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Oligohydramnios: A review of etiology and management options

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Abstract

Oligohydramnios is both a consequence of fetal malformations and of uteroplacental insufficiency. Its existence is associated with a high rate of both antepartum and intrapartum complications. It is vital that its occurrence is detected as early as possible so that we can manage it correctly. The main causes of its occurrence are identified and described in this review. The management of oligohydramnios is most often expectant, the timing of delivery also being determined by Doppler examination and changes in parameters measuring fetal growth and development.

Keywords: oligohydramnios, Potter syndrome, pulmonary hypoplasia, polycystic kidney, multicystic renal dysplasia, posterior urethral valve, urethral atresia, amnioinfusion

INTRODUCTION

Amniotic fluid, with a composition similar to extracellular fluid, containing 98% water, is present in the amniotic cavity since the beginning of pregnancy, when the main mechanisms of production are represented by: intramembranous pathway, transmembranous pathway and through the fetal integument. The intramembranous pathway is the one by which the exchange of water and substances is carried out through the fetal vessels at the surface of the placenta, the process being favored by the osmotic gradient given by the hypotonicity of the fluid (260 mOsm / l), water is transferred by passive mechanism and proteins and soluble substances by active mechanism. The transmembranarous pathway represents the transport of water and soluble substances between the amniotic and chorionic membranes. The fetal integument plays a role in the production of amniotic fluid until 22 to 25 weeks, when tissue permeability decreases and skin keratinization begins [1].

From the second half of pregnancy, the main mechanisms of amniotic fluid production and circulation are diuresis and fetal swallowing. Urine production starts from 8 to 11 weeks and becomes the main mechanism from the second trimester onward, as the fetus is able to respond to changes in the amount of fluid in the amniotic cavity through changes in diuresis. The production and elimination of pulmonary secretions is another mechanism present from the second half of pregnancy, and the swallowing that occurs from weeks 8 to 11 keeps the volume of fluid constant, the amount swallowed at term being 200 to 800 ml/day [2].

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The role of amniotic fluid is multiple, it contributes to the normal development of the lungs through breathing, of the gastrointestinal tract through swallowing, allows free movement for normal musculoskeletal development, protects against umbilical cord compression, provides a source of hydration and mechanical protection [3].

MATERIALS AND METHODS

PubMed, NCBI and Medical Journals were searched for studies written in English was performed using keywords like "oligohydramnios", "Potter syndrome", "pulmonary hypoplasia", "polycystic kidney disease", "multicystic renal dysplasia", "posterior urethral valve", "urethral atresia" and "amnioinfusion". The aim of this article was to briefly highlight up to date data regarding the etiology and the management of oligohydramnios, as it may associate various and serious fetal anomalies, as well as increased risk for fetal and maternal perinatal outcome.

Following the systematic research, we found approximately 764 articles, which included randomized controlled trials, review articles, systematic reviews and meta-analyses. In our review we chose to include 30 articles that we found to be most relevant to the subject.

RESULTS

Definition of oligohydramnios

The normal amount of amniotic fluid between weeks 22-39 is considered to be 750 ml, and the percentiles used as threshold are: the 5th percentile corresponding to 300 ml and the 95th percentile corresponding to 2000 ml [4]. Oligohydramnios is defined as a decrease in the amount of amniotic fluid below the 5th percentile for gestational age [5].

To correctly define normal amniotic fluid volume and oligohydramnios, accurate ultrasound measurement is required. Currently three methods are used with 2 being objective: AFI (amniotic fluid index), MVP (maximal vertical pocket) and the estimation method, which is subjective and requires experience from the operator. The AFI method measures the amniotic fluid index by dividing the gravid uterus into four quadrants and measuring the maximum vertical diameter in each quadrant, the resulting sum being in centimeters. A normal AFI is defined by values between 5 and 24 cm [6]. Maximum vertical pocket is the measurement of the largest vertical diameter in the sagittal plane. Normal values are between 2 and 8 cm [5]. Ultrasonographical, oligohydramnios is defined as AFI <= 5, or MVP <= 2 and occurs in 1 to 2% of pregnancies [7,8].

Etiology

Depending on the time of pregnancy when it is discovered, the causes of oligohydramnios can be: fetal structural abnormalities, utero-placental insufficiency, intrauterine growth restriction, premature rupture of membranes, prolonged pregnancy, drugs [8].

Fetal malformations

Bilateral renal agenesis with a frequency of 1 in 8000 births leads to absence of amniotic fluid, called anhydramnios, and to the development of Potter syndrome characterized by pulmonary hypoplasia, limb contracture, facial abnormalities such as small chin, low-set ears, flattened nose [9]. Somatic malformations are due to lack of space for normal movements and pulmonary hypoplasia occurs due to low pulmonary fluid pressure and fetal respiratory movement deficit. When these abnormalities occur on the basis of another malformation that produces oligohydramnios, it is called the Potter sequence. The prognosis is poor, with death occurring from pulmonary hypoplasia [10].

