كلية : كلية الطب العام

القسم او الفرع : النسائية والتوليد

المرحلة: الرابعة

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اسم المادة باللغة العربية :التوليد

اسم المادة باللغة الإنكليزية : Obstetrics

اسم المحاضرة السادسة عشر باللغة العربية: تمزق الاغشية المبكر وامراض كمية السائل الامينوسي

اسم المحاضرة السادسة عشر باللغة الإنكليزية : Preterm Premature Rupture Of Membranes (PPROM) and Aberrant liquor volume

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Preterm Premature Rupture Of Membranes (PPROM) and Aberrant liquor volume

Defined as rupture of the membranes before labor and prior to 37 weeks, preterm premature rupture of membranes can result from a wide array of pathological mechanisms, including intra-amnionic infection. Other factors implicated include low socioeconomic status, low body mass index—less than 19.8, nutritional deficiencies, and cigarette smoking. Women with prior preterm ruptured membranes are at increased risk for recurrence during a subsequent pregnancy. Most cases of preterm rupture, however, occur without risk factors.

PPROM occurs in approximately 2% of all pregnancies and accounts for up to one-third of preterm deliveries. The earlier in pregnancy that PPROM occurs the shorter the interval to delivery. Although postnatal survival following PPROM is directly related to birthweight and gestational age at delivery, in pregnancies complicated by PPROM prior to 23 weeks, pulmonary hypoplasia may develop leading to an increased risk of neonatal death, even if delivery occurs at later gestational ages. Pulmonary hypoplasia following PPROM occurs in approximately 50% of women with PPROM at 19 weeks, falling to about 10% at 25 weeks. The presence of amniotic fluid greater than 2 cm on ultrasound is associated with a lower incidence of pulmonary hypoplasia.

Clinical features of preterm pre-labour rupture of the membranes

History:

The most reliable diagnostic feature of PPROM from the history is the report of a ‘gush of fluid’ vaginally, usually followed by a more-or-less continuous dribble. This must be distinguished from leaking urine (ask about frequency, urgency, leakage and dysuria), as incontinence or a urinary tract infection (UTI) may present in a similar way. The presence of any vaginal discharge should be ascertained. Fetal movements may be reduced in strength or frequency after PPROM, and occasionally uterine irritability or contractions may be reported.

Examination:

Infection may lead to an increased pulse and temperature and a flushed appearance. Abdominal examination may reveal a clinical suspicion of oligohydramnios or uterine tenderness if chorioamnionitis is present. The definitive diagnosis of PPROM can only be made by performing a sterile speculum examination, preferably after the patient has been resting supine for 20–30 minutes. A pool of amniotic fluid in the posterior vagina is diagnostic.

It is also important at this point to visualize the cervix. Fluid may be seen trickling through the external os and dilatation can be assessed. Digital vaginal examinations should be avoided if possible in PPROM, as they are associated with a significant reduction in the latent interval before labour. This reduction is most dramatic at the earliest gestations.

Differential diagnosis

1. Urine loss: incontinence and UTIs are both more common in pregnancy.
2. Vaginal infection.
3. Leukorrhoea: the cervical glands often become overactive during pregnancy.

Investigations

Nitrazine testing:

Amniotic fluid is alkaline, whereas the vaginal secretions are usually acidic. An elevated pH turns a nitrazine stick blue. Unfortunately,false positives occur, with blood, semen and even urine limiting its usefulness. However, the predictive value of a negative test is very high.

Genital tract swabs:

A high vaginal swab may help to guide antibiotic therapy, if subsequently required. Screening for group B streptococcus (GBS) can also be performed, as there is a substantial risk of labour in the next few days.

Maternal well-being

This should include regular assessment of the mother's blood pressure, pulse and temperature. Some advise serial white cell counts and C-reactive proteins as early markers of infection, although this has not been shown to improve management.

Fetal well-being

Serial antepartum cardiotocography is important after PPROM, as a gradually increasing baseline heart rate or fetal tachycardia can be the first sign of intrauterine infection.

Ultrasound

Ultrasound can give valuable information about the amniotic fluid volume. The presence or absence of oligohydramnios provides further diagnostic support.

In established PPROM, there is a direct correlation between the amount of amniotic fluid remaining and the latency period. Unlike preterm labour, cervical length measurements do not have predictive ability in PPROM.

Amniocentesis

A sample of amniotic fluid can be sent for Gram stain, microscopy and culture, to establish whether there is an intrauterine infection (chorioamnionitis). There is, however, a risk of stimulating preterm labour by performing an invasive test, and amniocentesis can be technically very difficult when there is little amniotic fluid.

