Hydration Therapy in Oligohydramnios

Thesis

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Marwa Mohammed El-Araby

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Introduction

Amniotic fluid serves to cushion the fetus allowing musculo skeletal development and protecting it from trauma. It also maintains temperature and has a minimal nutritive function. Amniotic fluid may promote growth and differentiation of tissues of the lung and gastrointestinal tract through inhalation and ingestion of the amniotic fluid respectively ***(Cunningham et al., 2005).***

Amniotic fluid volume increases steadily from about 250 ml at 16 weeks of gestation to 800 ml at 28 weeks, slowing down in quantity or volume at this time to reach 1000 ml by 34 weeks, after this, there is a decline in amniotic fluid volume to approximately 800 ml at 40 weeks or less postterm ***(Gabbe et al., 1986 and Cunningham et al., 2005).***

Oligohydramnios is one of the prevalent threatening conditions to fetal health. Intrauterine growth restriction, respiratory distress syndrome, post-maturity syndrome and chronic fetal hypoxia are associated with Oligohydramnios. The condition may be responsible for fetal malpresentation, umbilical cord compression, meconium staining, increased prenatal mortality and morbidity, and increased operative delivery ***(Deka, 2001 and Brian, 2000).***

In the first half of pregnancy, amniotic fluid volume appears to increase in association with growth of the fetus, and the correlation between fetal weight and amniotic fluid is very close. The serum osmolality and sodium, urea, and creatinine content of maternal serum and amniotic fluid are not significantly different. This suggests that amniotic fluid is an ultrafiltrate of maternal serum ***(Aghajanian et al., 2007)*.**

Amniotic fluid is composed largely of the fetal urine. It also contains desquamated fetal cells, vernix, lanugo, and various secretions. Pulmonary fluid contributes a small proportion of the amniotic volume, and fluid filtering through the placenta accounts for the rest ***(Cunningham et al., 2005).***

Although there is some question about the mechanisms of the lowered volume of amniotic fluid, if it is due to an altered fetal circulation preferentially to the brain, heart and adrenal glands at the expense of other organs systems or if it is the result of an increased intramembranous uptake of water without a change in fetal urine production, the result is similar, and low amniotic fluid volume is observed with chronic placental insufficiency ***(Gagon et al., 2002).***

Hydration can be considered an effective way of decreasing the chance of Oligohydramnios because maternal dehydration increases the chance of the condition. Therefore, both serum and oral hydration therapies are hypothesized to

be effective treatment for Oligohydramnios. Previous studies have supported this hypothesis. Maternal hydration may theoretically increase amniotic fluid volume by causing fetal diuresis. An effective, non-invasive method of increasing amniotic fluid volume may have several applications in obstetric practice ***(Hofmeyr et al., 2010)***.

Previous studies have demonstrated that maternal hydration is beneficial in treating pregnancies with Oligo-hydramnios ***(Hofmeyr et al., 2010)***.

Aim of the Work

The aim of this study is to find out is there significant increase in amniotic fluid index (AFI) after acute maternal hydration in pregnant women with Oligohydramnios and to evaluate therapeutic effectiveness of two different modalities of hydration therapy (oral and intravenous).

Oligohydramnios

Oligohydramnios refers to amniotic fluid volume that is less than expected for gestational age. It is typically diagnosed by ultrasound examination and may be described qualitatively (eg, normal, reduced) or quantitatively (eg, amniotic fluid index [AFI] <5). Methods of amniotic fluid volume assessment are reviewed separately ***(Locatelli et al., 2004).***

An adequate volume of amniotic fluid is critical to allow normal fetal movement and growth, and to cushion the fetus and umbilical cord. Oligohydramnios may inhibit these processes and can lead to fetal deformation, umbilical cord compression, and death ***(Nicksa et al., 2011).***

Incidence

Reported rates of oligohydramnios are influenced by variations in diagnostic criteria, the population studied (low or high risk, screening or indicated ultrasound examination), the threshold used, and the gestational age at the time of the ultrasound examination (preterm, term, or postterm) ***(Locatelli et al., 2004).***

A study of 3050 uncomplicated pregnancies with singleton non-anomalous fetuses between 40 and 41.6 weeks of gestation noted oligohydramnios (defined as AFI less than 5) in 11 percent ***(Locatelli et al., 2004).*** The incidence is high

in laboring women, largely due to rupture of fetal membranes during or just before labor ***(Sarno et al., 1989).***

Pathophysiology:

The volume of amniotic fluid is ultimately determined by the volume of fluid flowing into and out of the amniotic sac. Fetal urination, lung fluid, and swallowing all make important contributions to fluid movement in late gestation, with minimal contributions from other sources. Fetal disorders that affect any of these processes will affect the amniotic fluid volume. As an example, growth restricted fetuses may redistribute blood flow away from their kidneys, which decreases fetal urine production, resulting in oligohydramnios ***(Yoshimura et al., 1997).***

Homeostatic mechanisms, such as intramembranous absorption (transfer of amniotic fluid across the amnion into the fetal circulation), also exist and work to maintain amniotic fluid volume. These mechanisms appear to be more successful in limiting excess fluid volume than in preventing reduced fluid volume. As an example, only half of fetuses with esophageal atresia, and two-thirds of fetuses with duodenal or proximal jejunal atresia develop polyhydramnios, whereas renal agenesis invariably results in oligohydramnios The physiology of normal amniotic fluid production and volume regulation are discussed separately ***(Underwood et al., 2005).***

Physiology of amniotic fluid volume regulation:

