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Red Cell Alloimmunization and Perinatal Health

Perinatal health care is essentially targeted to reduce the still birth rate, pre-maturity, hypoxic brain injury, neonatal deaths and to prevent congenital malformations. Attempts have been made to reduce perinatal deaths over the years of Millenium Development Goals (MDG). Sustainable Development Goals (SDG) 3 and 5 stress on maternal health and perinatal health. In India current national figure of maternal mortality is 167 per 100,000 total births and those of perinatal mortality is 41 per 1000 total births. SDGs target to bring the rates for the both below 100 and 20 respectively.

Hemolytic disease of the fetus and newborns (HDFN) due to red cell alloimmunisation is an important area of perinatal morbidity and mortality. In Rhesus hemolytic disease, there is development of anti D antibodies following fetomaternal hemorrhage (FMH) in a Rhesus negative woman with a Rh D positive fetus. Haemolysis resulting in hypoxia and hyper bilirubinemia is the cause of death for the fetus and the newborn. We all now understand the pathological basis of the disease lies on the genetic and molecular background. Progress in the management is so much that we can prevent the disease successfully and in the rare occasion of failure of prevention during pregnancy, we can treat the fetus successfully even in utero. Currently perinatal mortality due to rhesus disease has decreased by 100 fold in the major parts of the globe. Well organized prophylaxis programmes have brought this major change. Failure of prophylactic anti D administration in the post partum period is about 1-2%.¹

A meta-analysis has shown that additional administration of 300 mcg of anti D at around 28 weeks of pregnancy to a woman in her first pregnancy can reduce the risk of immunization further from 2% to 0.2%.²

Immunization during pregnancy without any clinical signs of fetomaternal hemorrhage, is a known cause. Many pregnancy events may result in fetomaternal hemorrhage (FMH). Few common such conditions are: miscarriage problems, early pregnancy interventions (chorion villus sampling, aminocentesis), ectopic pregnancy, external cephalic version and medical termination of pregnancy. All these women need antenatal immunisation prophylaxis. Women receiving antenatal prophylaxis should also be given the dose of 300 mcg Rh IG within 72 hours of delivery additionally.

Anti D Ig should be given as soon as possible after the sensitizing event and should always be given within 72 hours. When the appropriate timing of anti D Ig is missed, it should be given within 10 days as it may provide some protection.

The assessment of the amount of antibody present in maternal serum depends upon individual laboratory cut off value following indirect antiglobulin (Coomb) test and the titre value. Significant anemia is not expected when the antibody titre is below 1:64.

Currently serial quantification using autoanalyzer is done. Anti-D levels <4 IU/ml excludes severe fetal hemolytic disease. However, it is important that the correlation between the antibody levels and the severity of disease in non D alloimmunization is poor.

It is observed that in India, most of the cases of Rhesus alloimmunization are due to failure of organized approach in the management of such cases during pregnancy and postpartum. The important areas of failure are: failure of recognition of Rhesus blood group, utilization of anti-D prophylaxis in the post partum period and or following the sensitizing events in pregnancy (mentioned above). There are other areas of failure also. The dose schedule of 300 mcg of anti D IG is based on the amount of FMH in majority of cases during delivery. It is accepted that 90% of women have FMH of less than 4 ml during delivery. However there are cases where FMH exceeds 4 ml and results in allommunisation unless additional dose of anti-D Ig is given. The cases where FMH exceeds 4 ml are: traumatic delivery, cesarean delivery, manual removal of placenta, twin pregnancy, and abdominal trauma during third trimester. Therefore, it is logical that we should perform Kleihauer–Betke screening test to quantify the FMH and to estimate the exact need of additional anti-D Ig.

There are several other antibodies to red cell antigens. These non Rh antibodies to red cell antigens are: Lewis, I and P. These antibodies are of the IgM class. These antibodies are not associated with HDFN. There are other antibodies known to cause significant fetal hemolysis. Presence of these antibodies may need treatment with intrauterine fetal transfusion (IUFT). These antibodies are: anti Rhc, anti-Kell (K1) and anti Duffy (antiFya). For these reasons, all pregnant women ideally should have red cell antibody screening in the first trimester and again as 30 weeks of gestation.

The significant progress in the management of red cell alloimmunisation is mainly due to the understanding of molecular biology. Use of ultrasonography (USG) for fetal assessment, USG Doppler study with middle cerebral artery (MCA) peak systolic velocity (PSV), is to determine the degree of fetal anemia.³ Cordocentesis is done for direct fetal assessment and therapy with intra uterine transfusion.

Ultrasound assessment is commonly used to assess the fetus with different parameters like placental thickness, hepatosplenomegaly due to compensatory increased erythropoiesis, and pleural or pericardial effusion.

Presence of sinusoidal heart rate pattern with cardiotocography (CTG), indicates severe fetal anemia.

Currently middle cerebral artery (MCA) peak systolic velocity (PSV) is considered the mainstay of fetal surveillance in a case with rhesus alloimmunisation. MCA PSV > 1.5 mom, predicts moderate to severe fetal anemia. Cordocentesis and intrauterine fetal transfusion have improved management outcome in terms of fetal and neonatal survival remarkably. Doppler study with MCA PSV has currently replaced aminocentesis and amniotic fluid spectrophotometry.

Specialized fetal medicine units are coming up in this country. Obstetricians, neonatologists, laboratory technicians and hematologists are working in close collaboration. There is progressive decline in fetal loss in this country due to rhesus alloimmunisation for the last few years. In this issue, the article, "*Perinatal outcome in Rh negative pregnancy*" presents the experience of the centre.

Rhesus disease cannot be eradicated. Obstetricians are to manage the pregnancy and need to decide the optimum timing for delivery. The risk of FMH and the antibody levels rise with progress of pregnancy. Antenatal corticosteroid therapy is given as appropriate to gestational age. Induction of labor or cesarean delivery may be done when antibody levels rise significantly.

Neonatal care has improved remarkably. Neonatologists need to manage for the correction of anemia and hyperbilirubinemia with exchange transfusion.⁴ In mild cases photo therapy may be sufficient. Neonates born following therapy with intrauterine transfusion may not need exchange transfusion. The results of IUFT are excellent. Neonates developing anemia following 2-6 weeks of birth may be treated with recombinant erythropoietin. This is found to reduce the need of top up transfusion.

Improvement in the understanding and management of Rhesus alloimmunisation, fetal loss rate is decreasing progressively. With limited exposure to patients with rhesus disease, the clinicians find it increasingly difficult to maintain the management expertise. Specialized fetal medicine centers are developed to maintain sufficient experience with full range of perinatal care for red cell alloimmunisation.

Antenatal prophylaxis is essential as a preventive measure. With current progress in the management issues, pregnancy outcome is excellent even in immunized women with high antibody levels.