Natural History of Preterm Ruptured Membranes

The time from preterm ruptured membranes to delivery is inversely proportional to the gestational age when rupture occurs .very few days were gained when membranes ruptured during the third trimester compared with midpregnancy.

Hospitalization

Most clinicians hospitalize women with preterm ruptured membranes. Concerns about the costs of lengthy hospitalizations are usually moot, because most women enter labor within a week or less after membrane rupture. No benefits were found for hospitalization, and maternal hospital stays were reduced by 50 percent in those sent home—14 versus 7 days.

Intentional Delivery

Prior to the mid-1970s, labor was usually induced in women with preterm ruptured membranes because of fears of sepsis. Intentional delivery reduced the length of maternal hospitalization and also reduced infection rates in both mothers and neonates. Among those intentionally delivered, there were neonatal deaths—from sepsis and one from pulmonary hypoplasia. Thus, neither management approach proved to be superior.

Expectant Management

Includes intensive clinical surveillance for signs of chorioamnionitis including regular recording of maternal temperature, heart rate, cardiotocography and maternal biochemistry, with a rising white cell count or a rising C-reactive protein indicating development of chorioamnionitis. Lower genital tract swabs are routinely taken. In the majority of cases of PPROM there is time for administration of corticosteroids and in utero transfer before the onset of preterm labour(PTL).

Risks of Expectant Management

Maternal and fetal risks vary with the gestational age at membrane rupture. Some expectantly managed singleton pregnancies with ruptured membranes found no improved neonatal outcomes with expectant management beyond 33 weeks. In contrast, Many found that prolonged latency after membrane rupture was not associated with an increased incidence of fetal neurological damage.

The volume of amnionic fluid remaining after rupture appears to have prognostic importance in pregnancies before 26 weeks. Forty percent of women developed oligohydramnios defined by the absence of fluid pockets 2 cm or larger. Virtually all women with oligohydramnios delivered before 25 weeks, whereas 85 percent with adequate amnionic fluid volume were delivered in the third trimester. No observed cases of pulmonary hypoplasia in fetuses born after membrane rupture at 24 weeks or beyond. This suggests that 23 weeks or less is the threshold for development of lung hypoplasia. Further, when contemplating early expectant management, consideration is also given to oligohydramnios and resultant limb compression deformities.

Other risk factors have also been evaluated. In neonates born to women with active herpetic lesions who were expectantly managed, the infectious morbidity risk appeared to be outweighed by risks associated with preterm delivery. Some found that expectant management of women with preterm ruptured membranes and noncephalic presentation had an increased rate of umbilical cord prolapse, especially before 26 weeks.

Clinical Chorioamnionitis

Most authors report that prolonged membrane rupture is associated with increased fetal and maternal sepsis. If chorioamnionitis is diagnosed, prompt efforts to effect delivery, preferably vaginally, are initiated. Fever is the only reliable indicator for this diagnosis, and temperature of 38°C (100.4°F) or higher accompanying ruptured membranes implies infection. Maternal leukocytosis alone has not been found to be reliable. During expectant management, monitoring for sustained maternal or fetal tachycardia, for uterine tenderness, and for a malodorous vaginal discharge is warranted.

With chorioamnionitis, fetal and neonatal morbidity is substantively increased. They had a higher incidence of sepsis, respiratory distress syndrome, early-onset seizures, intraventricular hemorrhage, and periventricular leukomalacia. The investigators concluded that these very-low-birthweight neonates were vulnerable to neurological injury attributable to chorioamnionitis.

Other evidence that very small newborns are at increased risk for sepsis. Some found that intra-amnionic infection in preterm neonates was related to increased rates of cerebral palsy at 3 years. During labor, 1.6 percent of all women had fever, and this was a strong predictor of infection-related death in both term and preterm neonates.

Accelerated Pulmonary Maturation

A variety of clinical events—some well defined—were once proposed to accelerate fetal surfactant production. These included chronic renal or cardiovascular disease, hypertensive disorders, fetal-growth restriction, placental infarction, chorioamnionitis, and preterm ruptured membranes. Although this view was widely held for many years, subsequent observations do not support this association.

Corticosteroids:

According to the American College of Obstetricians and Gynecologists (2007), single-dose therapy is recommended from 24 to 32 weeks. There is no consensus regarding treatment between 32 and 34 weeks. They are not recommended prior to 24 weeks.

Membrane Repair:

Tissue sealants have been used for a variety of purposes in medicine and have become important in maintaining surgical hemostasis and stimulating wound healing.