Bilateral multicystic renal dysplasia is characterized by the transformation of the kidney into a mass of large microcysts or cysts. It is associated with severe oligohydramnios from early pregnancy and Potter's sequence with poor prognosis [11].

Autosomal recessive polycystic kidney disease with a frequency of 1 in 20,000 births is characterized by transformation of the renal collecting ducts into 1-2 mm cysts and renal cortical necrosis. It is manifested by renal failure with oligohydramnios, liver fibrosis and poor prognosis and increased mortality rate due to pulmonary hypoplasia [12,13].

Posterior urethral valve in male children is the most common distal obstruction of the urinary tract, characterized by the presence of membranes at the posterior part of the urethra that prevent bladder emptying, leading to dilated and hypertrophied bladder, urethral dilatation, ureteral dilatation and renal pelvis. The unfavorable prognosis is present in cases with oligohydramnios occurring before mid-pregnancy, or those associated with aneuploidy or renal pelvic dilatation [14,15]. Urethral atresia is more common in females with complete obstruction and poor prognosis [16].

Cloacal malformations are rare 1 in 50,000 births, more common in females, are characterized by common opening of the genitourinary apparatus and gastrointestinal tract with association of multiple other malformations renal agenesis, bladder duplication, neural tube anomalies), and oligohydramnios is frequently present [17].

Utero-placental insufficiency can cause oligohydramnios by hypoxemia which redistributes blood flow to vital territories (brain, heart), with decreased renal perfusion and decreased amount of urine formed [18].

Different drugs can also lead to oligohydramnios. Treatment of the mother with renin-angiotensin system blockers such as angiotensin-converting enzyme inhibitors and angiotensin-receptor blockers, as well as non-steroidal anti-inflammatory drugs such as indomethacin produce oligohydramnios (by decreasing renal blood flow), renal failure, anuria, intrauterine death, pulmonary hypoplasia, persistent patent ductus arteriosus, cerebral complications [19,20].

Consequences of oligohydramnios

Pulmonary hypoplasia is defined as a reduction in the number of alveoli, with oligohydramnios being the main cause, especially when it occurs before 20 to 22 weeks [21].

Three possible mechanisms are proposed: thoracic compression, inhibition of fetal breathing motion, and loss of lung fluid.

In oligohydramnios there is extrinsic thoracic compression through the uterine wall which constricts the fetal thoracic cavity, lack of fetal breathing movement with limit long expansion and loss of lung fluid through decreased intra-amniotic pressure and leakage of lung fluid into the amniotic cavity [22].

In most cases it is lethal, and survivors develop pulmonary hypertension, emphysema, pneumothorax, decreased chest circumference [23].

Prognosis

Oligohydramnios is associated with an increased incidence of intrapartum complications. Oligohydramnios pregnancies have an increased risk of preterm labor, intrauterine growth restriction, preterm delivery, need for cesarean delivery, Apgar score < 7 at 5 minutes, umbilical arterial Ph < 7. Incidence of umbilical arterial Ph < 7 is 1.5 times higher in girls with oligohydramnios [24].

Cases of premature placental separation have also been reported, oligohydramnios being present based on placental dysfunction [25]. Caesarean section for abnormal heart rhythm occurs in about 35% of cases (mercer) [26]. Several studies report poor perinatal outcome and increased perinatal mortality [26,27].

Management

Pregnancies detected with oligohydramnios require strict surveillance because of unfavorable prognosis. Early detection of malformations, intrauterine growth restriction, Doppler signs of fetal compromise is necessary. Assessment of fetal growth should be performed every 3-4 weeks, supplemented with color Doppler studies on the umbilical artery, weekly amniotic fluid measurements. In general, conservative treatment with strict surveillance is recommended if fetal parameters and Doppler study are within normal limits, and at term, the presence of oligohydramnios is considered an indication for delivery [28].

Transvaginal amnioinfusion with saline may be used during labor to avoid compression of the umbilical cord and changes in fetal cardiac function. However, study results are conflicting, there are no significant differences in cesarean delivery rates for heart rhythm changes or newborn status between the amnioinfusion and conservative-treated groups. The use of amnioinfusion as a treatment for oligohydramnios is not recommended [29,30].

CONCLUSIONS

Oligohydramnios occurring between the second and third trimester of pregnancy has been associated with increased rates of fetal malformations, most commonly renal, intrapartum complications, and perinatal morbidity. Strict surveillance with serial ultrasound examinations is necessary for early detection of fetal malformations, intrauterine growth restriction, or other factors that may influence prognosis. Treatment is generally conservative, with careful timing of delivery. Caesarean birth occurs quite frequently because of abnormalities in the fetal heart rate during labor.

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