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Preterm Perlabour Repture Membranes – Overview

Dr. Milind R. Shah

Introduction:

Preterm Perlabour Repture Membranes (PPROM) is more likely to occur in populations of lower socioeconomic status and complicates one-quarter to one-third of preterm births.¹ As such, PROM and PPRM complicate more than 400,000 and 120,000 pregnancies, respectively, in the United States each year.² Lack of education, poverty, living at high altitude, poor nutritional status of women in this area, and improper utilization of available health resources may be the causes of this high prevalence.³ Nutritional deficiencies that predisposes women to abnormal collagen structure have also been associated with an increased risk of preterm premature rupture of membranes.⁴

The membranes surrounding the amniotic cavity are composed of the amnion and the chorion, which are closely adherent layers consisting of several cell types, including epithelial cells, mesenchymal cells, and trophoblast cells, embedded in a collagenous matrix. They retain amniotic fluid, secrete substances both into the amniotic fluid and toward the uterus, and guard the fetus against infection ascending the reproductive tract.

At term, 8 to 10% of pregnant women present with PROM, these women are at increased risk for intrauterine infection when the interval between the membrane rupture and delivery is prolonged.

Preterm premature rupture of the membranes occurs in approximately 1-3% of all pregnancies and is

associated with 30 to 40 percent of preterm deliveries.⁵ Accurate diagnosis is very important in managing a case of PPRM. In management, decision making of whether to continue the pregnancy or to terminate the pregnancy is very crucial and depends on many factors. Clinician should take decision after thorough evaluation of risks and benefits of both these options.

Preterm premature rupture of membranes (PPROM) occurs in 3% of pregnancies and is responsible for approximately one third of all preterm births. Preterm PROM is an important cause of perinatal morbidity and mortality, particularly because many times there is some time lapse between rupture of membranes and delivery, risk of perinatal infection chance of it increases as delivery gets delayed after PPRM, and risk of umbilical cord compression due to oligohydramnious. Even though with conservative management, 50 - 60% of women with preterm PROM remote from term will deliver within one week of membrane rupture. Amnionitis (13 - 60%) and clinical abruptio placentae (4 - 12%) are commonly associated with preterm PROM which add to maternal and fetal morbidity. The earlier the PPRM there is more potential for pregnancy prolongation. In 2.8 - 13% of patients have spontaneous cessation of fluid leakage.

In babies which survive respiratory distress syndrome (RDS) is the most common serious complication after preterm PROM at any gestation. In babies who survive of RDS there is risk of other serious acute morbidities due to necrotizing enterocolitis, intraventricular hemorrhage, and sepsis. These complications are common with early preterm birth but relatively uncommon near term.

Dr Milind R Shah, MD, DGO, DFP, FICOG, FIAOG
President ISOPARB (2016-18)

It has been observed that perinatal sepsis is two-fold more common after preterm birth after PPRM than preterm labor with intact membranes.

Definitions

We need to understand following definitions related to PPRM.

PROM: Premature rupture of the membranes is defined as spontaneous membrane rupture that occurs before the onset of labor.

PPROM: When spontaneous membrane rupture occurs before 37 weeks' gestation but after 26 weeks, it is referred to as preterm PROM.

Latency refers to the time from membrane rupture to delivery.

Conservative management is defined as treatment directed at continuing the pregnancy.

Midtrimester PROM: Preterm PROM that occurs at or before 26 weeks' gestation.

As period of viability is decreasing over years and as we know nowadays babies more than 23 weeks can survive with NICU support, following definitions are more appropriate

Previable PROM: Which occurs before the limit of viability that is less than 23 weeks Preterm PROM remote from term: Which occurs from viability that is 23 weeks to about 32 weeks' gestation

Preterm PROM near term: Which occurs approximately 32 - 36 weeks' gestation

The importance of this definition is management and outcome differs in each group like in previable PROM occurs, immediate delivery will lead to neonatal death. Conservative management may lead to previable or periviable birth, but may also lead to extended latency and delivery of a potentially viable infant.

Immediate delivery after preterm PROM remote from term is associated with a high risk of significant perinatal morbidity and mortality that decreases with advancing gestational age at delivery.

As against, preterm PROM near term, immediate delivery of a noninfected and nonasphyxiated infant is associated with a high likelihood of survival and a low risk of severe morbidity.

Pathophysiology of PPRM

Over years obstetricians use to correlate PPRM with physical stress but now it is realized that Preterm rupture of membranes is multifactorial in nature. However if we narrate all causes which can be one or more in a case of PPRM. Simultaneous occurrence of more than one pathophysiologic processes could be responsible for PPRM. Basically it is suggested that membrane rupture is also related to biochemical processes, including disruption of collagen within the extracellular matrix of the amnion

and the chorion and programmed death of cells in the fetal membranes which could be stimulated by following causes.

1. Infection: Choriodecidual infection or inflammation appears to play an important role in etiology of preterm PROM, especially at early gestational ages.

2. Decreased membrane collagen content has been demonstrated in the setting of preterm PROM and with increasing gestational age. In support of this, increases in amniotic fluid matrix metalloproteases as well as decreases in tissue inhibitors of matrix metalloproteases have been identified among women with preterm PROM.

3. Other factors associated with preterm PROM include lower socioeconomic status, cigarette smoking, sexually transmitted infections, prior cervical conization, prior preterm delivery, prior preterm labor in the current pregnancy, uterine distention (eg, twins, hydratnnios), cervical cerclage, amniocentesis, and vaginal bleeding in pregnancy. Each of these may be associated with preterm PROM through membrane stretch or degradation, local inflammation, or a weakening of maternal resistance to ascending bacterial colonization.

Tobacco smoking, which independently increases the risk of preterm PROM, has been associated with decreased serum concentrations of ascorbic acid. Cadmium in tobacco has been found to increase the metal-binding protein metallothionein in trophoblasts, which may result in sequestration of copper.⁶

Smoking and history of previous PPRM were found to be risk factors for PPRM in black population.⁷

The incidence increases fourfold after third trimester PV bleeding.⁸

Though it is said that coitus, cervical examination, parity or exercise are not associated with PPRM, there is evidence that incidence increases fourfold after coitus in third trimester.⁹

4. The ultimate cause of premature membrane rupture is unknown in many cases.

Membranes that rupture prematurely, however, appear to be focally defective rather than generally weakened. The area near the rupture site has been described as a “restricted zone of extreme altered morphology” that is characterized by marked swelling and disruption of the fibrillar collagen network within the compact, fibroblast, and spongy layers.

Prediction of Preterm PROM

As there is association of preterm labor and PPRM and as there is risk of infection associated with it, It is always preferred if we can prevent and predict it.

The study the investigators found medical complications, work in pregnancy, symptomatic contractions, bacterial vaginosis, and low body mass index to be associated with preterm birth due to preterm PROM in nulliparas.