Antimicrobial Therapy

The proposed microbial pathogenesis for spontaneous preterm labor or ruptured membranes has prompted investigators to give various antimicrobials in an attempt to forestall delivery. It were *possibly* benefited: (1) fewer women developed chorioamnionitis, (2) fewer newborns developed sepsis, and (3) pregnancy was more often prolonged 7 days in women given antimicrobials. A7-day treatment of ampicillin, amoxicillin plus erythromycin, or placebo were given. Antimicrobial-treated women had significantly fewer newborns with respiratory distress syndrome, necrotizing enterocolitis, and composite adverse outcomes. The latency period was significantly longer.

Recommended Management

1.34 weeks or more: Proceed to delivery, usually by induction of labor

- Group B streptococcal prophylaxis is recommended.

2.32 weeks to 33 completed weeks: Expectant management unless fetal pulmonary maturity is documented.

- Group B streptococcal prophylaxis is recommended.
- Corticosteroids—no consensus, but some experts recommend.
- Antimicrobials to prolong latency if no contraindications.

3. 24 weeks to 31 completed weeks: Expectant management

- Group B streptococcal prophylaxis is recommended.
- Single-course corticosteroids use is recommended.
- Tocolytics—no consensus.
- Antimicrobials to prolong latency if no contraindications.

4. Before 24 weeks: Patient counseling

- Expectant management or induction of labor.
- Group B streptococcal prophylaxis is not recommended.
- Corticosteroids are not recommended.
- Antimicrobials—there are incomplete data on use in prolonging latency.

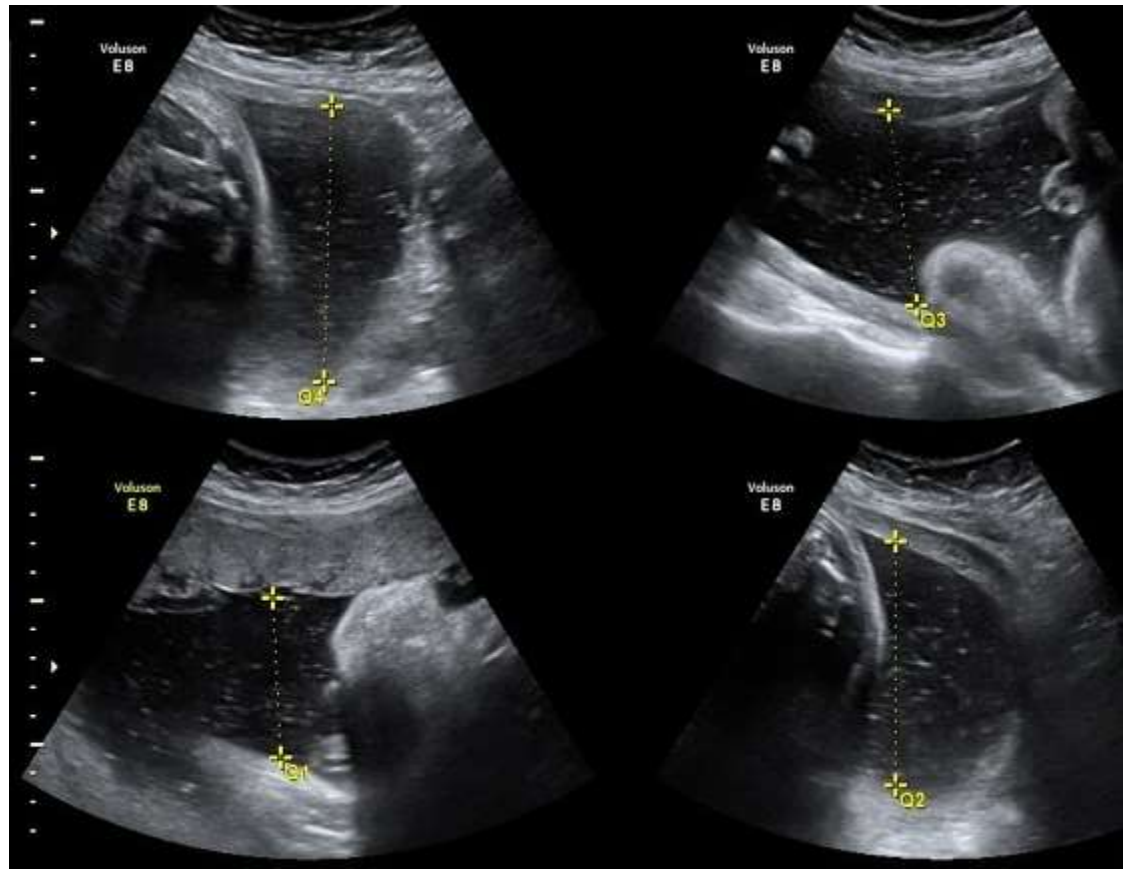
Aberrant liquor volume

Amniotic fluid is produced almost exclusively from fetal urine from the second trimester onwards. It serves a vital function in protecting the developing baby from pressure or trauma, allowing limb movement, hence normal postural development, and permitting the fetal lungs to expand and develop through breathing.

