methods of delivery of fetal head during second stage Cesarean section

The fetal head is delivered as routine cephalic extraction but with the difficulty encountered in reaching below the impacted head deep in pelvis and thinned out and stretched lower uterine segment. This may result in extension of incision laterally or downwards and may involve uterine vessels, the ureter or trauma to fetal head.[16]

The five most reported important techniques for safe delivery were:

- High uterine incision
- Assistance to push fetal head transvaginally
- Correct flexion of fetal head
- Determine fetal position prior to starting
- Disimpaction of the fetal head in the caudal direction prior to elevation

'The Push technique'

Similar to the traditional cephalic delivery in a routine Cesarean section, but with the difference that the fetal head is extracted following pushing through the vagina assisted from below by another person and is hence referred to as the push method.[2]

The oldest procedure of abdomino-vaginal delivery in the presence of an impacted fetal head was described during the early 1980s as a modified Cesarean section,[17] the wedged fetal head was pushed by an assistant's hand introduced through the vagina. On the other hand, Lippert[18,19] suggested that the surgeon avoids further deflexion of head with one hand in the uterus and the other hand in the vagina pushes the head up. It was seen that the fetal head gets compressed between the surgeon's hand and the pelvic bones during manipulations combined with thinning of LUS because of prolonged labor results in lateral extension of a transverse lower uterine segment incision and involves laceration of uterine vessels.[20] A deeply impacted fetal head leads to a lack of space between the bony pelvis, pelvic muscles, and the fetal

head, making it difficult for the surgeon to insert a hand in order to dislodge it from the pelvis.

Another modification of the push technique was described by Landesman et al[17] The abdomino-vaginal delivery was carried out in modified lithotomy positions, the legs abducted in either the 'Whitmore' or the 'frog' position, and with a cupped hand in the vagina gently lifted through the uterine incision.

'Reverse Breech Extraction' or the 'Pull technique'

In the reverse breech extraction, the fetus in cephalic presentation is extended through the uterine incision by the podalic pole.[2,16] In this method after opening the uterus, the surgeon introduces a hand through the uterine incision towards the upper segment, grasps both feet and gently pulls the fetus up to extract it through the uterine incision.[21]

In simple terms, this maneuverer entails grasping the fetal feet, performing a semi version and delivering the fetus by total breech extraction.[22] Kafali[20] described a low vertical uterine incision for fetal extraction compared to low transverse incision as it provided more space for manipulation as well as its safety as it did not lead to lateral extensions of uterine incision. In most of the circumstances, the fetal feet can be easily reached through a transverse uterine incision. An inverted T or J shaped incision is not a prerequisite of this method,[23] which is usually used in situation of extreme difficulties in fetal extraction. An upward extension of segment incision at one of the lateral ends leads to the J shaped incision whereas an inverted T is an extension in the center of the lower segment flap of uterine incision.[24,25] While dis-impacting fetal head, fetal trauma can be prevented by avoiding hyperextension of fetal cervical spine and forceful pull on neck.

The Patwardhan technique (Shoulders first method)

This technique was first described by Dr. B.D. Patwardhan.[26] In case of occipito-transverse or occipito – anterior positions with the head deeply impacted in the pelvis, an incision is made in the LUS, at the level of anterior shoulder which is then delivered out. The posterior shoulder is also delivered with gentle traction on this shoulder. Now the fingers are locked through both the axillae and with gentle traction aided by fundal pressure by an assistant, the body of the fetus is brought out of the incision, the fetal head is then gently lifted out of the pelvis. When the fetus in occipito- posterior position after delivering the anterior shoulder as described earlier, the hand is intended into the uterus upto fundus and a foot is grasped. Gentle traction on this foot along with fundal pressure, the body of the fetus is extracted out followed by head.

Methods of delivery for various positions of baby in utero are depicted in [Box 1].

BOX 1: Method of delivery for various positions of baby in utero:

'Shoulders First' Patwardhan Maneuver: for back anterior position
- Both shoulders delivered first
- Trunk is delivered by flexion
- Both legs delivered
- Head lifted out last

Modified Maneuver: for occipito transverse positions
- Anterior shoulder delivered first
- Posterior shoulder delivered next
- Trunk is delivered by flexion
- Both legs delivered
- Head lifted out last

Modified Maneuver: for occipito posterior (back posterior) position:
- Both legs delivered first
- Trunk is delivered by flexion at thoraco-lumbar region
- Both shoulders delivered next
- Head lifted out last

Modified Maneuver: for occipito posterior (back posterior) position:
- One side shoulder and arm delivered first
- Same side leg delivered next
- Opposite side leg delivered next
- Trunk delivered by flexion
- Other shoulder and arm delivered next
- Head lifted out last

Comparison of push and pull method in delivery of head is depicted in [Table 3]:

Table 3: Comparison of Push Method and Pull Method for Delivery of Head

Push Method	Pull Method
Longer operation time	Less operation time
Incision extension [8 times more] Lateral > vaginal	Less extension
Infections are more [like UTI, endometritis, Febrile morbidity]	Infections are less
Wound infection more common	Wound infection less common
Postpartum fever [8.6%]	Postpartum fever [8.1%]
Need for blood transfusion higher	Need for blood transfusion lesser
Blood loss higher Apgar score was high	Blood loss lesser

The 'Fetal Disimpacting System' and 'Fetal Pillow'

The fetal disimpacting system is manufactured by Safe Obstetrics Systems UK. It has a foldable base plate that is 11 cm long and 4.5 cm wide, with a balloon attached to it which is inserted below the fetal head vaginally, at the time of inserting a foley catheter before the Cesarean. Just before making the uterine incision an assistant inflates the balloon with 180 ml of saline. This straightens the base plate which opens to become flat against the pelvic floor. The inflated balloon gently elevates the fetal head 3-4 cm from its original position, making it easier to deliver. This balloon is deflated once delivery is achieved, and the device is gently pulled out using the attached tubing or by locking a finger into the base plate.[27]

Conclusion

Delivery of baby in second stage via Cesarean section has more maternal and neonatal complications as compared to Cesarean done in first stage of labor. Timely decision-making regarding need for Cesarean, timely judgement and expertise in doing Cesarean reduces many intraoperative and postoperative maternal and neonatal complications. Patwardhan technique is the most opted method of delivery of deeply impacted head which increases the ease of delivery and has less complications but depends on expertise. Pull method has less complications than push method but is less preferred than Patwardhan technique.

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Original Article : Obstetrics

Effect of Oral L-Arginine on Low Amniotic Fluid Volume

Ojaswini Patel,[1] Pranati Pradhan,[2] Liza Bishi[3]

Abstract

Background: Oligohydramnous is defined as decrease in AFI. It complicate approximately 1 to 2% pregnancies. It is associated with increase operative interference and perinatal morbidity and mortility. L arginine is an essential aminoacid which increase nitric oxide which cause local vasodilatation. Hence it increases aminotic fluid in oligohydramnous and can improve pregnancy outcome.

