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PREMATURE LABOUR

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Abstract

Premature labour is also called preterm labour. Labour that begins before 37 weeks gestation is considered preterm. Risk factors include premature rupture of membranes, uterine abnormalities, infection, cervical incompetence, previous preterm birth, multifetal pregnancy and foetal or placental abnormalities. Management typically antibiotics, tocolytics and corticosteroids. Bed rest and hydration are commonly used initially. The earlier a baby is born, they have more higher risk of certain health problems including lung problems, autism, hearing loss and intellectual disabilities.

Key words:

Decidual haemorrhage, Birth asphyxia, Persistent pulmonary hypertension and Meconium aspiration syndrome

INTRODUCTION

Premature labour occurs when regular contractions result in the opening of your cervix after week 20 and before week 37 of pregnancy.

Premature labour can result in premature birth. The earlier premature birth happens, the greater the health risks for your baby. Many premature babies (preemies) need special care in the neonatal intensive care unit. Preemies can also have long-term mental and physical problem autism, cerebral palsy, lung problems, hearing loss and intellectual disabilities.



Fig 1: Preterm baby in NICU

GENERAL CONCEPTS ABOUT PREMATURE LABOUR

Premature labour (PTL) is defined as cervical changes and regular uterine contractions occurring between 20 and 37 weeks of pregnancy. Many patients present with premature contractions, but only those who demonstrate changes in the cervix are diagnosed with premature labour (ACOG,2001).

INCIDENCE:

According to the Global Action report released by the partnership for Maternal, New-born & Child health and the World Health Organization, India tops the list of 10 Nations contributing 60% of the world's premature deliveries- with the maximum number of preterm births with 3,519,100 of them, almost 24% of the total number.

ETIOLOGY AND RISK FACTORS

The defining physiological mechanism that triggers the onset of premature labour is largely unknown but many include decidual haemorrhage (abruption), mechanical factors (uterine over distention or cervical incompetence), hormonal changes (perhaps mediated by foetal or maternal stress) and bacterial infections (ACOG,2001). However, a number of risk factors have been associated with premature labour.

Preterm babies are often associated with Intrauterine growth restriction.

Intrauterine growth restriction:

The term intrauterine growth restriction (IUGR) generally is reserved for infants who are at less than the 10th percentile at birth on standard graphs in weight, length and circumference. IUGR is classified.

1. Symmetric intrauterine growth restriction:

The term symmetric intrauterine growth retardation (IUGR) is used when the measurements of the head, weight, and length are less than the 10th percentile. Symmetric IUGR implies that the cause of the growth restriction occurred early in pregnancy and was genetic or intrinsic in nature. Symmetric IUGR is more likely to result from an intrinsic cause, that is, something that affects the foetus from within and starts early in gestation. A poor prognosis is associated with major chromosomal disorders and congenital infection.

2. Asymmetric intrauterine growth restriction:

The term asymmetric IUGR is used when the measurements of the head circumference and length are in a higher percentile than is the measurement for weight. This growth pattern occurs later in pregnancy than does symmetric IUGR and may be caused by placental insufficiency, maternal malnutrition, or other extrinsic factors. Extrinsic factors are those that affect the foetus from the outside, such as maternal hypertension and low caloric intake. Outcomes are better in infants who have asymmetric IUGR compared with those who have symmetric IUGR. Asymmetric IUGR is more likely to be caused by extrinsic factors than is symmetric IUGR.

Factors associated with foetal growth restriction:

Several factors may affect foetal growth, including foetal, maternal, and placental factors. Some of these factors may cause Intra Uterine Growth Restriction. Foetal growth restriction (FGR) may affect perinatal mortality and the infant's short and long-term morbidity.

1. Fetal factors:

Infants with chromosomal anomalies such as trisomy 13 (patau syndrome), trisomy 18 (Edward syndrome), or trisomy 21(Down syndrome) may be SGA, Congenital malformations such as anencephaly, gastrointestinal (GI) atresia, renal agenesis, and some cardiovascular defects may be associated with FGR. Congenital infection may be a cause of infants being SGA. Inborn errors of metabolism, such as transient neonatal diabetes, galactosemia, and phenylketonuria, also are associated with small foetal size.

2. Maternal factors:

Maternal factors include maternal hypoxemia due to sickle cell disease, respiratory disease, cardiovascular disease, or living in a high-altitude environment. Other maternal factors include short stature, young maternal age, low socio-economic status, primiparity, grand multiparity, and low pregnancy weight. Maternal exposure to teratogenic agents, such as alcohol, cigarette smoke, and anticonvulsant medications also may be implicated in the cause of FGR.

