Rohilla et al., Reprod Syst Sex Disord 2016, 5:3

DOI: 10.4172/2161-038X.1000183

#### **Case Report** Open Access

# Idiopathic Recurrent Polyhydramnios: A Rare Case Report

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Rec date: July 4, 2016; Acc date: July 19, 2016; Pub date: July 26, 2016

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### **Abstract**

Polyhydramnios occurs in 1-2% of pregnancies and may be idiopathic in more than half of the cases. The diagnosis of idiopathic polyhydramnios can be made only in the absence of maternal diabetes, fetal anomalies, aneuploidies, multiple gestation, Rh incompatibility, placental tumours and non-immune hydrops. Rarely, idiopathic polyhydramnios may be recurrent in future pregnancies and sometimes neonatal examination may reveal cause of polyhydramnios. Preterm delivery and associated fetal anomalies are the main concerns with recurrent polyhydramnios. Very few cases of recurrent idiopathic polyhydramnios have been reported in literature with a maximum number of four pregnancies being affected consecutively. We report here a case of idiopathic polyhydramnios which recurred consecutively in three pregnancies with a good neonatal outcome.

Keywords: Polyhyramnious; Recurrent; Idiopathic

### Introduction

Excessive amount of liquor amnii with amniotic fluid index more than 25 cm is known as polyhydramnios, it complicates 1-2% of pregnancies and may be associated with adverse maternal and fetal pregnancy outcomes. Common causes of polyhydramnios are multiple gestation, maternal diabetes, placental tumours, Rh isoimmunisation, fetal infections, fetal structural and chromosomal anomalies. In more than half cases of polyhydramnios, no definitive cause is attributable; these are labelled as Idiopathic polyhydramnios [1]. Some of these cases of idiopathic polyhydramnios may reveal some congenital anomaly or chromosomal abnormality in the postnatal period [2].

Polyhydramnios may be recurrent in pregnancies, though the entity is rarely reported in literature. Common causes of recurrent polyhydramnios are pregestational diabetes mellitus, recurring fetal anomalies e.g. hydrocephalus, non-immmune hydrops or may be idiopathic. The risk of recurrent polyhydramnios has been reported by as 1 in 1720 pregnancies with a perinatal mortality rate of 16.2% [3].

We searched PubMed using the terms "recurrent polyhydramnios". Less than ten cases of recurrent polyhydramnios have been reported in the literature where there was no definite etiology found and only contributory factors like amniotic fluid prolactin levels, fetal Bartter's syndrome and human leukocyte antigen incompatibility have been postulated [3-7]. In comparison to normal pregnancies, perinatal mortality is reported to be two to five fold higher in pregnancies with idiopathic polyhydramnios [1]. The maximum number of pregnancies affected by recurrent polyhydramnios is reported to be four [3,5,7]. We hereby report a case of idiopathic polyhydramnios recurring in three pregnancies consecutively.

### **Case Report**

A 24 year old Gravida 3 with history of polyhydramnios in the previous 2 pregnancies was supervised at antenatal clinic since early gestation. First pregnancy resulted in previable pPROM (preterm premature rupture of membranes) and had abortion at 24 weeks period of gestation (POG). This was an unsupervised pregnancy with history of early onset polyhydramnios at around 20 weeks POG. In the second pregnancy, polyhydramnios was detected at 22 weeks. Glucose tolerance test (GTT), TORCH serology, anomaly scan (level II) and fetal echocardiograpgy were normal. Her respiratory discomfort due to polyhydramnios necessitised administration of indomethacin and therapeutic amnioreduction thrice during her antenatal period at 295/7, 303/7 and 312/7 weeks. Amniotic fluid was sent for karyotype and was reported normal. She had a placental abruption followed by preterm delivery at 32 weeks POG which also culminated in and atonic postpartum hemorrhage requiring blood transfusion. Gross morphology and histopathological examination of placenta was normal. She delivered a baby boy of weight 2.8 kg who is currently fine at 4 years of life.

In the index pregnancy, aneuploidy screening (Dual screen, Triple screen ) and targeted Ultrasonography including placental morphology were normal. She developed gross polyhydramnios at 28 weeks and was admitted. Amniotic fliud reduction was required at 29 weeks POG for respiratory distress. She was started on tab Indomethacin since and serial fetal echocardiography was done to monitor patency of ductus arteriosus. GTT repeated in the third trimester was within normal limits. Dose of Indomethacin was increased with the progressive polyhydramnios. Despite medical therapy, amnioreduction was required thrice more during pregnancy at 30 6/7, 31 2/7 and 32 weeks respectively. She had preterm labour at 33 weeks and delivered a newborn of 2 kg by lower segment caesarean section (LSCS) due to intrapartum fetal distress. There was no obvious malformation in the newborn, and neonate did not developed any features suggestive of neuromuscular disorder at 3 and 6 month of life. At one year followup, both the infant and mother are doing well.