Material & Methods: A prospective study conducted in the Department of Obstetrics and Gynecology V.S.S. Institute of Medical Sciences and Research, Burla involving 50 pregnant women with gestational age 28 weeks to 36 weeks diagnosed with AFI</=8 on ultrasonography, over a period of 2 years.

Conclusion: To avoid preterm labor, induction of labour and to prevent its serious consequences for the mother and newborn, therapeutic intervention is desirable to prolong the pregnancy. Keeping in mind the cost of rearing preterm baby in NICU, oral l-arginine can be used as a cheap and feasible method in resource-poor countries.

Keyward: Oligohydramnos, L arginine, Amniotic fluid index.

Background

Amniotic fluid provides a protected milieu for the growing foetus, by cushioning the foetus against mechanical and biological injury, supplying nutrients and facilitating growth of it. Decreased amniotic fluid is associated with placental insufficiency, impaired lung development in foetus and fetal growth restriction. Long-term complications of oligohydramnios are cord compression and variation in fetal heart rate during labour and increased chance of operative deliveries. Amniotic fluid volume (AFV) increases to about 800–1000 ml at 28 week gestation, plateaus near term and declines to about 400 ml at 42 weeks.[1] The amount of amniotic fluid (AF) is most commonly evaluated by ultrasound using amniotic fluid index (AFI) or single largest pocket (SLP). An AFI of 8 cm and above is considered normal, between 5 cm and 8 cm is borderline and <5 cm is oligohydramnios. When AFI is >25 lt is called polyhydramnios.[2]

In chronic placental insufficiency, the fetus tries to acclimatize by redirecting blood flow to vital organs such as in reduction of available intrauterine space for adequate fetal growth. Subjected to pressure from all sides, the fetus assumes a peculiar appearance like potter facies and musculoskeletal deformities such as club foot, talipes and wry neck may be seen. Lack of movement of amniotic fluid within the tracheobronchial tree results in pulmonary hypoplasia.

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Different medical interventional methods have been tried to treat oligohydramnios such as maternal hydration both oral and intravenous, trans abdominal amnioinfusion and intrapartumamnioinfusion. A recently propagated treatment of oligohydramnios is the administration of L-arginine which has been found to be effective in cases of intrauterine growth restriction and Pregnancy Induced Hypertension. However there are very few studies where oral l-arginine was found to increase AFI. This study was done to find out the effect of L-arginine on AFI, the mode of delivery and the foetal outcome.

L-Arginine (Arg) a nutritionally essential amino acid for the fetus,[3] is a precursor for synthesis of nitric oxide (NO) and polyamines in cells.[4] L-arginine is a semi essential amino acid acting as a substrate for synthesis of NO.[5] NO has a diverse role in obstetrics as it plays a vital role in labour, cervical ripening, preeclampsia and intrauterine growth restriction.[6] L-Arginine is also reported to improve growth hormone releasing hormone secretion, and as a consequence increase in plasmatic growth hormone influencing somatic growth.[7] It is also suggested that it may play a significant role in fetal growth by stimulating insulin secretion and as a precursor for both polyamine synthesis and NO production.[8]

Objective

To assess the effect of oral L-Arginine on amniotic fluid volume in pregnant women with low AFI (AFI</=8) after treatment. To assess the effectiveness of oral L-Arginine on fetal outcome in pregnant women with low AFI in terms of birth weight, APGAR scoring, Need for NICU admission & Perinatal mortality.

Material and Method

A prospective study was conducted in the department of obstetrics and gynaecology V.S.S Institute of Medical Sciences and Research, Burla involving pregnant women with gestational age 28 weeks to 36 weeks diagnosed with AFI</=8 on ultrasonography, over a period of 2 years after getting approval from the institutional research ethics committee. Fifty cases were studied from November 2016 to October 2018

Women attending outpatient department of obstetrics and gynaecology with AFI</=8 on ultrasonography enrolled, who satisfied eligibility criteria between 28-36weeks, Singleton pregnancy, Intact membrane. Patients diagnosed with major congenital anomaly, with PROM, already received treatment for low AFI, Multiple pregnancies,Pregnancy with chronic diseases like diabetes mellitus, renal disease,severe preeclampsia,severe IUGR at the time of presentation were excluded.

Women who fulfilled inclusion criteria who gave consent were prescribed with sachet of L-arginine containing 3 gm of active ingredient one sachet once a day mixed with 200 ml of water per orally after breakfast for varying period of 1 to 4 weeks. Follow up and repeat scan of patient were done and findings were analysed. Repeat scans evaluated the followings; Fetal weight,Amniotic fluid volume, Placental maturity. AFI value if </=5 scan repeated after 1 week, AFI is 5-7 scan repeated after 2 weeks & AFI 7-8 repeated after 3 weeks

By follow up ultrasound AFI were noted and treated accordingly. All women continue and no maternal complications due to therapeutic intervention were found. No patients were discontinued from the study because of any adverse effects. Appropriate management measures as per the standard protocol were taken in addition to the above mentioned intervention. All patients were monitored regularly with serial non-stress test (NST) and biophysical profile (BPP) till delivery. However, patients were considered for delivery if the liquor remained less or when fatal distress occurred.

Observation

Out of 50, 25 cases (50%) has AFI </=5, 23 cases (46%) has AFI 5-7and only 2 (4%) has AFI 7-8 at the time of presentation.

Table 1: Distribution of women according to duration of treatment with respect to gestational age at diagnosis.

Gestational age at Gestational age at diagnosis (weeks)	Duration of treatment in weeks		
	No of cases who received 1 week treatment	No. of cases who received 2 weeks treatment	No. of cases who received 3 weeks treatment
28-30	1	2	6
30.1-32	0	4	9
32.1-34	1	7	9
34.1-36	5	5	1
Mean duration of treatment 2.36			
SD 0.54			

Table 1 shows distribution of women according to duration of treatment with respect to gestational age. Maximum no. of cases belongs to either 30.1 to 32 GA or 32.1 to 34 GA receiving 3 weeks treatment. Mean duration of treatment is 2.36 week with standard deviation 0.5456.

Table 2: Average change in AFI after treatment.

Gestational age at diagnosis (weeks)	Average change in AFI after treatment for 1 week	Average change in AFI after treatment for 2 weeks	Average change in AFI after treatment for 3 weeks
28-30	5.5	3.75	2.78
30.1-32	0	2.92	3.23
32.1-34	-3	1.64	2.94
34.1-36	0.46	1.08	1.9
Avg. change in AFI	2.28		
SD	0.34		

Table 2 shows average change in AFI with respect to GA and duration of treatment. Mean change in AFI after treatment was 2.21 with SD of 0.34.