3. Placental factors:

Placental insufficiency is the leading cause of infants who are SGA because of the delivery of inadequate nutrients for appropriate foetal growth. Other physical attributes of the placenta and placental circulation also may affect foetal growth. Multiple infarcts, aberrant cord insertions, and small placental size

Diagnostic evaluation:

The diagnosis of preterm labour can be incredibly challenging since many of the symptoms are subtle and common during pregnancy. For example, women experiencing preterm labour may complain of backache, pelvic aching, menstrual-like cramps, increased vaginal discharge, pelvic pressure, urinary frequency, and intestinal cramping with or without diarrhoea.

A diagnosis of preterm labour is made when the following criteria are met - A gestation of 20 to 37 weeks, Documentation persistent uterine contractions (4 every 20 minutes or 8 in 1 hour), Documented cervical effacement of 80% or greater and Cervical dilation of more than 0.4 inch (1cm) or a documented change in dilation.

Infection has been implicated as a contributing factor in premature labour. Prostaglandin production by the amnion, chorion, and decidua is stimulated by cytokines (extracellular factors) that are released by activated macrophages. Group B streptococci, chlamydia, and gonorrhoea have been associated with premature labour and premature rupture of the membranes (Cunninghametal., 2005). It is always prudent for the nurse to obtain a clean-catch, mid-stream, or catheterized urine specimen to identify and treat infection if the patient presents with signs of premature labour or premature rupture of the membranes.



Fig 2: Foetal Monitoring

Biochemical Markers:

Early predictive factors for premature labour and birth include Foetal fibronectin (fFN) and salivary estriol fibronectins are proteins produced by the foetal membranes. They are normally secreted in the cervicovaginal fluid until 20 weeks of gestation. Foetal fibronectins are best described as the "glue" that attaches the foetal membranes to the underlying uterine decidua. Their presence between 24 and 34 weeks in a patient with intact membranes suggests an increased risk for premature labour in both symptomatic and asymptomatic women(Bernhardt &Dorman, 2004). The fFN is retrieved with a sterile cotton-tipped swab placed in the posterior vaginal fornix or in the ectocervical region of the external cervical os for a maximum of 10 seconds. The collection swab is then removed, placed into a manufacturer-supplied medium, and sent to a laboratory that performs the test. Results are reported in 24 to 48 hours. For women, whose foetal fibronectin test is negative (e.g., No fFN is detected) the likelihood of giving birth in the following week is less than 1%(Iams&creasy,2004).

Observational studies have shown that maternal levels of serum oestradiol and salivary estriol increase before the spontaneous onset of term and premature labour (PTL). Salivary estriol (a biochemical marker under study for its predictive value in premature labour) is an estragon produced by foetal trophoblasts. The estriol is derived from adrenal and liver steroid precursors and then converted into estriol in the placenta. Levels of estriol present in maternal saliva has been shown to increase just before premature labour begins. However, this test has not proven to be highly effective since maternal estriol levels normally peak at night. The negative predictive value of both tests is high (95% for fFN; 98% for Salivary estriol). Therefore, the tests are better indicators of who will not experience PTL(ACIG,2001;Bernhardt& Dorman, 2004).

Management:

The gestation of the pregnancy in premature labour influences the management. Generally, the earlier the gestational age the higher is the possibility of an infective cause, which is often followed by rapid labour and delivery. Caesarean section of cephalic premature infants offers no reduction on foetal morbidity or trauma and is associated with its own morbidity. It is generally accepted that the mode of delivery of gestations less than 26 weeks does not alter the outcome. Prolonging pregnancies beyond 34 weeks does not improve neonatal outcomes; therefore, no attempt is usually made to arrest labour if pregnancy has advanced to 34 weeks' gestation.

Skilled care is required for the woman and the foetus during labour. The mother is faced with an unexpected emotional crisis because of the interruption of the normal progress of pregnancy. In extreme prematurity (22-25 weeks) a high perinatal mortality rate means the woman and her partner have to face the possibility of the death or disability of their baby. Full discussion regarding possible outcomes and whether to attempt resuscitation should be carried out with the senior clinicians involved in the care, and of course the parents. Continuous electronic heart rate monitoring is difficult to interpret at less than 30 weeks' gestation and should therefore be used and interrupted with caution. Baseline variability may be reduced on the CTG must also be dated and signed. The purpose of records is to serve the interest of the woman, to demonstrate the chronology of events as well as all significant consultations, assessments, observations, decisions, interventions and outcomes.