The presence of a short cervix (less than 25mm by transvaginal ultrasound) was associated with preterm PROM in both nulliparas and multiparas.

A positive fetal fibronectin screen was also associated with preterm PROM in multiparas. Nulliparas with a positive cervicovaginal fetal fibronectin and a short cervix had a 16.7% risk of preterm birth due to preterm PROM, whereas multiparas with a prior history, a short cervix, and a positive fetal fibronectin had a 25% risk of preterm PROM. Unfortunately, despite our developing ability to identify women at increased risk testing is expensive and inconvenient to the patient for preterm PROM, such, and will identify only a small fraction of those ultimately delivering preterm. Because of this, our clinical efforts remain focused on treatment of preterm PROM once it has occurred, rather than its prevention.

Diagnosis:

It is more of a clinical diagnosis but many times there is no correlation between history and actual leaking seen on observation.

It is always advisable to do sterile speculum examination to see for clear fluid coming from cervix rather than per vaginal examination which increases risk of infection and more disruption of membranes. One should also take sample for culture and sensitivity at the same time. Ruptured membranes is confirmed by direct visualization of fluid draining from cervical os upon valsalva or spontaneously or there may be pooling of fluid in posterior fornix. In advanced cases fetal scalp or hair on sterile speculum exam may be visualised.

Secondary investigation would be ultrasonography to see for oligohydramnios.

The presence of Nitrazine positive or ferning positive in fluid from vaginal pool of fluid would confirm the diagnosis. One should understand the fallacies of these test as vaginal pH can be increased by blood or semen contamination, alkaline antiseptics or due to bacterial vaginosis.

Cervico-vaginal fetal fibronectin (fFN) is 100% accurate if membranes are ruptured. If negative, fFN rules out ruptured or leaking membranes. False positive tests can occur with heavy bleeding, labor, and recent intercourse.

The tests like ultrasound guided transabdominal amnioinfusion of indigo carmine and observation of dye by per speculum examination is too invasive and not practical, rather there are more chances of infection.

Management:

Survival and outcome in PPRM is correlated with three variables; gestational age at rupture, amount of residual fluid and duration of fluid loss. It is noted that mortality rate more than 90% when there is PPRM earlier in mid trimester and when there is significantly reduced amniotic fluid volume.

Standard approach in management is either termination of pregnancy or expectant management to wait for PPRM to stop. It should depend on gestational age e.g. near term pregnancy and PROM always demands for termination. But in PPRM, decision of delivery or expectant management should

depend on individual assessment of maternal, fetal and neonatal complications.

Here ultrasound plays vital role which will tell about gestational age, amniotic fluid volume and fetal position but also gives information about polyhydramnios, multiple pregnancies and fetal anomalies. If AFI is less than 5 after 26 weeks in cases of PPROM, there is increased risk of infection and more chances of caesarean section.¹⁰

In cases with clinical evidence of advanced labor, chorioamnionitis, fetal distress or abruption placenta it is better to expedite delivery irrespective of gestational age.

It is necessary to start broad spectrum course of antibiotics immediately after culture to avoid further morbidity.

In PPROM between 32-36 weeks, it is always advisable to terminate pregnancy as it not only reduces risk of infection but also increases chances of baby survival. Additionally there is risk of cord prolapse or occult cord compression. Cord prolapse risk is more common in non vertex presentation.

Other non infectious risks with PPROM are placental abruption which increases to 25% if there is bleeding after PPROM as against overall risk of 5%.

It is necessary to have extensive consultation regarding fetal risk such as possibilities of poor neurological outcome in survivors and maternal risks of bleeding, infection and even death.

There is risk of pulmonary hypoplasia in PPROM which decreases as gestational age advances. For example, it is 50% if fetus is 20 weeks, 25% if it is 22 weeks and less than 10% if pregnancy goes beyond 26 weeks.¹¹

There are several other therapies tried in cases of PPROM with variable success. The success has to be yet proved with randomized trials. These therapies include serial amnioinfusions, intracervical tissue sealants and amniopatches.

For patients managed conservatively, daily fetal assessments, daily assessment of fetal activity as appreciated by the patient, assessment of uterine tenderness, temperature, persistent leaking of

amniotic fluid associated with purulence or bleeding is required.

Antibiotic administration is must in all cases of PPROM irrespective of evidence of infection. The NICHD regimen was ampicillin and erythromycin for 48 hours, followed by the same agents orally for 5 days if delivery did not occur. Two large meta-analyses done from studies on PPROM in the last ten years have both shown a benefit in using adjunctive antibiotics with expectant management. A large multicenter study with antibiotics but no steroids or tocolytics also showed a benefit. Another prospective randomized double blinded study looked at patients with PPROM and treated with antibiotics and steroids for all patients showed similar results.

Prophylactic tocolytics after PPROM have not been shown to prolong latency. Similarly, therapeutic tocolysis has not been shown to prolong the latency period. The effect of tocolytics to permit antibiotic administration and corticosteroid administration has not yet been investigated.

We have understood of role of Magnesium Sulphate as neuroprotector. Magnesium sulphate is given IV from 28 weeks to 33 weeks 6 days (4 gram IV bolus over 15 minutes followed by 1 gram per hour for 24 hours or until birth, whichever is sooner.

Another controversy associated with PPROM is whether we should give steroids in these cases for lung maturity. Lewis demonstrated a significant reduction in RDS almost 18 percent with steroid administration verses 44 percent when steroids were not given. The NICHD Research Study demonstrated a benefit regardless of membrane status. The NICHD Panel recommends steroids in patients from 24 to 32 weeks gestation in the absence of infection. It would seem better to adopt a rescue approach rather than a routine administration regimen.

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Study on Perinatal Outcome in Rhesus Negative Pregnancy

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Abstract

Hemolytic disease of the newborn secondary to Rhesus-D isoimmunisation contributes significantly to perinatal morbidity and mortality. There is a need for adequate counselling of pregnant women regarding the importance of detection of blood grouping and Rh typing during the antenatal period in order to prevent hemolytic disease of the newborn.

Aims and Objective: The aim of this study is to evaluate perinatal outcome of Women with Rh -ve blood group during the period of September 2012 to August 2014.

Material and Methods: All Rh-negative pregnant women who presented in labour room and neonates in NICU of Patna Medical college and hospital, Patna were included in study.

Results: Majority of neonate delivered vaginally 105 out of 130 (80.76%) and only 20 out of 130 (15%) delivered by cesarean section either emergency or elective. Out of 130 cases, 3 (2.3%) were as fresh stillbirths and 4 (3%) were macerated stillbirths. 109 women (83.84%) had negative indirect Coombs test and 21 women (16.15%) had positive indirect Coombs. 17 babies had Apgar less than 8 in which 12 was born from ICT positive mother. Out of 23 preterm babies, 19 had isoimmunised. Jaundice was present in almost all babies of ICT positive mother. Out of 34 babies admitted in NICU, 21 had isoimmunisation due ICT positive in mother. Phototherapy was needed in 26 babies in 21 were from ICT positive mother. Exchange transfusion was needed in only babies having ICT positive mother. Out of 5 neonates admitted to NICU, 4 had early neonatal death.