Table 3 Mean AFI change after treatment with respect to AFI at presentation.

AFI at presentation (cm)	Mean AFIbefore treatment (cm)	Mean AFI after treatment (cm)	Mean change in AF I(cm)
</=5	3.95	5.95	2
5-6.9	5.8	8.34	2.54
7-8	4.8	9.9	5.1

This table shows the mean AFI change after treatment in relation to AFI at presentation. In group of women with AFI </=5, mean AFI change was 2 cm, in group with AFI 5-6.9 mean AFI change was 2.54 cm and in group with AFI 7-8 mean AFI change was 5.1cm after treatment.

Before treatment mean AFI was 4.95 cm. After treatment the mean AFI was 7.23cm.Mean increase in AFI with Larginine treatment is 2.28 cm.From 50 cases 21 cases were vaginal delivery, 28 cases were delivered by LSCS and 1case was instrumental delivery. Among 50 cases at the time of delivery in 7 cases there was meconium stained liquor.

Table 4: Neonatal Outcome

Average birthweight(in grams)	2439.8
Small for gestational age(SGA)	6
APGAR Score at 5 min:-	
< 4	1
4-6	2
7-10	47
Still birth/ Neonatal death	1
NICU admission	12
Transient tachypnoea of newborn	7
Continuous positive airway pressur Ventilation	2
Invasive ventilation	1
Respiratory distress	2

Table 4 shows neonatal outcome after delivery.Average birthweight was 2439.8 +/-446gms. From 50 delivery one was still birth.Among 49 neonate 6 were SGA and 12 neonates were admitted to NICU, maximum NICU admission was due to transient tachypnoea of newborn.

Discussion

The mean AFI was 7.23 cm, and thus, an AFI increase of 2.28cm could be obtained which we presume was definitely helpful for the fetus.

The Amniotic fluid has multiple functions. Its main role is to permit fetal lung development by two-way movement of fluid into fetal bronchioles, and early severe oligohydraamnios is associated with pulmonary hypoplasia in the neonate. Amniotic fluid allows free movements of growing fetus and prevents limb contractures. It prevents adhesions between fetus and amnion and protects the fetus from mechanical injury. Reduction in amniotic fluid in labour is associated with variable amount of umbilical cord compression and fetal hypoxia.

With the easier availability of ultrasonography nowadays more cases of oligohydramnios are being identified. This helps us to be more cautious and anticipate problems especially during labor. However the need for an effective, economical, easily available treatment modality remains unmet. Maternal dehydration has been always believed to cause oligohydramnios though it cannot be coined as the cause in every case. Recently, serial ultrasound guided amnioinfusions have been tried but with varying success rates. Moreover it carries the inherent danger of fetal loss as it is an invasive procedure. L-arginine is a versatile amino acid with a wide range of biological functions. It serves as a precursor not only to proteins but also nitric oxide which has been identified as endothelium-derived relaxing factor.[9] L-arginine increases uteroplacental blood flow through nitric

oxide mediated dilatation of vessels thereby increasing the supply of nutrients to the fetus aiding its growth.

In a study by Ropacka et al, L-arginine was found to be effective in cases of Intrauterine growth restriction.[10] Similarly in another study in growth restricted and pre-eclamptic patients by Dera et al, use of L-arginine was associated with lower rate of operative deliveries and higher Apgar scores at both 1 and 5 minutes.[11]

Nabhan and Abdelmoula, in their Cochrane review which included five randomized controlled trials comprising 3226 women, found that with AFI of <5 cm, there were more induction rates, higher induction failures, more cesarean deliveries for fetal distress without much difference in NICU admission rates, and improved perinatal outcome.[12] They opined that lower cut offs such as 3rd percentile (equal to AFI of 4) or single deepest vertical pocket (less than 2 cm) used for definition of oligohydramnios may be more benificial.

In an ovine study with maternal fluid overloading, it was reported that a reduction of maternal fetal osmotic gradient facilitated water transfer to the fetus, leading to an increase in fetal urine production.[13]

Nitric oxide (NO) is an important regulator of placental perfusion, as it plays a role in placental vascular endothelial function. NO is synthesized from the physiologic precursor l-arginine by the stereospecific enzyme NO synthase in what is called the l-arginine/NO pathway, and l-arginine is the only substrate for the production of NO.[14] NO diffuses into the underlying vascular smooth muscle cells and mediates vasodilatation and platelet stabilization by a cyclic GMP-dependent process.[15]

It is not known whether an improvement of endogenous NO production could enhance fetal growth. NO-induced vasodilation in renal vessels may improve glomerular filtration rate (GFR) and thereby enhance fetal urine production.

Abida Ahmad studied the effect of intravenous infusion of 200 ml of aminoacids and 500 ml of 10% fructodex on alternate day basis in 20 clinically and sonographically proven cases of oligohydramnos.

Sreedharan et al. studied the effect of l-arginine in 100 women diagnosed to have oligoamnios between 28 and 36 weeks of gestation.[16] The expectant mothers were prescribed sachets of l-arginine containing 3 g of the active ingredient for periods varying between 1 and 4 weeks. There was significant improvement in AFI (by 2.03 ± 0.39 cm), and they opined that l-arginine can be used as a cheaper alternative to ultrasound-guided amnioinfusion in pregnancy complicated by low liquor remote from term.

Shripad Hebbar et al studied the effect of Maternal hydration and L-arginine supplementation in improving liquor volume in patients with decreased liquor and prolongs pregnancy.[17] Treatment with l-arginine and fructodex resulted in significant improvement in liquor mean increase in AFI was found to be 2.4 and mean increase in gestational age was 2.9 weeks in their study .

In our study the mean duration of treatment was 2.36 ± 0.54 weeks which was similar to study by Anita Soni et al in which duration of therapy in study was 2.4 ± 1.1 weeks[18] Therefore, studies recommend the supplementation of L-arginine and antioxidants in pregnancy to maintain the levels of NO so as to facilitate the required vasodilatation and have a beneficial role in the fatal growth.

In our study, the mean AFI at the time of recruitment was 4.95 cm and the mean gestational age was 32.28+/-0.54 weeks. Prior to delivery, these patients were delivered at 35.4 ± 1.5 weeks, and thus, pregnancy could be prolonged by 3.12 weeks.

Conclusion

Management of pregnancy less than 36 weeks of gestational age with oligohydramnios is a challenging situation. To avoid preterm labor, induction of labour and to prevent its serious consequences for the mother and newborn, therapeutic intervention is desirable to prolong the pregnancy, so that risks of prematurity are minimized and the obstetrician buys time to administer steroid prophylaxis. Keeping in mind the cost of rearing preterm baby in NICU, oral l-arginine can be used as a cheap and feasible method in resource-poor countries.