Etiology

Conditions commonly associated with oligo-hydramnios are listed in the table (table 1). The most likely etiologies of oligohydramnios vary according to severity and the trimester in which they are diagnosed. The majority of women with oligohydramnios or borderline/low normal amniotic fluid volume have no identifiable cause ***(Locatelli et al., 2004).***

Table (1): Causes of Oligohydramnios

Maternal
Medical or obstetrical conditions associated with uteroplacental insufficiency (eg, preeclampsia, chronic hypertension, collagen vascular disease, nephropathy, thrombophilia)
Medications (eg, angiotensin converting enzyme inhibitors, prostaglandin synthetase inhibitors, trastuzumab)
Placental
Abruption
Twin to twin transfusion (ie, twin polyhydramnios-oligohydramnios sequence)
Placental thrombosis or infarction
Fetal
Chromosomal abnormalities
Congenital abnormalities, especially those associated with impaired urine production
Growth restriction
Demise
Postterm pregnancy
Ruptured fetal membranes
Idiopathic

(Locatelli et al., 2004).

First trimester — The etiology of first trimester oligohydramnios is often unclear. Reduced amniotic fluid prior to 10 weeks of gestation is rare because gestational sac

fluid is primarily derived from the fetal surface of the placenta, transamniotic flow from the maternal compartment, and secretions from the surface of the body of the embryo.

Criteria suggested for determining reduced amniotic fluid at this gestational age have included a difference between mean gestational sac size (MGSS) and crown-rump length of less than 5 mm or a mean gestational sac diameter/crown-rump length ratio outside the normal range for gestational age. This finding has been associated with poor outcome in selected populations. However, the prognostic value of these findings, when applied to large unselected populations, has not been adequately studied ***(Rowling et al., 1997)***.

Second trimester

By the beginning of the second trimester, fetal urine begins to enter the amniotic sac and the fetus begins to swallow amniotic fluid. Therefore, disorders related to the fetal renal/urinary system begin to play a prominent role in the etiology of oligohydramnios (table 2). Maternal and placental factors, as well as rupture of the fetal membranes, are also common causes of oligohydramnios in the second trimester. The etiologies and relative frequencies of midtrimester oligohydramnios were illustrated in a series of 128 fetuses first noted to have oligohydramnios at 13 to 24

weeks of gestation [12]. The following etiologies were observed: fetal anomaly (51 percent), preterm premature rupture of membranes (PPROM) (34 percent), placental abruption (7 percent), fetal growth restriction (FGR) (5 percent), and unknown (4 percent). Six of the 65 anomalous fetuses were aneuploid. The pregnancy outcome was generally poor due to fetal or neonatal death or pregnancy termination ***(Shipp et al., 1996).***

An elevated maternal serum alpha fetoprotein (MSAFP) concentration has also been linked to second trimester oligohydramnios, with or without an anomalous fetus. This combination (elevated MSAFP, decreased amniotic fluid volume) carries an extremely poor prognosis: fetal growth restriction, fetal death, preterm delivery, neonatal death ***(Los et al., 1994)***.

In one review of these cases, only 8 of 57 (14 percent) children survived past the neonatal period. Oligohydramnios associated with an elevated MSAFP level may be caused by fetal membrane or placental damage, with leakage of amniotic fluid or fetal blood into the maternal circulation ***(Los et al., 1992).***

Second trimester oligohydramnios related to amniocentesis appears to have a better prognosis. The membranes often "reseal" with reaccumulation of amniotic

fluid and normal pregnancy outcome. There are a few reports of the occurrence and outcome of oligohydramnios after chorionic villus sampling ***(Bronshtein et al., 1991).***

Third trimester — Oligohydramnios first diagnosed in the third trimester is often associated with PPROM or with uteroplacental insufficiency due to conditions such as preeclampsia or other maternal vascular diseases. Oligohydramnios frequently accompanies fetal growth restriction related to uteroplacental insufficiency. Fetal anomalies and abruptio placentae also play a role at this gestational age. Amniotic fluid volume normally decreases postterm and oligohydramnios can develop in these pregnancies. In addition, many cases of third trimester oligohydramnios are idiopathic. There may also be an association between pregnancy during the summer season and oligohydramnios, likely related to suboptimal maternal hydration in hot weather ***(Feldman et al., 2009)***.

Mechanisms of isolated oligohydramnios also may include alterations in the expression of water pores (aquaporin 1, aquaporin 3) in fetal membranes and placenta ***(Zhu et al., 2009).***

Clinical manifestations and diagnosis

Oligohydramnios may be first suspected because the uterine size is less than expected for gestational age. Clinical

diagnosis is based on the finding of decreased amniotic fluid on ultrasound examination. Methods for assessment of amniotic fluid volume are discussed in detail separately. There are both objective and subjective ultrasound criteria for oligohydramnios. Although use of an objective criterion is generally preferable (amniotic fluid index ≤5; single deepest pocket <2 cm), subjective suspicion of amniotic fluid volume by experienced examiners has similar sensitivity for diagnosing reduced amniotic fluid volume confirmed by the dye-dilution method, the gold standard for quantifying volume ***(Magann et al., 1997).***

There are no large studies in women with oligohydramnios comparing the various sonographic methods of amniotic fluid volume assessment against the dye dilution method, and the latter is not useful clinically because it is invasive and time-consuming. Small series have found that objective sonographic measurements often grossly under- or over-estimated oligohydramnios ***(Magann et al., 1999).***

In the only study comparing sonographic and magnetic resonance imaging assessment of amniotic fluid volume, both modalities had similar, and relatively poor, efficacy for the detection of oligohydramnios ***(Zaretsky et al., 2004).***