Conclusion: In Rh-negative pregnancy, Rh isoimmunisation remains a determining factor responsible for perinatal morbidity and mortality.

Key words: Rhesus D Isoimmunisation, Hemolytic Disease of the Newborn, NICU, Neonatal jaundice

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Introduction

Hemolytic disease of the newborn secondary to Rhesus-D isoimmunisation contributes significantly to perinatal morbidity and mortality. Maternal Rh (D) alloimmunization occurs when Rh negative blood exposed to Rh (D) positive red blood cells of the fetus due to transplacental fetomaternal hemorrhage during pregnancy, accidental transfusion of Rh (D) positive blood and any intervention during pregnancy. Once, anti-D Ig antibodies are present in the pregnant women's circulation, they cross the placenta and destroy fetal RBC leading to hemolytic disease of the fetus or newborn. They range from hyperbilirubinemia, severe anemia to hydrops fetalis. Unlike the ABO blood group system, there is no preformed Rh antibody. Once an RhD-negative woman becomes immunized, all subsequent pregnancies with an RhD-positive child will be affected, requiring extensive monitoring and timely prenatal or postnatal interventions.¹

Postnatal anti-D-prophylaxis was introduced in the late 1960s and the risk of being sensitized decreased from 13% to approximately 1%.² More recently, routine antenatal anti-D prophylaxis (RAADP) in the third trimester was introduced in several countries, reducing the prevalence of RhD immunization further to 0.2–0.3%.^{3,4} Rh negative pregnancy is a high risk pregnancy as it can lead to perinatal loss of 1 to 2.5%.⁵ The genetic locus for the Rh antigen complex is on the short arm of chromosome 1. In all cases of Rh-negative women irrespective of blood grouping and parity, IgG antibody is detected by indirect Coombs' test.

India is a country with diversities based on race, religion and creed. Hence, diversity has been observed in the distribution of blood groups in the population. In India, the incidence of Rh-negative is 5-10%.⁶ The objective of this study was to know the perinatal outcome in Rh-negative pregnancies at tertiary centre of Bihar.

Material and Methods

This was a prospective observational study conducted in all Rh negative pregnant women who presented in labor room of Department of Obstetric and Gynecology and Department of Pediatrics in Patna Medical College and Hospital, Patna from October 2012 to September 2014. All pregnant women with

Rh negative blood group presented in labor room irrespective of their age, parity, gestational age and administration of Rh anti-D Ig in previous or present pregnancy were included in the study. A proper history of patients was taken, general and obstetrical examination was done and all the routine antenatal investigations along with Indirect Coombs' Test (ICT) were sent. At time of birth cord blood was sent for CBC, bilirubin, hemoglobin and Direct Coombs' Test (DCT). All neonates required NICU admission referred to Pediatrics Department and further management was done there. Perinatal outcome in form of gestational age, mode of delivery, baby weight, NICU admission, Stillbirth, Neonatal Death and neonatal jaundice.

Result

The total number of Rh negative deliveries were 130 from a period of two years. Highest incidence was found in 21-25 years age group, i.e. 60% as many couples plan family during this age group (Table 1). In our study, 44.61% were primigravida, which predominated over other parity (Table 1). Many of the antenatal women had O negative blood group, i.e. about 39.23% (Table 1). 83.84% had negative indirect Coombs test and 16.15% had positive indirect Coombs test (Table 1). Majority of neonate delivered vaginally i.e. 80.76% and only 15% delivered by cesarean section either emergency or elective (Table 2). Out of 130, 35% delivered before 37 week and 2.30% was fresh stillbirths and 3% macerated stillbirths (Table 3,4). 4.86% of neonates had Apgar of 0-4, whereas 9.75% of neonates had an Apgar score of 5-8 (Table 3). Majority of the babies were weighing between 2.5 and 3 kg (Table 3). Most of the babies had Rh-positive blood group and only 16.26% of babies had Rh-negative blood group (Table 3). Majority of newborn babies, i.e. about 78.86% had bilirubin levels between 10-15 mg/dL (Table 3).

Out of 7 Stillbirths, 5 were in ICT positive mother. 17 babies had APGAR less than 8 in which 12 was born from ICT positive mother. Out of 23 preterm babies, 19 had isoimmunised. Jaundice was present in almost all babies of ICT positive mother. (Table 4). Out of 34 babies admitted in NICU, 21 had isoimmunisation due ICT positive in mother. (Table 5). Phototherapy was needed in 26 babies in 21 were from ICT positive mother. Exchange transfusion was

Table 1 : Maternal characteristics

	Number	Percentage
Age		
<20 years	15	11.53
21-25 years	78	60
26-30 years	32	24.61
>30 years	05	3.84
Gravida		
primigravida	58	44.61
G2	36	27.69
G3	24	18.46
G4 or >	12	09.23
Indirect comb test (ICT)		
ICT +ve	21	16.15
ICT -ve	109	83.84

Table 2 : Gestational age and Mode of delivery

	Number	Percentage
Gestational age at time of delivery		
< 34 week	20	15.38
34-36 week	26	20
37- 42 week	77	59.23
>42 week	7	5.38

needed in only babies having ICT positive mother. Out of 5 neonates admitted to NICU, 4 had early neonatal death. (Table 6).

Discussion

Rhesus isoimmunization causing erythroblastosis fetalis is a distressing obstetric problem which is still seen in large numbers in India. It is the single most common yet preventable cause of HDN and also an important cause of neonatal hyper bilirubinemia.

Factors contributing to the grave sequelae resulting from mismanagement of pregnancy in Rh negative women are: No prenatal care (home deliveries), non-availability of Rh testing in many health centers especially in peripheries; inadequate or no anti-D prophylaxis antenatally (after abortion including medical termination, ectopic pregnancy, threatened abortion, ante partum hemorrhage) or even post-natally many a times.⁷

There are two prophylaxis, one is antenatal anti-D prophylaxis and second is post-natal anti-D prophylaxis. FOGSI recommends a single dose of 300 mcg at 28 weeks followed by post-natal prophylaxis by 300 mcg as soon as possible if the baby in Rh

Table 3: Neonatal outcome

	Number	Percentage
Delivery Outcome		
Alive	123	94.61
Fresh IUD	3	2.30
Macerated IUD	4	3.07
Blood group		
Rh -ve	20	16.26
Hemoglobin status		
<12 gm/dl	13	11.38
12-18 gm/dl	110	89.43
Cord blood hyperbilirubinemia		
10-15 mg/dl	104	84.55
16-20 mg/dl	13	11.56
21-25 mg/dl	06	4.87

Table 4: Comparison of outcome according to ICT status in mother

	ICT positive (n=21)	ICT negative(n=109)
IUD	05	02
APGAR less than 8	12	05
Baby weight <2.5	19	07
Hemoglobin status <12	11	02
Bilirubin > 15	17	002

Table 5: NICU admission and treatment given

Cause of admission	ICT positive	ICT negative
Neonatal jaundice	19	09
Meconium aspiration	01	01
Respiratory distress syndrome	01	03
Treatment given		
phototherapy	16	05
Exchange therapy	5	00

Table 6: NICU Outcome

Outcome	Number
Early neonatal death	4
improved	30

positive and DCT is negative and 100 mcg anti-D after the sensitising event of the first trimester.⁸ This postpartum anti-D dose is sufficient enough to neutralise 30 mL of fetal blood. The present study was undertaken to show the burden of HDN due to Rh incompatibility, which is a preventable condition.