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Case Report - Obstetrics

A Rare Case of Spontaneous Prelabour Fundal Rupture of Unscarred Uterus in a Multigravida at 34 Weeks of Pregnancy

Nishat Parveen Begg,[1] Swarup Chowdhury,[2] Sima Biswas,[3] Subhankar Dasgupta,[4] Picklu Chaudhuri[5]

Introduction:

Uterine rupture can be complete when all the layers of uterine wall are separated, or incomplete when the uterine muscle is separated but the visceral peritoneum is intact.[1] There are several risk factors for uterine rupture with the most common being separation of a previous hysterotomy/classical cesarean scar. Other risks factors include trauma, injury, prior myomectomy or other uterine surgeries, or uterine anomalies.[2] Blunt trauma results in uterine rupture in < 1 % of severe cases (American College of Obstetritians and Gynaecologists, 2010). Rupture is more likely in a previously scarred uterus and is usually associated with a direct impact of substantial force.[3] Spontaneous nonobstructive rupture, which is rare and solely confined to high parous women, usually involves the upper segment and often involves the fundus. Whereas in obstructive type, the rupture involves the anterior lower segment transversely and often extends upwards along the lateral uterine wall.[4] We are hereby reporting a case of spontaneous prelabor uterine rupture at the fundus in a G7P2+4 patient at 34 weeks gestation.

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Case Report

Mrs S B, 28 year old, residing at Pakur, Jharkhand was admitted at 34 weeks of pregnancy with features of shock on 3/11/2019. She was G7 P 2+4 with vaginal deliveries, 3 years and 1 year back and 4 induced abortion followed by dilatation and curettage. She had no prior antenatal check ups and no investigations were done. On 3/11/2019, she went to the toilet on getting up from bed when she experienced sudden acute pain in the abdomen and dizziness. There was no history of trauma or bleeding per vagina. She was brought to the emergency in a semiconcious state. Her BP, on admission was 80/40 mmHg, pulse rate 130/min, and she was severely pale. Uterine contour was lost with superficial fetal parts with severe diffuse tenderness all over abdomen. Fetal heart sound could not be localised with stethoscope or Doppler. On vaginal examination, her os was closed and she was not in labour. After simultaneous resuscitation patient was prepared for a laparotomy. On laparotomy, about 1.5 lts blood mixed with amniotic fluid with a dead fetus was found floating in the abdominal cavity. There was a fundal rupture of the uterus (Figure 1 and Figure 2). Decision of obstetric hysterectomy was taken because the damage was irrepairable and

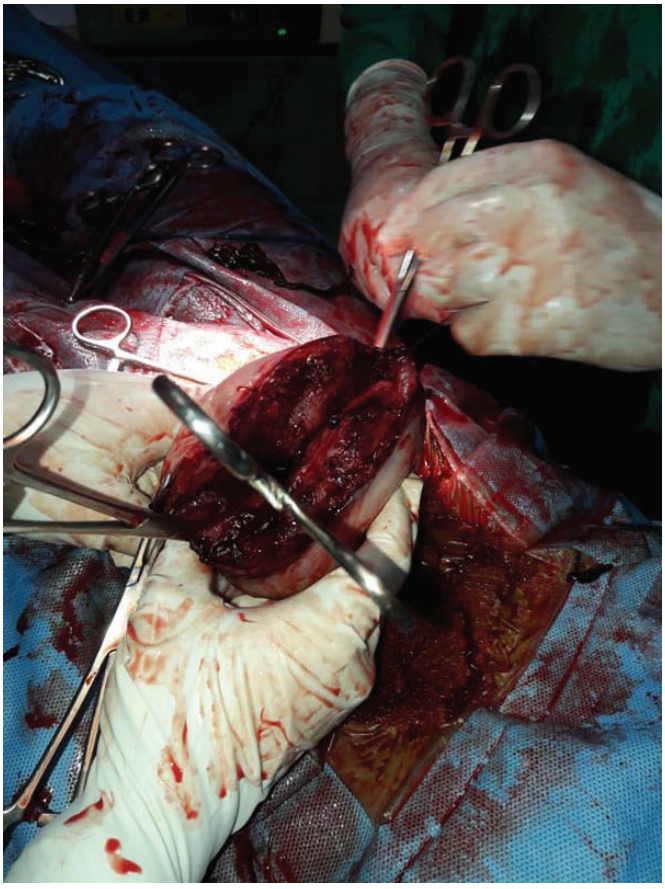

Figure 1

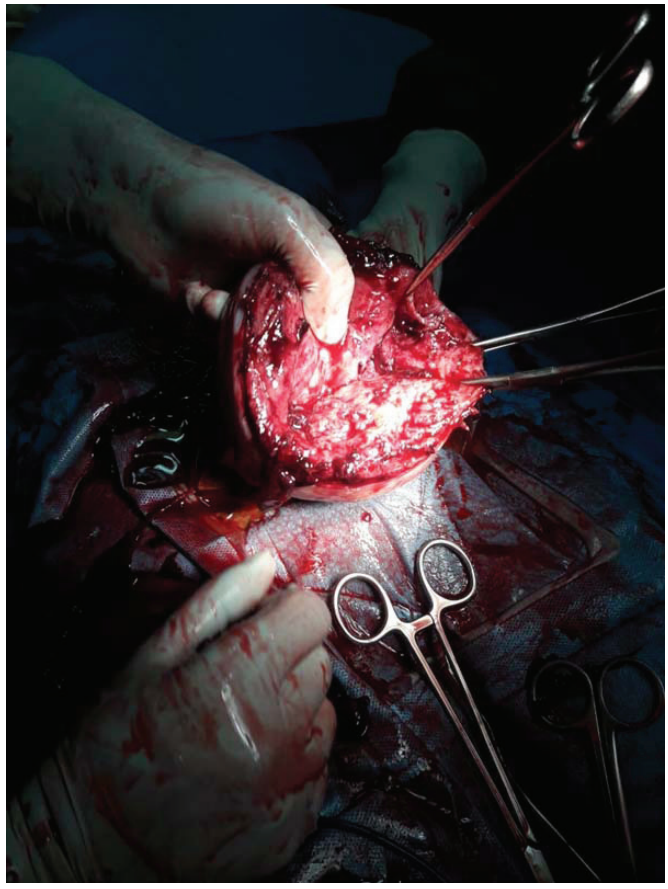

Figure 2

abdomen was closed after putting an intra-abdominal drain. The fresh stillborn weighed 1.9 kg. During intra operative and post operative period 3 units of blood were transfused. Patient was discharged in stable condition on the 10th postoperative day.