A written individualised care plan should be recorded in labour following examination and consultation with the woman. This should attempt to follow the birth plan, which was devised in pregnancy. If the woman changes her mind as her labour progresses, or the situation changes, adjustments can be made. Whether or not a formal birth plan has been prepared, the midwife who is caring for the woman should communicate effectively with her, evaluate whether the labour is proceeding as expected and listen to her requests. A comprehensive record of the discussions that take place about changes in the plan or about proposed measures will ensure that the closest possible attention is paid to achieving the outcome that the parents are hoping for and will also provide an excellent documented history of the labour and improve communication.

Care of Mother:

Once the infant has been determined to be SGA, steps may be taken for anticipate problems and provide early interventions. Complications may include prematurity, birth asphyxia or birth depression, thermal instability, metabolic imbalances, and hematologic concerns. Birth depression encompasses infants who have low heart rate (HR) at birth. These infants may require intervention from caregivers to establish adequate HR and respirations.

At birth the infant is at risk for hypoxia from perinatal asphyxia, persistent pulmonary hypertension, and meconium aspiration syndrome. Perinatal asphyxia is metabolic acidosis at birth associated with Apgar scores of 3 or less that persists after 5 minutes, multisystem organ dysfunction, and neurologic manifestations. Persistent pulmonary hypertension in the new born (PPHN) is a condition in which abnormally elevated vascular pressure result in continuation of flow through foetal blood pathways such as the ductus arteriosus and foramen ovule. These infants should be delivered in a location where immediate access to resuscitation equipment and personnel who are expert in the resuscitation of high-risk new-borns are available.

After birth, the infant should be stabilized and complications such as hypothermia, hypoglycaemia, hypocalcaemia, polycythaemia, and hyper viscosity should be anticipated screened for and corrected early. The infant who is SGA should be observed for respiratory distress and care should be rendered as needed. Prevention of heat loss is essential. Blood glucose levels should be checked, and hypoglycaemia corrected promptly. A central haematocrit measurement also is important.

A physical examination should be performed to screen for congenital anomalies. Screening for congenital infection also may be indicated. Families need to be kept informed the infant's condition and necessary referrals.

Outcome and Follow up:

The mortality risk is significantly higher for the infant who is SGA compared with the infant who is appropriate for gestational age. The infant who is SGA shows more gross motor and minor neurologic dysfunctions over time. Cognitive function also is lower for the infant who is SGA than for the infant who is appropriate for gestational age (Figueras et al.2009; Winchester et al.2009). The infant's outcome is determined by the cause of the growth restriction.

Complications:

Infants who are SGA are at risk for a while range of problems. Knowing that an infant is SGA can assist the nurse to anticipate, prevent, or provide early intervention for problems. To determine whether an infant is at risk for the complications of the SGA infant the gestational age must be determined. The complications of preterm labour includes Anaemia, Breathing problems, Infections or neonatal sepsis, Interventricular haemorrhage New born jaundice, Necrotizing enterocolitis, Patent ductus arteriosus, Retinopathy of prematurity

Summary:

Premature labour (PTL) is defined as cervical changes and regular uterine contractions occurring between 20 and 37 weeks of pregnancy. At birth the infant is at risk for hypoxia from perinatal asphyxia, persistent pulmonary hypertension, and meconium aspiration syndrome. Placental insufficiency is the leading cause of infants who are SGA because of the delivery of inadequate nutrients for appropriate foetal growth. Maternal factors include maternal hypoxemia due to sickle cell disease, respiratory disease. Infants with chromosomal anomalies such as trisomy 13(patau syndrome), trisomy 18(Edward syndrome), or trisomy 21(Down syndrome) may be SGA. The term intrauterine growth restriction (IUGR) generally is reserved for infants who are at less than the 10th percentile at birth on standard graphs in weight, length and circumference.

Conclusion:

Preterm labour can be prevented by proper pre-natal care, control and monitoring diabetes and hypertension, intake of balanced diet with food containing iron and folic acid. washing hands frequently to prevent infection and reduces stress in our daily life by adopting relaxation techniques. Smoking cessation and adopting healthy lifestyle is the important strategies to prevent the occurrences or preterm labour.

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