In our study, highest prevalence was found in 21-25 years age group, as many couples plan family during this age. In our study, the most common blood group with Rh-negative phenotype was O (39.23%), followed by B (31.53%), A (27.69%), AB (4.61%).

The study from Enugu, South East Nigeria, showed that the most common blood group with Rh-negative phenotype was O (64.5%), followed by A (20%), blood group B 12.1% and AB 3.2%, respectively.⁹ In the study by Agarwal S et al, the most common blood group with Rh negative phenotype was O (39%), A (17%), B (16%), AB (17%).⁶

In the present study, 44.61% were primi gravida, which predominated over other parity. Primipara constituted 48.5% of the study population in a study by Adeyemi AS et al.¹⁰ Since primipara constituted the greatest proportion of the RhD-negative obstetric population, there is need for defined protocol, which will make for proper and adequate management of this population so as not to compromise the reproductive career of these women. Nearly, 80.76% of women had normal deliveries and 15% delivered by caesarean section.

Regarding neonatal outcome, out of 130, 123 were liveborn babies, 3 was fresh stillbirth and 4 was old IUD and 5 were early neonatal deaths in our study. One patient had positive indirect Coombs test leading to stillbirth and another two patient with positive ICT reported with IUD. Five babies had severe anemia. In our study, 4.87% had Apgar score of 0-4, 9.75% had 6-8 Apgar score and 86.17% had 8-10 Apgar score. This finding was comparable with other study.¹¹

In our study, out of 123 liveborn babies, 34 babies were admitted in NICU. Out of 34 babies, 28 babies had early onset of jaundice as they were Rh-positive babies needing NICU admission. 6 babies had bilirubin levels more than 21 mg/dL and died within 7 days of birth. In a study by a higher proportion of neonates with Rh incompatibility, had hyper bilirubinemia within 72 hours of life. Thus, neonates with Rh isoimmunisation had significantly higher incidence of jaundice within 72 hours of life.¹²

Conclusion

Despite the fact that the prevalence of Rh-ve pregnancy is lower than 5%, Rh isoimmunisation

remains significant factor for perinatal morbidity in most developing countries. Hence, the primary aim should be prevention of alloimmunization in Rh negative pregnancy. Every Rh negative pregnancy should be screened for antibody visoimmunization at first antenatal visit.

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Silent Rupture of Inverted 'T' shaped Uterine Caesarean Scar in Late pregnancy : A Tragic Tale of Ignorance

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Abstract

With the increasing trend towards cesarean section, the incidence of uterine rupture is increasing, more so in third world countries, making it a public health hazard.

We report a near miss mortality case of a 30 year old unbooked patient, gravida 2nd, at 34 weeks gestation with history of previous one cesarean section with no living issue presenting to emergency department in a state of shock. Clinically uterine rupture was suspected. At emergency laprotomy, 2.5 litres hemoperitoneum noted, dead foetus and placenta removed from peritoneal cavity and sub-total hysterectomy done. Patient shifted to intensive care unit post operatively. The tragic fate met by this patient with her ending up in hysterectomy and having no living issue and more tragically no chance of future pregnancy can be attributed to her "Delay in seeking appropriate medical care". There is ignorance amongst Indian rural women regarding need for seeking appropriate medical care during pregnancy and labour, hence, leading to catastrophes and adverse foeto-maternal outcome.

Background

Uterine rupture is a catastrophic and life threatening obstetric emergency and is associated with high maternal and perinatal mortality rates. It may be incomplete or complete. In incomplete rupture the peritoneum is still intact whereas with a complete rupture the contents of the uterus may spill into

the peritoneal cavity or the broad ligament. Usually uterine rupture occurs in active labour, but rarely it may occur in late pregnancy as well, more so in previously scarred uterus. A uterine scar from a previous cesarean section is the most common risk factor as cesarean section is the most common surgical procedure in obstetrics.¹ The rates of cesarean section are currently above the levels of reference stated by the World Health Organization.¹⁻² With the increasing trend towards cesarean delivery,³ its complications, including uterine rupture are increasing. The incidence of uterine rupture following lower segment cesarean section is 0.2-1.5%, much lesser than that associated with classical cesarean section i.e. 4-9%. Sometimes during lower segment cesarean section, there is encountered difficulty in delivery of the baby (e.g transverse lie, obstructed labour), hence there

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may arise a need to extend the lower segment uterine incision into the upper segment in a 'J' shaped or inverted 'T' shaped fashion. Such incisions involving the upper segment place the woman at higher risks for uterine rupture in future pregnancies, the risks being same as those for classical cesarean sections. Other risk factors for uterine rupture include: myomectomy,⁴ dilatation and curettage, dysfunctional labour, labour augmentation by oxytocin or prostaglandins, and high parity.⁵ Most cases of ruptured uterus are preventable with good antenatal care, intra-partum care and proper identification of high risk cases. Hence, its incidence is higher in developing countries like India as compared to developed nations, as former have to still go a long way to provide basic maternal health care facilities to the women. In India, despite strict implications of government programmes like National Rural Health Mission, Janani Shishu Suraksha Karyakram and Millennium Development Goals, such catastrophes being rampant, is a harsh reality. Poverty & ignorance on part of women add to this menace. Delays in seeking appropriate care, a poor referral system, non-attendance of antenatal care, and delay in receiving care due to lack of skilled human resources and medical consumables have made uterine rupture a public health concern. Worldwide annually there occur 529,000 maternal deaths and 3.3 million still births.^{6,7} More than 90% of these occur in developing countries, and ruptured uterus accounts for more than 31.9% of maternal and 96.3% of perinatal deaths.⁸ We hereby report a tragic case of a woman presenting with silent rupture in Inverted 'T' shaped Uterine Cesarean Scar in Late pregnancy with fetal demise.