A reduction in amniotic fluid volume is referred to as ‘oligohydramnios’ and an excess is referred to as ‘polyhydramnios’. Definitions of oligohydramnios and polyhydramnios are based on sonographic criteria. Two ultrasound measurement approaches give an indication of amniotic fluid volume. These are maximum vertical pool and amniotic fluid index.

The maximum vertical pool is measured after a general survey of the uterine contents. Measurements of less than 2 cm suggest oligohydramnios, and measurements of greater than 8 cm suggest polyhydramnios. The amniotic fluid index (AFI) is measured by dividing the uterus into four ‘ultrasound’ quadrants. A vertical measurement is taken of the deepest pool of fluid that is free of umbilical cord in each quadrant and the results summated.

The AFI alters throughout gestation, but in the third trimester it should be between 10 and 25 cm; values below 10 cm indicate a reduced volume and those below 5 cm indicate oligohydramnios, while values above 25 cm indicate polyhydramnios.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Oligohydramnios

Too little amniotic fluid (oligohydramnios) is commonly defined as amniotic fluid index (AFI) less than the 5th centile for gestation. Oligohydramnios may be suspected antenatally following a history of clear fluid leaking from the vagina; this may represent PPROM. Clinically, on abdominal palpation the fetal poles may be very obviously felt and ‘hard’, with a small for dates uterus.

Possible causes of oligohydramnios and anhydramnios

1.Renal agenesis: diagnosis by ultrasound no renal tissue and no bladder.

2.Multicystic kidney, Ultrasound: enlarged kidneys with multiple cysts, no visible bladder.

3.Urinary tract abnormality/obstruction: ultrasound, kidney may be present but urinary tract dilatation.

4.FGR and placental insufficiency: Clinical: reduced SFH and reduced fetal movement, possibly abnormal CTG.Ultrasound:FGR, abnormal fetal doppler wave forms.

5.Maternal drugs(e.g.NSAIDS):Withholding NSAIDS may allow amniotic fluid to reaccumulate.

6.Post date pregnancy.

7.Leakage: diagnosed by PPROM:Speculum examination:pool of amniotic fluid on posterior blade.

The fetal prognosis depends on the cause of oligohydramnios, but both pulmonary hypoplasia and limb deformities (contractures, talipes) are common to severe early-onset (<24 weeks' gestation) oligohydramnnios. Renal agenesis and bilateral multicystic kidneys carry a lethal prognosis, as life after birth is impossible without functioning kidneys. In this situation, the fetal lungs would probably be hypoplastic; this may also be true of severe urinary tract obstruction. Oligohydramnios due to FGR/uteroplacental insufficiency is usually of a less severe degree and less commonly causes limb and lung problems.

Polyhydramnios

Polyhydramnios is the term given to an excess of amniotic fluid (i.e. AFI >95th centile for gestation on ultrasound estimation). It may present as severe abdominal swelling and discomfort. On examination, the abdomen will appear distended out of proportion to the woman's gestation (increased SFH). Furthermore, the abdomen may be tense and tender and the fetal poles will be hard to palpate. The condition may be caused by maternal, placental or fetal conditions.

Causes of polyhdramnios

- **Maternal:**
- Diabetes.
  Placental:
  Chorioangioma.
  Arteriovenous fistula.
  Fetal:
  Multiple gestation (in monochorionic twins it may be twin-to-twin transfusion
  syndrome).
  Idiopathic.
  Esophageal atresia/tracheoesophageal fistula.
  Duodenal atresia.
  Neuromuscular fetal condition (preventing swallowing).
  Anencephaly.

The management of polyhydramnios is directed towards establishing the cause (and hence determining fetal prognosis), relieving the discomfort of the mother (if necessary by amniodrainage) and assessing the risk of preterm labour due to uterine overdistension.

Polyhydramnios due to maternal diabetes needs urgent investigation, as it often suggests high maternal blood glucose levels. In this context, polyhydramnios should correct itself when the mother's glycaemic control is optimized. Twin-to-twin transfusion syndrome is a rare cause of acute polyhydramnios in the recipient sac of monochorionic twins. It is associated with oligohydramnios and a small baby in the other sac. The condition may be rapidly fatal for both twins; amniodrainage and removal by laser of the placental vascular

THANK YOU