Discussion

The predisposing factors of uterine rupture are scarred uterus, multi-party, multiple pregnancies, big babies, the injudicious use of oxytocics, contracted pelvis, malpresentation, internal podalic version and instrumental deliveries. In our case we think following multiple epi-sodes of curettage, there must have been a weak area at the fundus which had healed on its own with a scar formation. The scar had given way at 34 weeks without any obvious trauma or uterine contraction leading to a fundal rupture of the uterus.

There has been reported case of silent uterine fundal ruptures in a 36 yr old, grand multipara (G7P6) presenting with vaginal bleeding and intrauterine fetal death and sonographic apperance of placenta covering internal os in a hemodynamically stable condition at 36 weeks. Fundal rupture was found on laparotomy.[5]

Fundal rupture uterus in women with previous curretage has also been reported in a 35 yr old G2 P 1+1, with one vaginal twin delivery 12 yrs back and one dilatation and curettage for MTP, presenting with IUFD at 34 weeks, posing diagnostic dilemma and diagnosis was delayed till she became hemodynamically unstable.[6]

Literature review even shows a few rare cases of primigravida without any obvious risk factor presented with spontneous unscarred prelabor rupture of uterus. Due to low index of suspicion in primigravid rupture of uterus, the diagnosis was often delayed. The intervention was mostly done when evidence of fetal or maternal compromise occurred and preservation of uterus was mandated.[7]

In our case considering the fact that she is multigravid and hemodynamically unstable, the intervention

was immediate and decision for hysterectomy was obvious.

Conclusion

Spontaneous antepartum fundal rupture of uterus before onset of labor are rare and symptoms and signs may widely vary posing diagnostic dillema. However, the diagosis should be always considered in muligravida with history of termination of pregnancy as timely intervention can be life saving.

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Etiological Factors of Irregular Periods in Young Women in a Semi Urban Tertiary Care Hospital of West Bengal, India

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Abstract

Objectives: To find out the causes of menstrual irregularities in young women attending gynecology clinic.

Materials and Methods: This cross sectional study included 150 women aged 15-25 years attending single consultant-led gynaecology clinic of this semi urban tertiary care hospital. We excluded women who attended emergency, those with pregnancy complications, with diagnosed anatomical abnormalities and with previously diagnosed haematological problems. All the patients underwent clinical examination, analysis of the specified hormonal status and ultrasonography of the whole abdomen.

Results: The data from our study showed that 57.3% of women with menstrual dysfunction have only polycystic ovary syndrome according to Rotterdam criteria, 22% had normal findings suggesting only anovulation without any known cause, 10.6% had polycystic ovary syndrome with hyperprolactinemia, 2% had only hyperprolactinemia, 2% had subclinical hypothyroidism and 0.66% had premature ovarian insufficiency and hypothyroidism.

Conclusions: Polycystic ovary syndrome alone or along with hyperprolactinemia and /or hypothyroidism remains the main cause for menstrual abnormality in young women. Basic hormone analysis and ultrasonography will promptly diagnose these conditions leading to early and appropriate management. Polycystic ovarian syndrome and its association with overweight also need significant attention.

Key words: menstrual irregularities, young women, polycystic ovary syndrome.

Introduction:

Menstrual disorders are common in young women attending gynecological clinic.[1] Acyclic vaginal bleeding in girls within two years of menarche is most commonly attributed to an immature hypothalamic-pituitary-ovarian axis. Persistent heavy periods, persistent irregular periods causing disruption of daily routines or persistence of menstrual abnormality two years post-menarche may be because of other abnormalities which needs evaluation.

A significant proportion of menstrual abnormality persisting two years post menarche may be because

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of polycystic ovary syndrome alone[1,2] or with other endocrine abnormalities.

Early detection will not only be helpful for short term management but also may prevent medium term problems and long term problems. It may alleviate the significant anxiety among the patient and their parents.[3]

Objectives:

To find out the causes of menstrual irregularities in young women attending gynaecology clinic in semi urban hospital settings.

Materials and Methods:

This was a cross sectional study of prospectively collected data. The study had included 150 women aged between 15-25 years attending single consultant-led gynaecology clinic of a semi urban tertiary care hospital. The study was conducted from January 2016 to December 2019, a period of 48 months.

Inclusion criteria:

1) Age – 15 years to 25 years.
2) Presenting symptoms – irregular periods, excessive hair growth or both.
3) Menarche – more than two years ago.
4) No history of previous pregnancy.

Exclusion criteria:

1) Women who attended emergency with heavy periods.
2) Women with pregnancy complications.
3) Women less than two years after menarche.
4) Women with diagnosed anatomical uterine abnormalities.
5) Women with previously diagnosed haematological problems.
6) Women already receiving progesterone or oestrogen – progesterone combinations.
7) Women on metformin or myo- inositols.
8) Women on anti obesity medications.
9) Women on aspirin or anticoagulants.
10) Women with any surgical interventions in genital tract.

All patients underwent clinical examination including general and systemic examinations. Local examination was not done. All women underwent complete hemogram, blood group and Rhesus status estimation, fasting blood sugar estimation. They also had estimation of day three follicle stimulating hormone, luteinizing hormone, thyroid function test, fasting prolactin estimation. All women had transabdominal ultrasonography of the whole abdomen by a consultant ultrasonologist with experience of more than two decades.

Results:

The data from our study showed that 57.3% of women with menstrual dysfunction have only polycystic ovary syndrome according to Rotterdam criteria. 10.6% had polycystic ovary syndrome with hyperprolactinemia, 2% had only hyperprolactinemia. Another 2% had subclinical hypothyroidism and 0.66% had premature ovarian insufficiency along with hypothyroidism. 22% had normal findings suggesting only anovulation without any detectable cause. (Table 1).

Table 1. Etiological factors of irregular periods in young women

Etiological factor	Number	Percentage
PCOS	86	57.3
Hypothyroidism	3	2
Hyperprolactinemia	3	2
Premature ovarian insufficiency and hypothyroidism	1	0.66
Normal	33	22
PCOS and hypothyroidism	4	2.6
PCOS and hyperprolactinemia	16	10.6
PCOS and hypothyroidism and hyperprolactinemia	3	2
Hypothyroidism and hyperprolactinemia	1	0.66
Premature ovarian insufficiency	0	0

Of 109 patients whom PCOs was diagnosed, 86 (78.8%) patient showed only PCOS, 4 (3.6%) patient detected to have PCOS and hypothyroidism, 16 (14.6%) patients have PCOS and hyperprolactinemia, and 3 (2.7%) patient had PCOS and hypothyroidism and hyperprolactinemia (Table 2).

