POLYHYDRAMNIOS IN SINGLETON GESTATION

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1. INTRODUCTION

The measurement of the amount of amniotic fluid (AF) can be performed using different techniques, the two most commonly used are the deepest vertical pocket (DVP) and the amniotic fluid index (AFI).

Deepest vertical pocket: this is performed by measuring the maximum vertical column of fluid free of fetal parts and umbilical cord in a vertical plane. It is considered normal between 2 to 8 cm.

Amniotic fluid index (AFI): this is the value obtained from the sum of maximum vertical columns of fluid, free of fetal parts or umbilical cord, in each of four quadrants delimited by the intersection of two perpendicular lines on the maternal abdomen: longitudinal midline with longitudinal transverse midline between pubic symphysis and fundus. The transducer is placed in a sagittal plane and as perpendicular as possible to the floor. AFI values between 5 and 25 cm are considered normal. It is feasible to perform it from 24 weeks of gestation.

There is controversy about which is the best method for assessing the amount of AF. DVP detects normality, while the use of AFI more frequently diagnoses oligohydramnios and polyhydramnios. However, higher detection has not been shown to correlate with better perinatal outcomes. Therefore, DVP should be used as a screening method. In those cases where DVP ≥ 8 cm is detected, AFI should be measured to confirm polyhydramnios and to assess its severity.

Polyhydramnios affects 1-2% of all pregnancies and has been associated with a variety of adverse pregnancy outcomes including preterm premature rupture of membranes (PPROM), preterm birth (PTB), cord prolapse, and admission to the neonatal unit. Maternal complications include an increased risk of cesarean sections and postpartum haemorrhage. The perinatal morbidity and mortality are higher if polyhydramnios develops early in gestation

2. DIAGNOSIS

The diagnosis of polyhydramnios should be established when DVP ≥ 8 cm and AFI ≥ 25 cm are detected.

Three levels of severity are defined:

- AFI 25-29 cm: mild polyhydramnios.
- AFI 30-34 cm: moderate polyhydramnios
- AFI ≥35 cm: severe polyhydramnios

3. AETIOLOGY

Polyhydramnios is caused by an imbalance between the inflow and outflow of amniotic. About 50-60% of polyhydramnios are idiopathic.

We can define three groups of causes of polyhydramnios:

- Fetal causes: 30% are associated with a fetal anomaly. Multiple fetal pathologies have been described in relation to polyhydramnios, with gastrointestinal alterations being the most frequent malformations (table 1). The risk of presenting a fetal anomaly increases significantly as the amount of AF increases, being present in up to 80% of cases of severe polyhydramnios.

- Maternal causes: some maternal morbidities have been described as possible causes of polyhydramnios: poor metabolic control in diabetes (mainly type 1) is responsible for up to 25% of polyhydramnios; Rhesus and other blood group isoimmunisation leading to immune hydrops; and, less frequently, drug exposure, such as lithium leading to gestational diabetes insipidus.

- Placental causes: Some placental abnormalities are accompanied by an increase in the amount of amniotic fluid (e.g. chorioangioma).

4. STUDY PROTOCOL

1. Perform TTOG to rule out diabetes

2. Request Coombs test and irregular antibody screening to rule out isoimmunisation. To assess anaemia, measurement of peak systolic velocity (PSV) in the middle cerebral artery (MCA) should be used initially.

3. Ruling out infectious causes:
    a. Request maternal serology of B19 parvovirus IgG and IgM.
    b. Request Toxoplasma IgG, if the patient is not immune and was not tested in the last 6 weeks.
    c. Request Lues serology test (Treponemal IgG ELISA) in pregnant women who do not have a test during gestation or who are considered to be at epidemiological risk.
    d. Other serologies should not be requested in the absence of ultrasound markers

4. Ruling out malformative pathology: perform a complete anatomical ultrasound scan. Fetal attitude and the presence of movements should also be assessed in an ultrasound scan.

5. Genetic assessment: the risk of aneuploidy depends on the associated anomalies. In apparently idiopathic polyhydramnios, the risk of aneuploidy is 0.2-1%, being higher in more severe cases without macrosomia. Karyotyping (primarily by a genetic array) is indicated in cases of severe polyhydramnios (AFI ≥ 35) of unknown aetiology and moderate cases (AFI ≥30) with other markers (including estimated fetal weight below the 3rd percentile). When an amnioreduction is performed as per clinical indication, a QF-PCR is recommended.

5. FOLLOW-UP

Polyhydramnios should be monitored according to its severity.

- AFI 18-24 cm (high limit of NORMALITY)
    - Rule out structural abnormalities
    - Follow-up in two weeks to assess progression. If it is stable, routine obstetric monitoring is recommended.
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- AFI 25-29 cm
    - Perform the previously described study (section 4)
    - Measurement of cervical length
    - In one week, follow-up the results and assess the progression of polyhydramnios. If it is stable, control every 2 weeks.