Case Description

A 30 year old, unbooked patient, gravida 2, para 1 with no living issue, at 34 weeks of gestation, with history of previous one cesarean section for an unknown indication, presented to us for the first time, in state of hypovolemic shock and chief complaint of continuous dull aching abdominal pain. The patient had neither taken any antenatal checkups nor had got any investigations done in present pregnancy. Her past obstetric history revealed that in her previous pregnancy three years back, during labour, she was handled by a local village dai, who failing desperate attempts to deliver her baby vaginally for few hours, finally told her relatives to seek medical help. Following this she was taken up for cesarean section at

the district hospital where she delivered a dead baby. However records related to her previous pregnancy were not available.

On clinical examination, she had severe pallor, pulse was 120 beats per minute, blood pressure 100/60 mm Hg, respiratory rate 20 per minute. On per abdomen examination, abdomen was tense distended and tender, uterine contour lost and fetal parts felt superficially in left para colic gutter. There was fluid thrill and shifting dullness. Fetal heart sound was not audible. An emergency positive culdocentesis was done. On per vaginal examination, there was no bleeding, cervix was uneffaced and os was closed. High suspicion of rupture of uterus was made clinically and patient was immediately taken up for emergency laprotomy and meanwhile, resuscitated and blood arranged. Her investigations revealed haemoglobin of 5.7 gm/dl and TLC of 22,000/cumm. General anaesthesia was administered. Abdomen was opened longitudinally (sub umbilical midline incision). There was approximately 2.5 litres of hemoperitoneum. A midline longitudinal anterior uterine wall rupture 14 – 15 cm in length was noted extending from fundus upto utero-vesical fold of peritoneum. Also, a transverse rupture, at lower uterine segment was noted, hence giving characteristic inverted 'T' shape to the uterine rupture (Figure 1). A dead macerated female baby weighing 1.9 kg, along with placenta, was removed from the peritoneal cavity (Figure 2). The uterus was however beyond repair. Quick life saving subtotal hysterectomy had to be taken which was tragic as the patient had no living issue. Patient was transfused two units of PRBC intra-operatively. Patient was shifted to ICU and was administered three units of PRBCs in post-operative period. Her post operative period was otherwise un-eventful and she was shifted out to maternity ward at 3rd post operative day. On 10th post operative day the patient was discharged after stitch removal in stable condition.

Discussion

Uterine rupture is a known complication during pregnancy and labour. The incidence of uterine rupture is more with scarred uterus than with unscarred uterus. In this case, the woman in her current and first pregnancy had not taken any antenatal care. In her previous pregnancy, she was taken to a untrained village dai, who failed to deliver her baby, following



Figure 1: A midline longitudinal anterior uterine wall rupture 14 – 15 cm in length noted extending from fundus upto utero-vesical fold. Also ,a transverse rupture, at site of previous lower segment cesarean scar was noted, hence giving characteristic inverted 'T' shape to the uterine rupture.

which she was taken to a district hospital where she was taken up for emergency cesarean section. Probably, the doctor might have encountered difficulty to deliver the baby, which might have led him to convert the lower segment uterine incision into an upper segment inverted 'T' shaped incision. This in her current pregnancy would have made her highly susceptible to uterine rupture. The patient had presented to us for the first time in state of shock. The woman and her relatives in this case had to face the trauma of peripartum hysterectomy and fetal loss owing to their ignorance. Delay in seeking appropriate medical care in both her pregnancies made her lose her two babies and ended up with her hysterectomy, with her having no living issue and unfortunately no chance of future pregnancy.



Figure 2: A dead macerated baby weighing 1.9 kg, along with placenta, was removed from the peritoneal cavity.

Conclusion

Ensuring regular antenatal check-ups, timely intervention, strengthening the referral system help improve maternal and perinatal outcomes. This case represents just a "tip of the iceberg ". It points out that despite governments desperate efforts to improve maternal and child health care facilities, we still have a long way to go to ensure safe motherhood to each and every woman in our country.

Acknowledgment

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Disclosure

"We confirm that the article is original and is not under consideration by another journal. We sign for and accept responsibility for releasing this article ."

ABBREVIATIONS:

TLC : Total Leucocyte Count

PRBC: Packed Red Blood Cells

ICU: Intensive Care Unit

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Evaluation of Periodontal Status Among Post Menopausal Women

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Abstract

Puberty, menses, pregnancy and menopause have a varied influence on oral health. Menopause is associated with significant adverse changes in the orofacial complex and has been associated with destructive periodontal diseases. Periodontal disease refer to both gingivitis and periodontitis.

The aim of our study was to evaluate the periodontal status of postmenopausal women.

A sample of 50 postmenopausal women were evaluated for plaque index (PI), gingival index (GI), probing depth (PD), and clinical attachment loss (CAL). The data so collected was statistically analysed.

In the study group, mean age of patient was 55 years, mean PI was 1.59, mean GI was 1.59, PD was 5.46, CAL was 4.46.

The results of this study suggest that females after menopause are at a risk of developing destructive periodontal disease. Attention to oral care and regular treatment can help manage periodontal problems.

Getting to the root issue of hormone imbalances can help women to avoid the risk of periodontal disease.

Introduction

The homostasis of the periodontium involves complex multifactorial relationships. Puberty, menstruation, pregnancy and menopause have varied influences on oral health.

Menopause is defined as permanent cessation of menses for one year and is physiologically correlated with the decline in estrogen secretion resulting from the loss of follicular function. Because the oral mucosa contains estrogen receptors menopause is associated with significant adverse changes in the orofacial complex and has been associated with destructive periodontal diseases.¹

Progesterone level may change vascular permeability and then result in gingival swelling and inflammation and reduce resistance to dental plaque (i.e. bacteria), while change in estrogen hormone level can cause alteration in immune function and changes in flora ecology of the mouth.²

Studies suggest that low estrogen production after menopause is associated with increased production of interleukin 1 (IL-1), IL-6, IL-8, IL-10, tumour necrosis factor alpha, granulocyte colony stimulating factor, and granulocyte-macrophage colony-stimulating factor, which stimulates mature osteoclasts, modulates bone cell proliferation, and induces resorption of both skeletal and alveolar bone.^{3,4}

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Periodontal disease refers to various forms of gingivitis and periodontitis. Gingivitis is a reversible inflammatory response to bacterial plaque build up that is limited to the gingiva. Gingivitis may progress in some patients to periodontitis, an inflammation of the supporting tissues of the teeth, including the gingiva, alveolar bone, and periodontal ligament. Periodontitis leads to progressive and irreversible loss of bone and periodontal ligament attachment, as inflammation extends from the gingiva into adjacent bone and ligament.

Aims and Objectives

The aim of this study was to evaluate the periodontal status of postmenopausal women.

Material and Methods

A sample of 50 postmenopausal women attending Gynaecology OPD of Nalanda Medical College and Hospital, Patna were randomly selected.