Table 2. Subgroup analysis of etiological factors among women with PCOS

Causes	Number	percentage
PCOS	86	78.8
PCOS and hypothyroidism	4	3.6
PCOS and hyperprolactinemia	16	14.6
PCOS and hypothyroidism and hyperprolactinemia	3	2.7

Phenotype A (hyperandrogenism, ovulatory dysfunction, polycystic ovarian morphology) was found in 19 (17.43%) women, phenotype B (hyperandrogenism and ovulatory dysfunction) was found in 12 (11%) women, phenotype C was not found, phenotype D was found in 78 (71.5%) cases (Table 3).

Table 3. PCO phenotypes

Type		Number	Percentage
A	Hyperandrogenism, ovulatory dysfunction, polycystic ovarian morphology	19	17.43
B	Hyperandrogenism, ovulatory dysfunction	12	11
C	Hyperandrogenism, polycystic ovarian morphology	0	o
D	ovulatory dysfunction, polycystic ovarian morphology	78	71.5

BMI was calculated according to WHO criteria for Asians. In the women diagnosed with PCOS (109 women), One woman (0.91%) was underweight, BMI were normal in 34 (31.1%) women, overweight in 30 (27.5%) women, pre obese in 33 (30.2%) women, Obese type 1 in11(10.09%) women , no women was in obese type 2 or obese type 3. In the non PCOS group (41 women) BMI was normal in 18 women (43.9%), overweight in 16 women (39.02%), preobese in 7 women (17.07%) (Table 4).

Table 4. Weight of the PCOS and Non-PCOS women (WHO Asian criteria)

Class	PCOS (Number)	PCOS (%)	Non-PCOS (Number)	Non-PCOS (%)
Underweight	1	0.91	0	0
Normal	34	31.1	18	43.9
Overweight	30	27.5	16	39.02
Preobese	33	30.2	7	17.03
Obese Type1	11	10.09	0	0
Obese Type2	0	0	0	0
Obese Type3	0	0	0	0

Discussions:

Our study at West Bengal, India showed Polycystic ovary syndrome is the commonest cause of irregular periods in young women. Out of 150 women, PCOS was found as the main diagnosis in 109 cases (72.6%). Our study revealed a higher incidence of PCOS than the study by Chabbra et al[1] may be because of strict inclusion criteria or may be due to geographical variation or semi urban study population.

Our study also revealed that sometimes PCOS may be associated with hyperprolactinemia. Estimation of prolactin is very important as 14.6% of cases PCOS was associated with hyperprolactinemia. Our study is very similar to Filho RB et al 4 and Kyritgi EM et al.[5] interestingly this study mentioned a cut off level over which pituitary adenoma was diagnosed. However, our study did not reveal high level of prolactin in any of the cases.

One of the limitations of this observation is that, only in one fourth of cases routine PEG precipitation was done (not all laboratory perform PEG precipitation routinely). As we know that macroprolactin may be one of the important causes for elevated level of prolactin level. The prevalence of macroprolactinemia in hyperprolactinemic populations varies between 15% and 35% according to Kasum M et al.[6] So these findings should be interpreted with caution.

Subclinical hypothyroidism alone was detected in 2% of cases, PCOS and associated hypothyroidism was detected in 4.6% cases. Considering the high overall prevalence of subclinical hypothyroidism in general female population in India,[7,8] thyroid function test should be performed routinely in women presented with irregular periods. Although only estimation of thyroid stimulating hormone will be enough in most cases, we found estimation of thyroid function test may be more cost effective. However opinion varies in this issue.

Interestingly hypothyroidism and hyperprolactinemia was found in 0.66% cases.

Many authorities do not recommend routine estimation of early follicular phase FSH and LH.[9]

However routine estimation of Day three FSH and LH has several benefits. Firstly, a small number of premature ovarian insufficiencies can be detected early. Secondly disproportionately high LH gives out a small clue about the potential therapeutic area in midterm problems of PCOS line fertility problem.[10]

Our study revealed that in this population 78 out of total 109 cases of PCOS (71.5%) were in phenotype D. Phenotype A was found in 19 (17.43%) cases and phenotype B was found in 12 (11%) women. This was in contrast to the findings of other studies.[11,12,13]

Although this study was conducted in a semi urban hospital it was interesting to find a significant number of overweight and obese women both in PCOS group and non PCOS group. Our finding revealed that PCOS group was associated with BMI above normal in overall 67.79% of women. A recent study also revealed that 61% of women with PCOS were overweight.[14] These findings indicate that the problem of overweight in women with is no longer limited to urban areas.

Conclusions:

Polycystic ovary syndrome alone or along with hyperprolactinemia and /or hypothyroidism remains the main cause for menstrual abnormality in young women. Basic hormone analysis and ultrasonography will promptly diagnose these conditions leading to early and appropriate management. A significant percentage of women with PCOS and non PCOS women have above average weight which may be a cause for concern.

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Case Report : Gynecology

Spontaneous First Trimester Anterior Uterine Rupture in a Multiparous Woman with Scarred Uterus

Swayamsiddha Mohanty,[1] Radhakanta Panigrahi,[2] Ramanjamma Madagani,[3] Maya Padhi[4]

Abstract

Uterine rupture in the first trimester of pregnancy or even in the early second trimester is very rare. Most uterine rupture occur in the third trimester at the onset of the contractions and mainly in a previously scarred uterus. The incidence of uterine rupture is 1 in 4,800 deliveries in developed countries and the rupture of an unscarred uterus is a few as 1 in 10,000-15,000 birth.[2] The greatest risk factor for either form of rupture is a prior cesarean delivery or other myometrial surgical incision. Other risk factors include grand multiparity, trauma, malpresentation, obstructed labour, misuse of uterotonic drugs, particularly for sequential labour induction.[2,3] Considering the rarity of uterine rupture in the first trimester of pregnancy, we present a rare case of uterine rupture at 10 weeks of gestation in a 24-year-old woman with a history of one prior cesarean section.

Introduction

Uterine rupture (UR) is a life threatening peripartum complications that can occur during any trimester of pregnancy. It carries a high a rate of morbidity and mortality for both the mother and her foetus. Presence of uterine scar is the main risk factor for either lower segment scar as cs section[4] or upper segment scar as myomectomy,[5] cornual section or previous repair of UR.[6] First trimester UR is rare and the actual incidence of occurrence in the literature is unclear. The risk is increased in multi para with short pregnancy interval[7] misoprostol used in termination of pregnancy,[8] Mullerian anomalies and presence of uterine scar.[9]

Hemoperitoneum is defined as collection of blood in the peritoneal cavity due to non traumatic or non iatrogenic cause. In the first trimester most of cases of hemoperitoneum are associated with disturbed ectopic pregnancy.[10] So UR is rarely suspected based on the clinical presentation of patients in early pregnancy. Maternal consequences of UR are hemorrhage, hypovolemic shock, bladder injury, needs for hysterectomy and maternal death.[11]

Case:

On October 2019, a 24-year-old gravida 2 para1 pregnant woman at gestational age of 10 weeks (based on her last menstrual period) was presented to the

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labour room of our tertiary care hospital. The patient had acute onset severe lower abdominal pain with 2 episodes of vomiting. She had a history of caeserian section two years back. There was no history of other uterine surgery or procedure or any history of trauma. she had no antenatal visits and only proof of pregnancy was her positive home urinary pregnancy test.