- AFI ≥30 cm
    - Previously perform the described study (section 4)
    - Measurement of cervical length
    - Weekly follow-up

6. TREATMENT

Treatment of polyhydramnios may be aetiological or symptomatic:

Aetiological: when a potentially treatable cause is identified in utero.
Symptomatic: when there is no definite cause of polyhydramnios or it is not treatable in utero, symptomatic treatment should be considered:

6.1. AMNIOREDUCTION

Amnioreduction is a symptomatic treatment to reduce maternal symptoms and decrease the risk of preterm labour. It is only recommended before 35 weeks' gestation.

Recommendations on when to perform amnioreduction are as follows:

- Moderate/severe polyhydramnios (AFI ≥ 30) and cervical length less than 15 mm or clinical maternal uterine contractions.
- Significant maternal discomfort (sensation of dyspnoea or clinical uterine contractions) regardless of severity of polyhydramnios.

6.1.1. Amnioreduction technique

- Placement of a peripheral venous line.
- Check out maternal serological status of vertically-transmissible disease (HIV, HBV and HCV).
- In the presence of clinical uterine contractions before, during or after the procedure, tocolytic treatment with nifedipine at usual doses should be administered for at least 24 hours.
- Fetal lung maturation is recommended as per protocol.
- Administration of prophylactic antibiotics: Cefoxitin 2 g IV 1-2 hours before (if allergy to beta-lactam: Erythromycin 500 mg or Vancomycin 1 g IV).
- The patient is placed in a semi-lateral decubitus position (to avoid maternal hypotension) and always under direct ultrasound vision during the whole procedure.
- Identify the deepest vertical pocket of amniotic fluid, avoiding the fundal area due to the risk of dislocation when the uterus drops.
- Skin antisepsis and draping.
- Use an 18 Gauge needle and an aspiration system (vacuum) at the maximum speed allowed by the needle gauge.
- Removal of fluid until the AFI is left < 20 cm.
- After 26 weeks, perform a CTG 1 hour post-procedure.

6.1.2. Amniotic fluid work-up

- QF-PCR (or other genetic tests if indicated as discussed above)
- Study of infections if maternal IgG positive (PCR of toxoplasmosis and B19 PV).
- If suspected, consider measuring the amniotic fluid levels of chlorine to rule out tubulopathy (Bartter Syndrome).
- Consider a genetic study of myotonic dystrophy (Steinert disease) if suspected by reduced fetal movements or maternal phenotype.
- Additional amniotic fluid should be stored for possible further studies

6.2. PROSTAGLANDIN INHIBITORS

Prostaglandin inhibitors reduce the fetal glomerular filtration rate (secondarily decreasing the amount of fetal urine production) and favour pulmonary reabsorption and the passage of amniotic fluid through the membranes. However, these drugs have important fetal side effects, among which are the premature closure of the ductus arteriosus (most clinically relevantly from 32 weeks onwards). Therefore, its use is restricted as a second-line treatment.

Indomethacin is the drug of choice at a dose of 50 mg/8-12 hours for a maximum period of 5-7 days.

Before 32 weeks, its use should be individualised and it should be administrated under strict echocardiographic monitoring in 24-48 hours for early detection of a possible restriction of ductus arteriosus (DA PI<1 or significant tricuspid regurgitation [>200 cm/s]). If suspected, treatment should be discontinued.

At ≥ 32 weeks, prostaglandin inhibitors are not indicated.

7. DELIVERY

Timing of delivery depends on the aetiology of the polyhydramnios and its associated symptoms:

In idiopathic polyhydramnios, the degree of polyhydramnios has not been associated with an increased risk of adverse perinatal outcomes, and, therefore, the general recommendations are:

- Symptomatic: from 37 weeks' gestation onwards.
- Asymptomatic: from 40 weeks' gestation onwards.

Table 1. Fetal causes of polyhydramnios

MOST COMMON FETAL CAUSES OF POLYHYDRAMNIOS
• **Gastrointestinal disorders:** omphalocele, atresia (oesophagus, ileum, jejunum), gastroschisis • **CNS disorders:** neural tube defects • **Infectious causes:** B19 Parvovirus, CMV, TXP, Lues • **Congenital heart diseases**: arrhythmias, truncus, tricuspid dysplasia, aortic coarctation (Ao Co) • **Thoracic disorders:** cystic adenomatoid malformation , pulmonary sequestration, congenital diaphragmatic hernia (**CDH**), chylothorax • **Renal disorders:** renal tubulopathies • **Skeletal disorders:** achondroplasia, thanatophoric dysplasia • **Neuromuscular disorders:** myotonic dystrophy, arthrogryposis • **Metabolic disorders:** gangliosidosis, Gaucher disease • **Genetic diseases:** 18T, 21T, Turner syndrome, 22q11.2 deletion syndrome, Noonan syndrome, VACTERL/VATER association, Williams Syndrome • **Fetal tumours**: sacrococcygeal teratoma