Inclusion Criteria:

1. Systemically healthy postmenopausal women with age group between 45 and 55 years.
2. Not undergone any type of periodontal therapy 6 months prior to the initial examination.

Exclusion Criteria:

1. Patients who need antibiotic prophylaxis
2. Smokers
3. Patients on long term steroid medication, hormone replacement therapy (HRT) and calcium

Informed consent was obtained from all patients. A calibrated periodontal probe (UNC-15) was used to evaluate plaque index (PI), gingival index (GI), probing depth (PD), and clinical attachment loss (CAL). The data so collected was statistically analysed.

Results

In the study group, mean age of patient was 55 years (Range 45-55 years). Among 50 patients, 12 were clinically healthy with GI less than 2, 22 patients had gingivitis with GI more than 2 and PD less than 3 whereas 16 patients were suffering from periodontitis with PD more than 3. Mean Plaque index (PI) was 1.58 (Range 0.00- 3.0), mean Gingival index (GI) was 1.49 (Range 0.00-3.0), mean Probing depth (PD)

was 4.36mm (Range 2.0 - 6.0mm) and mean Clinical Attachment Level (CAL) was 5.46mm (Range 2.0 – 7.0mm).

Clinical Status	Gingival Index (GI) Probing Depth (PD)	Number
Clinically Healthy Subjects	GI < 2	12
Gingivitis	GI ≥ 2 PD < 3	22
Periodontitis	PD > 3	16

Table showing periodontal status among 50 postmenopausal women

Discussion

Throughout a woman's life, hormonal changes affect tissues throughout the body. The promptly fluctuating hormonal levels in menopausal women are one of the key factors that are answerable to the alterations detected within the oral cavity. An increase in gingivitis, periodontal disease, tooth loss and dry mouth has been reported.⁵

Endocrinal alteration induced bone resorption appears to be the principal pathogenic mechanism underlying accelerated bone loss in postmenopausal women with no direct relationship between the two phenomena.⁶⁻¹⁰

A number of studies have shown that changes in periodontal conditions might be associated with variations in sex hormone levels.¹¹ Sex steroid hormones have been shown to directly and indirectly exert influence on cellular proliferation, differentiation and growth in target tissues, including keratinocytes and fibroblasts in the gingiva.¹²⁻¹⁹

According to Payne et al, 1997; Reinhardt et al, 1994 estrogen deficiency is considered to be involved in the progression of periodontal disease during postmenopausal period.²⁰

Varghese et al compared periodontal status in pre and postmenopausal women and concluded that periodontitis was significantly greater in postmenopausal women. In their study, PI was 1.59 ± 0.26 , PD was 4.46 ± 1.03 , GI was 1.59 ± 0.34 and CAL was 4.46 ± 1.03 in post menopausal women. These values are in are in close similarity to the results of the present study.²¹

According to a study conducted by Thomas KE in 110 subjects of each of pre and post menopausal women, it was concluded that post menopausal women are subjected to severe oral health problem when compared to their premenopausal counterparts.²²

However, according to a cross-sectional study in a Portuguese population by Ricardo C Alves et al, the difference in attachment loss in post menopausal women suffering from periodontitis and the control group was not statistically significant, leading the authors to conclude that menopause does not appear to significantly influence the severity of periodontal disease and tooth loss. Other factors may exert a greater influence on the progression of periodontal disease rather than menopause itself.²³

After reviewing the literature on the effects of menopause on periodontium, Amit Bhardwaj and Shalu Bhardwaj have concluded that female sex hormones are neither necessary nor sufficient to produce gingival changes by themselves. However, they may alter periodontal tissue responses to microbial plaque and thus indirectly contribute to periodontal disease.²⁴

Thus, there is a contradiction in the literature regarding periodontitis in postmenopausal women. This requires further studies with larger sample sizes.

Conclusion

Although the sample sizes of this study was small, results of this study suggest that most females after menopause have gingival and periodontal conditions which are not clinically healthy. Attention to oral care and regular check-ups can help manage Periodontal problems.

This study will thereby help to create awareness among post menopausal women to go for a routine dental visit before progression to an active periodontal disease. Further studies are needed for the effect of menopause on periodontal status as periodontitis is a multifactorial disease.

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Segmental Mullerian Anomalies Causing Endometriosis: a Case Report

Dr Pallab Kumar Mistri,¹ Prof Bandana Biswas,² Dr Abhishek Bhadra,³
Dr Shouvik Das⁴

Introduction

The Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome is characterized by congenital aplasia of the uterus and the upper 1/3 of the vagina in women showing normal development of secondary sexual characteristics and a normal 46, XX karyotype. It affects about 1 out of 4500 women. The first sign of MRKH syndrome is primary amenorrhea in young women presenting otherwise with normal development of secondary sexual characteristics and normal external genitalia, with normal and functional ovaries. The phenotypic manifestations of MRKH syndrome overlap with various other syndromes or associations and thus require accurate delineation. Here, we report a rare case of MRKH syndrome who presented with cyclical lower abdominal pain along with primary amenorrhoea.

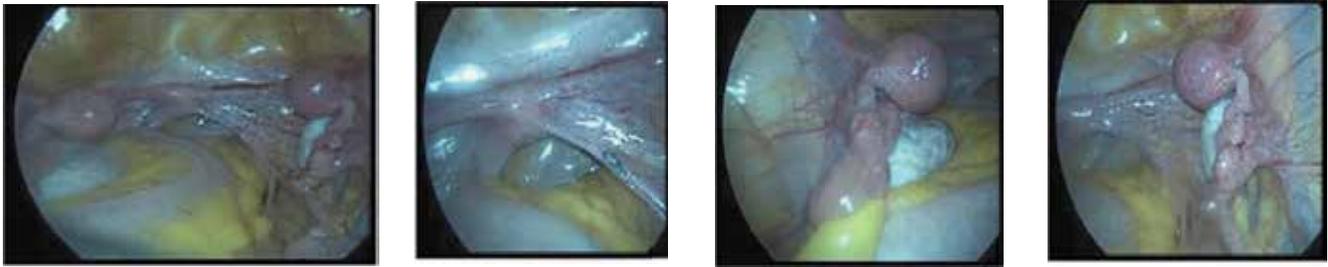
Case Report

A 22-year old unmarried nulliparous woman, resident of Hooghly, student by occupation, presented to our OPD with primary amenorrhoea associated with cyclic lower abdominal pain for last 4 years. Patient