## Discussion

Polyhydramnios is usually associated with fetal structural and/ or chromosomal abnormalities in majority of the cases. Structural anomalies interfering with fetal swallowing and fluid absorption e.g.

gastrointestinal obstruction in duodenal atresia and neuromuscular disorders like anencephaly lead to polyhydramnios. The incidence of chromosomal abnormality in fetuses of women with idiopathic polyhydramnios has been reported upto a rate of 3.2% [8]. Some authorities suggest amniocentesis if targeted scan is suggestive of fetal anomaly or in case of history of recent infection in the mother [9].

Maternal complications associated with idiopathic polyhydramnios include a higher incidence of malpresentation, macrosomia, preterm delivery and caesarean delivery [10]. The reported increased risk of perinatal morbidity and mortality with idiopathic hydramnios warrants increased antenatal fetal surveillance. Hershkowitz et al. have studied the middle cerebral artery (MCA). Doppler velocimetry of 113 pregnancies complicated by idiopathic hydramnios and found a higher rate of an abnormal MCA pulsatility index in the pregnancies with hydramnios compared to the controls [11]. Biophysical profile and Doppler study of umbilical artery done in the present case was within normal limits.

In women with severe and symptomatic polyhydramnios, Indomethacin can be administered to relieve discomfort and preterm labour and dose can be titrated to maximum of 2-3 mg/kg per day according to the relief. Indomethacin is discontinued at around 32-34 weeks due to the risk of premature ductal constriction. Serial fetal echocardiac evaluation is recommended if duration of therapy exceeds 48 h beyond 24 weeks gestation. Amnioreduction alone or as an adjunct to indomethacin therapy has been reported to be beneficial in women with polyhydramnios. In a review by Dickinson et al. in one hundred thirty eight women with symptomatic polyhydramnios amnioreduction was found to be an effective measure to prolong pregnancy for about 26 days with minimal morbidity [12]. Both Indomethacin upto the dose of 3 mg/kg and amnioreduction were used in the index case for symptomatic relief and prolongation of pregnancy.

Beischer et al. [3] have reported a case of recurrent polyhydramnios spanning four pregnancies, of which first was affected by aneuploidic fetus, second pregnancy was uneventful and subsequently polyhydramnios recurred consecutively in the third, fourth and fifth pregnancies where no cause except large for gestation fetus was found. Other causes of recurrent polyhydramnios in this review were Diabetes (37.8%), fetal malformation (18.9%), non- immune hydrops (0.05%) and chorioangioma (0.05%) [3]. In another review by Shimuzu et al. the cause of recurrent polyhydramnios was found to be HLA incompatibility of the couple [5]. Rare causes of polyhydramnios like fetal infection, Bartter syndrome, HLA incompatibility and abnormal amniotic fluid prolactin levels has also been reported in the literature [4,5,6]. Role of prolactin in the amniotic fluid affecting the permeability and fluid levels through receptors has been postulated to cause polyhydramnios. In a review by De Santis et al. the amniotic fluid levels were measured and were found to be high in women with recurrent acute polyhydramnios [4]. However, lack of data and heterogenous results do not justify measurement of amniotic fluid prolactin levels, electrolytes and HLA type for routine evaluation.

Neonatal examination and regular follow up of babies with idiopathic polyhydramnios may reveal some abnormality at follow up. In a retrospective analysis by Touboul et al. 4/23 infants had some abnormality like polyuria, infection and West Syndrome which could explain the polyhydramnios antenatally retrospectively [13]. Polyhydramnios recurred in three pregnancies in index case with normal fetal outcome in 2 pregnancies and no obvious cause could be detected at 1 year and 4 year follow up of the children.

### Conclusion

Diagnosis of idiopathic polyhydramnios warrants a complete evaluation, especially if the condition is recurrent. Diabetes screening, targeted anomaly scan, amniocentesis, detection of fetal karyotype and infection screen are important tools to rule out possible causes of polyhydramnios. The main concerns of idiopathic recurrent polyhydramnios are preterm delivery and fetal malformation. Preterm labour may be prevented by use of Indomethacin and serial amniocentesis. Regular follow up of neonate after delivery is essential to find any rare contributory cause.

## **Conflict of Interest**

None

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