Hydramnios Associated with Foetal Duodenal Atresia

Lt Col Shakti Vardhan*, Brig RC Behra (Retd)[†], Maj Tony Jose[#], Dr Aby Koshy^{**}

MJAFI 2005; 61 : 387-388

Key Words: Hydramnios; Duodenal atresia

Introduction

Hydramnios, an excessive accumulation of amniotic fluid within the amniotic cavity, is not associated with an identifiable cause in the majority of the cases. However, in about one third of the cases an underlying cause is present. Hydramnios may be a manifestation of underlying foetal anomaly in 13%, maternal disorder such as diabetes mellitus in 14% [1] and rarely Rh isoimmunization in less than 1% [2] of cases. A case of severe hydramnios associated with foetal duodenal atresia is presented.

Case Report

A 28 year old second gravida with one spontaneous first trimester abortion was found to have excessive abdominal distension at 29 weeks of gestation. Ultrasound done at 29 weeks, showed presence of duodenal atresia in the foetus with "Double Bubble Appearance" (Fig 1) and an amniotic fluid index AFI of 26cm. Her Haemoglobin was 12gm%, Blood group was B positive, VDRL was non reactor, HbsAg was negative, HIV negative and GTT was normal. She was admitted and given two doses of injection Dexamethasone 25 mg IM

12 hourly. She was put on bed rest and Indomethacin 12mg orally thrice a day. Aim of admission was to prolong the pregnancy to at least 34 weeks. Repeat ultrasound at 32 weeks showed features of non-immune hydrops in the form of pleural effusion and scalp oedema (Fig 2,3). She had an intra uterine foetal death (IUD) the next day and spontaneous premature rupture of membranes (PROM) two days later. Labour was induced with oxytocin drip and she delivered a macerated male baby weighing 1.5kg with no apparent external deformity. She had primary atonic post partum haemorrhage (PPH), which was controlled with massage of the uterus and oxytocics. She was transfused two units of blood. Postmortem studies could not be carried out on the baby because of parents unwillingness.

Discussion

Hydramnios occurs in 3% of all pregnancies [3]. Amniotic fluid index (AFI) is the sum of maximum depth of amniotic fluid in all the four quadrants of the uterus. The diagnosis of hydramnios is confirmed when it exceeds 25cm. It is called as mild between 8-11cm, moderate when 12-15cm and severe when it exceeds 15 cm. Pregnancy with mild hydramnios has an

Fig. 1 : Ultrasound showing double bubble appearance due to foetal duodenal atresia

Fig.2 : Ultrasound showing foetal pleural effusion and chest wall edema

*Associate Professor, **Resident, Department of Obstetrics and Gynaecology, Armed Forces Medical College, Pune. [†]Ex-Commandant, 167 MH, c/o 56 APO, [#]Graded Specialist (Obstetrics and Gynaecology), CH(CC), Lucknow.

Fig. 3 : Ultrasound showing foetal scalp edema

uneventful course. Moderate and severe hydramnios, often has an underlying foetal or maternal factor in more than 90% of the cases and has an unfavourable prognosis. Perinatal mortality is high in these cases, due to congenital anomalies, preterm premature rupture of membranes, preterm delivery and cord prolapse and placental abruption due to sudden decompression following rupture of membranes. Perinatal outcome gets further worsened if intrauterine growth restriction is associated with hydramnios [4]. Postpartum haemorrhage may occur due to uterine atony.

Congenital malformations may be present in 12-13% of the cases, the important ones being neural tube defects like anencephaly where swallowing may be impaired and fluid may ooze out in excess due to exposed meninges; chromosomal abnormalities like Down's syndrome and trisomy 18, oesophageal and duodenal atresia due to gut obstruction, congenital infections like syphilis and toxoplasmosis. Although in cases with hydramnios, it is common to find a foetal malformation, the reverse is often not true. It is seen that not more than 3-3.5% of congenital malformation have associated hydramnios [5]. Maternal diabetes remains cause of hydramnios in upto 13% of the cases [6], the postulated mechanism being maternal hyperglycemia leading to foetal hyperglycemia and polyuria, in turn. In cases with diabetes mellitus, amniotic fluid volume in the later half of pregnancy corresponds to the glycemic control [7]. Rh isoimmunisation in mother, no longer remains the important cause, with its incidence falling dramatically due to the widespread use of injection Anti D.

In the present case the foetus had developed features of non-immune hydrops. Non-immune hydrops is associated with gastrointestinal developmental defects like duodenal atresia and chromosomal anomalies like Turner's syndrome, trisomy 13, 16, 18 and triploidies. The underlying mechanisms responsible for the development of hydrops in such cases are hypoalbuminaemia along with malformation of the cardiovascular and lymphatic systems.

Moderate to severe degrees causing maternal dyspnoea can be offered amniocentesis to relieve maternal distress. Indomethacin is useful in reduction of amniotic fluid volume in dosage of 1.5-3mg per kg per day. It acts by decreasing lung liquid production, increasing fluid absorption by the lungs and reducing foetal urine production. However, the premature closure of foetal ductus arteriosus, remains a major concern, although the risk of ductal constriction is relatively small (about 5%) before 27 weeks of pregnancy, which rises significantly after 32 weeks (about 50%). In addition, use of indomethacin may be associated with increased incidence of intraventricular haemorrhage and necrotising enterocolitis in the infant. A safer alternative is Sulindac (prostaglandin synthetase inhibitor) which less often leads to premature closure of ductus arteriosus.

References

1. Carlson D, Platt L, Medearis A. Quantifiable polyhydramnios: Diagnosis and management. *Obstet Gynecol* 1990; 75: 989-93.
2. Lindon M, Breckle R, Thomas M. Polyhydramnios: Ultrasonically detected prevalence and neonatal outcome. *Obstet Gynecol* 1987; 69: 21.
3. Chamberlain PF, Manning FA, Morrison I, Harman CR, Lange IR. Ultrasound evaluation of amniotic fluid volume: The relationship of increased amniotic fluid volume to perinatal outcome. *Am J Obstet Gynecol* 1984; 150: 250-4.
4. Furman B, Erez O, Senior L, et al. Hydramnios and small for gestational age: Prevalence and clinical significance. *Acta Obstet Gynecol Scand* 2000; 79: 31.
5. Martinez-Frias ML, Bermejo E, Rodriguez - Pirilla E, Frias JL. Maternal and foetal factors related to abnormal amniotic fluid. *J Perinatol* 1999; 19: 514.
6. Fries JK. Polyhydramnios: Ultrasonically detected prevalence and neonatal outcome. *Obstet Gynecol* 1987; 69: 21-5.
7. Bar - Hava I, Scarpelli SA, Barnhard Y, Divon MY. Amniotic fluid volume reflects recent glycemic status in gestational diabetes mellitus. *Am J Obstet Gynecol* 1994; 171: 952.