On general examination the patient was pale, blood pressure was 90/60 mmHg, tachycardia (PR=118/min). Generalised tenderness and rebounding was noted all over her abdomen. Vaginal examination revealed a very tender enlarged uterus with mild vaginal bleeding and tenderness in the left fornix. Neither adnexa was palpable.

Urgent abdominal ultrasound revealed a bulky empty uterus with marked free intraperitoneal fluid collection. No gestational sac or fetal pole was seen. Hematological examination showed haemoglobin (Hb) level of 6.5 gm/dl. The patient was counselled concerning the possibility of ruptured ectopic pregnancy and informed consent for abdominal exploration was obtained.

Exploratory laparotomy through Pfannensteil incision was performed under general anaesthesia with one unit of blood. Marked free blood collection and blood clots were noted in the peritoneal cavity with around 2L blood being evacuated from peritoneal cavity. Evaluation of both tubes revealed no enlarged masses with intact serosa. Both ovaries were normal. However, on exteriornisation of uterus we discovered a scar rupture with active bleeding. The gestational sac with the fetus inside was present in the Douglas pouch covered with blood clots.

Repair was done by absorbable suture in two layers till hemostasis was achieved. The patient was given 2 units of packed RBC and 3 units of fresh frozen plasma. Intarperitoneal drain was left and the abdomen was closed after confirming hemostasis. The patient had smooth recovery from anaesthesia and her postoperative haemoglobin was 9 gm/dl and hospital stay was uneventful.

Discussion:

Uterine rupture accounts for 14% of all hemorrhage-related maternal mortality. Most often, uterine rupture occurs in the third trimester of pregnancy, during labor, or mainly in a previously scarred uterus because of the fibrosis of the myometrium at the scar. Its occurrence in early pregnancy is very rare even in the presence of predisposing risk factors.[12]

At term and near-term pregnant women, especially in the setting of a trial of labor after prior cesarean delivery, careful and close monitoring of both mother and fetus can help in its timely diagnosis. Abnormal labor progress, abnormal abdominal pain, vaginal bleeding, loss of station of presenting part, maternal tachycardia, and fetal bradycardia are indicative factors for detecting uterine rupture.[1] However, in early pregnancy, especially without the presence of any predisposing risk factors, the diagnosis may occur with latency or may never be detected; leading to life-threatening complications. Furthermore, signs and symptoms of uterine rupture in the early trimester are non-specific.[13,14]

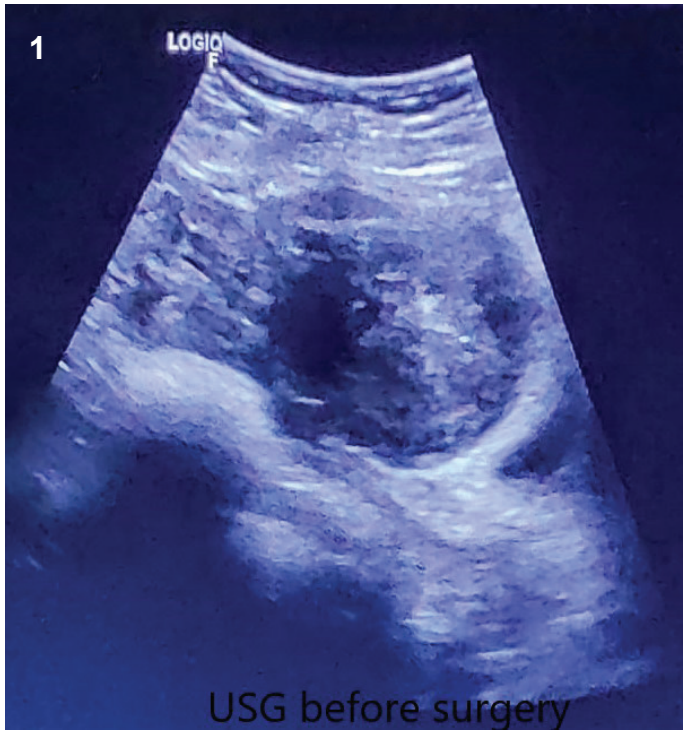
Although the presence of previous uterine scar has been described as the main cause of uterine rupture,[2,12] some recent studies have reported abnormal placentation (accrete, increta, and percreta) as the most common underlying etiology even in early pregnancy.[14,15]

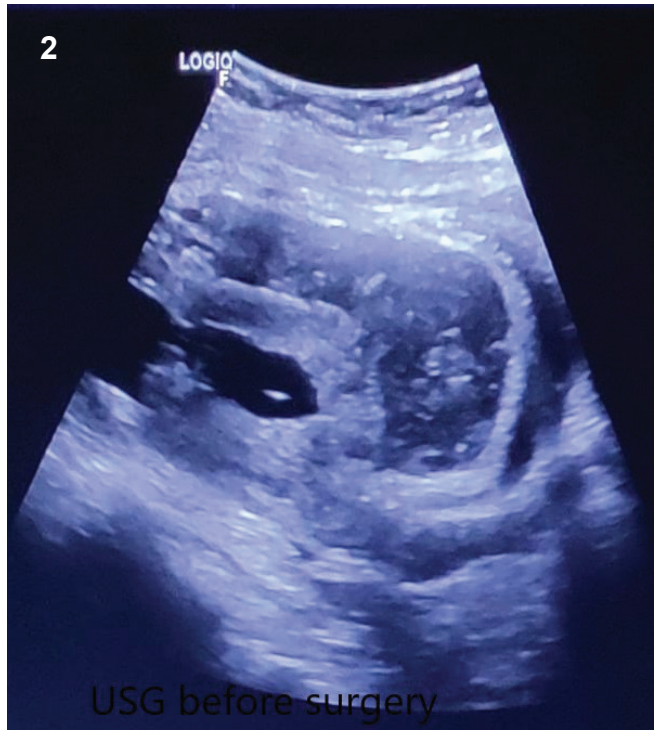
A review of 15 cases of uterine rupture in the first trimester of pregnancy showed that the most common causes were placenta percreta.[16] Considering a worldwide increase in the cesarean delivery rate, such an outcome was anticipated.