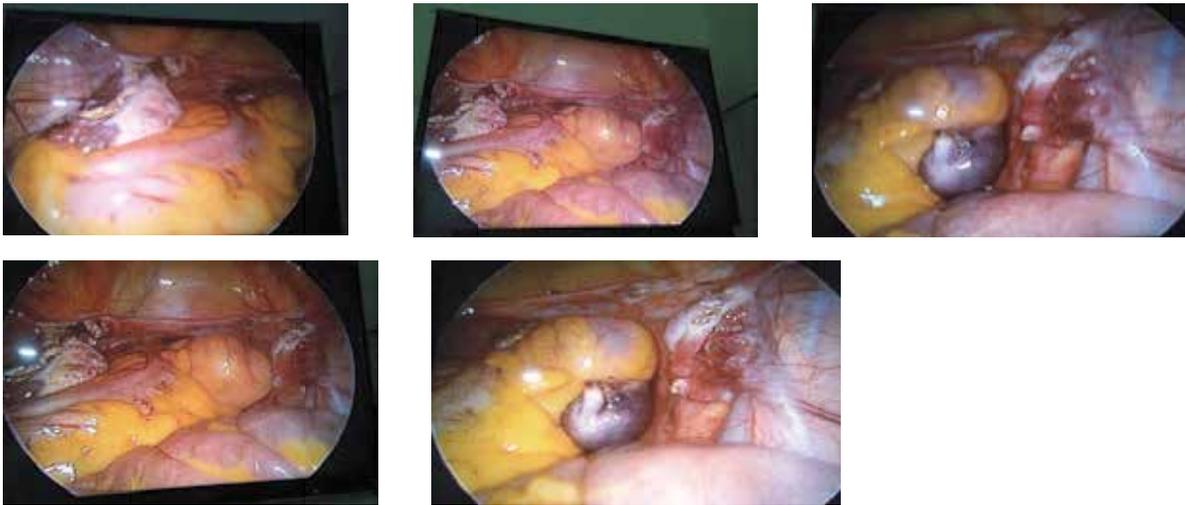
stated that she never menstruated and that there was gradually worsening dull-aching pain in the lower abdomen every month for the past 4 years, radiating to the lower back and associated with abdominal heaviness and usually relieved by NSAIDs. She noticed thelarche at the age of 11 years and developed pubic hair at 13 years. Her developmental milestones are normal. There was no history of galactorrhoea, excessive hair growth, or hoarseness of voice, visual disturbances, headache, anosmia, seizure disorder, head injury, strenuous exercise, anorexia, prolonged fasting, psychological stress, lethargy, constipation, cold intolerance, retention of urine or symptoms of TB. There was no history of systemic illness or chronic medication use. There was no family history of amenorrhoea. Her mother has normal menstrual cycles.

On examination, patient was alert, conscious, cooperative and oriented to time, place and person. She was 150 cm tall, weight 52 kg, BMI 21.9 kg/m² and was hemodynamically stable. She had unremarkable per abdominal findings, normal urethral position, normal-appearing female external genitalia, no inguinal or labial swelling, normal clitoris, normal hymen and a short vagina, normal breasts (Tanner 5) and also had normal pubic (Tanner 5) and axillary hair. On per rectal examination, there was a small nodular mass felt anterior to the rectum. There were no dysmorphic features.

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Figures showing diagnostic laparoscopy findings



Figures showing laparoscopic excision of rudimentary uterine horns

Pelvic ultrasonography showed absence of uterus and cervix, normal bilateral ovaries, kidneys and urinary bladder. Her X-ray spine, chest X-ray, ECG were unremarkable. Her karyotyping was normal (46, XX). The patient's blood investigations were as follows: hemoglobin 11.2 g/dl, fasting blood sugar 93 mg/dl, serum urea 26 mg/dl, serum creatinine 0.8 mg/dl, TSH 2.08 μ IU/ml, FSH 8.9mIU/ml and prolactin 10 ng/ml.

Patient underwent diagnostic laparoscopy followed by McIndoe's vaginoplasty in the same sitting. During laparoscopy, it was found that there was rudimentary uterine horns, about 3x3 cm in size, on both sides with no midline fusion, bilateral healthy Fallopian tubes and ovaries and one endometriotic spot over the uterosacral ligament. Patient was advised self-dilatation of neovagina. She was also administered Inj. Leuprorelin acetate (3.75 mg) intramuscularly monthly for 3 months. Removal of functioning uterine horns was planned in the next sitting. The patient was followed up over the next 3 months and she had a remarkable symptomatic relief. She also had adequate vaginal length. After 3 months,

patient underwent a second sitting of laparoscopy. Laparoscopy was done with 3 trocars – the main umbilical 10-mm port for laparoscope and 2 5-mm ancillary ports in lower abdomen. The rudimentary uterine horns and Fallopian tube of each side were excised one by one using Ligasure®. The specimens were retrieved through the umbilical port and sent for histopathological examination. Haemostasis was secured. The patient had an uneventful intra-operative and postoperative period.

Discussion

Congenital malformations of female genitalia are often a challenge for doctors due to the wide variety of possible diagnoses. The Müllerian and Wolffian ducts are essential for the development of the female and male reproductive system respectively. The Mullerian ducts mature to become fallopian tubes, uterus, cervix and upper two-third of the vagina, while the Wolffian duct degenerates. The absence of the Mullerian Inhibiting Factor (MIF) or anti-mullerian hormone is the driving force behind maturation of the mullerian ducts to become the above mentioned portions of the female

reproductive system. MRKH syndrome is characterized by aplasia of the Müllerian duct structures in a person who has karyotype 46, XX with female phenotype characteristic of primary amenorrhea in adolescence. The lower third of the vagina, the ovaries and external genitalia in these cases do not usually have alterations. MRKH syndrome is subdivided into two types: Type I (isolated) or Rokitansky sequence and Type II or MURCS association. Although most MRKH patients have a rudimentary nonfunctioning uterus, a small percentage (2-7%) do have a uterus with a functioning endometrium. Approximately 40-60% of patients have renal disorders such as unilateral agenesis, horseshoe kidney, ectopic or bilateral uteropelvic obstruction. In addition, 20% had bone changes, thoracocervical asymmetry, spinal fusion, scoliosis or Klippel-Feil syndrome. Cardiac abnormalities and hearing defects can also be encountered.

Treatment is usually delayed until the patient is ready to begin sexual activity. These patients often suffer from depression. Psychological counselling and surgical procedures to create a neovagina when the patient is ready to start sexual activity are the main modalities of treatment for MRKH. Reproductive options do exist for patients diagnosed with MRKH syndrome in the form of gestational surrogacy and probably, uterine transplantation in the near future.

Conclusion

Our patient was unique in the sense that she had an uncommon form of MRKH syndrome with bilateral rudimentary horns with functioning endometrium. The diagnosis of MRKH syndrome entails ethical and management challenges that should be discussed with the patient and her family. The patient's future marital and reproductive life is based upon such decisions.

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- [4] World Health Organization. WHO Recommended Surveillance Standards, Second Edition [WHO website]. 1999. <http://www.who.int/csr/resources/publications/surveillance/whocdscsr992.pdf>.

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