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Amniotic fluid volume

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Abstract. After a brief introduction concerning the physiology of amniotic fluid circulation, the different methods of volume estimation (eyeballing, maximum vertical depth, amniotic fluid index) are discussed. We conclude with the work-up in cases of oligo- and polyhydramnios.

Keywords: Amniotic fluid; Amniotic fluid index (AFI); Oligohydramnios; Polyhydramnios; Fetal surveillance

1. Introduction

In a few decades, evaluating amniotic fluid volume and its determinants has evolved from a topic of academic interest in feto-placental physiology [1] to an important tool for clinical fetal surveillance [2,3].

After the first trimester, amniotic fluid volume is nearly exclusively determined by fetal factors, except for the clinically identifiable cases where oligo- or anhydramnios are due to ruptured membranes.

Polyhydramnios may accompany suboptimally controlled diabetes with macrosomia, a number of conditions leading to non-immune hydrops, and a great range of fetal malformation, from esophageal atresia to chromosomal anomalies and skeletal dysplasias. Of particular ominous significance in these cases, is the combination of (symmetric) fetal growth restriction and hydramnios. Asymmetric amniotic sac volume, culminating in polyhydramnios with one twin and anhydramnios (stuck twin) in the other, is a major feature of the twin-to-twin transfusion syndrome (TTTS).

Of particular interest, however, is the need to identify oligohydramnios and monitoring its progress in cases of impaired fetal growth. Bashat [4] states that “amniotic fluid volume

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is determined by the effects of hypoxaemia and vascular status on renal perfusion and therefore on fetal urine production. Progressive deterioration of acid–base status and vascular status is accompanied by a progressive decline in amniotic fluid volume." Amniotic fluid monitoring in itself or as part of the fetal biophysical profile score is therefore of paramount importance [5].

2. Amniotic fluid physiology—a brief recapitulation

2.1. Functions of amniotic fluid

Amniotic fluid is the medium that creates space for the fetus. This may provide passive protection cushioning the fetus from trauma, but at least as important is its role in providing room for active movement and growth. Lack of amniotic fluid stunts growth and leads to fetal deformation, leading to facial deformities, lack of the normal rotation of the ears (Potter facies) as well as limb deformities [6,7]. Lack of amniotic fluid also hinders thorax expansion and respiratory movement, leading to lung hypoplasia and fibrosis causing neonatal respiratory difficulties and eventually death in outspoken cases.

Amniotic fluid also provides an environment of stable temperature for the fetus. It has certain antibacterial properties which to a certain extent protect the fetus from infection. Finally, while the notion that amniotic fluid serves as a source of nutrients for the fetus has long been discarded, it has regained favor and interest recently.

2.2. The origins of amniotic fluid

The source of amniotic fluid changes throughout pregnancy, as is indicated by the fact that while in the second and third trimester the fetal kidney is the main source of amniotic fluid. A fluid filled amniotic cavity is seen before the fetus has functional kidneys, and also in the presence a stunted or blighted embryo. In the first trimester of pregnancy and partly until the definitive fusion of amnion and chorion, amniotic fluid is exsudated from the loose, gel-like connective tissue in the amnio-chorionic space, the magma reticulare. There is active ion transport through the amniotic membrane toward the cavity and water follows the ensuing chemical gradient.

In the second and third trimester the "amniotic fluid cycle" is installed. The main source of production is the fetal kidney, with the fetal lung and the intramembraneous circulation as secondary sources. The fluid is drawn off through fetal deglutition, absorbed from the fetal gastro-intestinal tract and cleared through the placenta.

Equilibrium in the system thus requires the anatomical and functional integrity of the kidneys and urinary outflow tract, deglutition, involving oropharynx and oesophagus, and the gastro-intestinal passage. The cumulative effect of deficiency in the cycle can thus be calculated. In a fetus of 28 weeks with constant deglutition, but producing only 1 ml of urine per hour (less than 3% of normal) this would result in halving the amniotic fluid, reducing it by 800 ml, in 3 weeks [2]. On the other hand a fetus lacking normal deglutition or intestinal passage e.g. with oesophageal or duodenal atresia, may accumulate fluid at the same rate, resulting in quickly expanding polyhydramnios. Lower gastro-intestinal tract atresia (jejunal, anal) may not result in polyhydramnios, however, as residual resorption, capacity proximal of the atresia may be sufficient to dispose of the fluid.