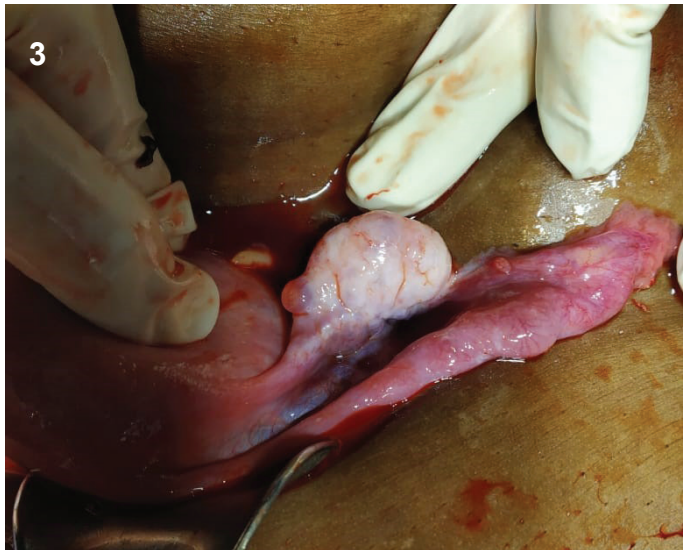
In most reported cases of uterine rupture in early pregnancy, patients were presented with acute abdominal pain and shock. This might be due to untimely diagnosis or, in most cases, the involvement of the fundus. It seems that despite the difficulty of detecting uterine rupture in early pregnancy, most ruptures can be repaired. The majority of the reviewed studies indicated two main causes of uterine rupture, namely abnormal placenta invasion and previous cesarean scar. This, combined with an increasing rate of cesarean sections worldwide, highlights the need for a high degree of clinical suspicion in early diagnosis of uterine rupture.

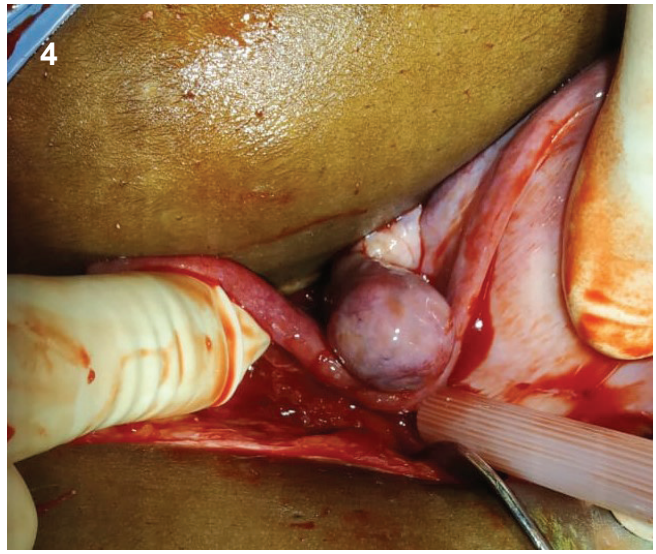
Conclusion:

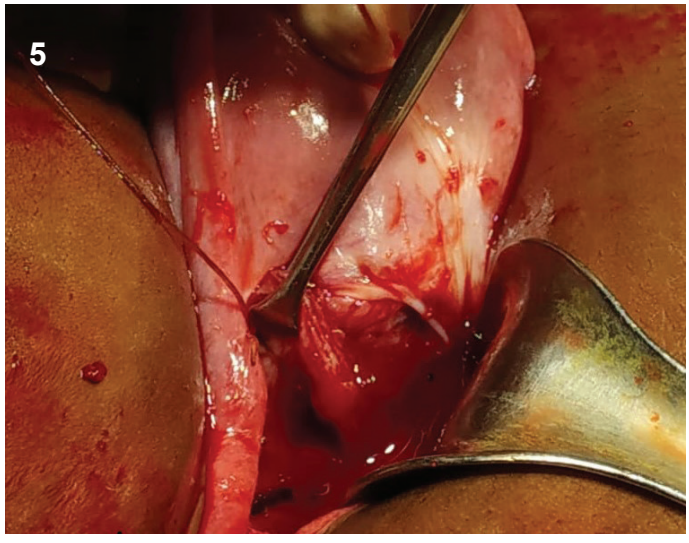
Awareness of probable diagnosis of uterine rupture in pregnant women with abdominal pain is important for timely diagnosis and proper management; even in the early gestational age of pregnancies and in the absence of known risk factors.











1. USG
2. USG1
3. Lt ovary and tube
4. Rt ovary and tube
5. Previous CS scar rupture

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OBITUARY

Dr. Ajit Chamanlal Mehta

(06.03.1930 — 06.06.2020)

Ajit C. Mehta was born to Dr. Chamanlal Mehta and Kusum Mehta, in Mumbai on 06.03.1930.

He was an alumni of the Seth G. S. Medical College and KEM Hospital, Mumbai and did his post-graduate training at KEM Hospital, Nowrosjee Wadia Maternity Hospital (NWMH), and Tata Memorial Hospital. He later joined NWMH as a Consultant. Later on he become the Dean of this prestigious institution. He considered NWMH as "The Temple of Learning" throughout his term of 39 years (1961 – 1999).

Indian Society of Perinatology and Reproductive Biology (ISOPARB) was established by Prof. (Dr.) Tarun Banerjee of Calcutta. Dr Ajit Mehta joined ISOPARB when the organization was young. He took keen interest in the organization of ISOPARB. He soon became the President of the organization. He initiated in organizing many ISOPARB Conferences. Once he was so deeply involved with ISOPARB that he was ready to donate some space at his own hospital for this organization to have its office.

Dr. Ajit Mehta was actively involved with National Neonatology Forum (NNF). For years there were joint annual meetings of ISOPARB – NNF.

Dr. Ajit Mehta was much respected in his field. His special interests were preventive Obstetrics, Gynecology and Perinatology. He was also the President of the Mumbai Obstetrics and Gynecological Society and was actively involved with FOGSI where he reached the position of Secretary General.

He was a teacher par excellence and won the hearts of all the residents who worked under him.

As a Dean of Nowrosjee Wadia Maternity Hospital he introduced many reforms in the interest of the Residents and patients. Dr. Mehta had a keen interest in research and after retiring as dean he was appointed as director of the research wing of NWMH. He initiated the computerized records of all patients delivering at NWMH. He designed a software for analyzing the data of the patients for benefits of the residents.

He was the recipient of several prestigious awards and orations by National and International organizations.

In personal life he was very simple, humble and a true gentleman. He was fond of reading and writing. He has published two books of his poems titled "Unfolding Life" and wrote a book on the story of NWMH as known and understood by him. He was an ardent "Bridge" player, and almost every weekend with his wife Rasilaben spent time at their farm in which nature abounds and peace prevails.

Dr. Ajit Mehta breathed his last at his residence on 06.06.20 after a brief illness.

He is survived by his wife Rasilaben, two daughters and a son.

May his soul rest in peace.

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