Polyhydramnios and pregnancy complicated with Bartter’s syndrome: A case report

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ABSTRACT

Bartter syndrome is a renal tubular defect that can be diagnosed prenatally, reports are limited on the prenatal course of pregnancies complicated with the disease in the current literature. In this case review we would like to define and debate on the course of pregnancy complicated with Bartter’s syndrome in regard with current literature. Rising levels of polyhydramnios without certain fetal or placental abnormalities should lead us to suspect renal functional disorders like this in which the evaluation of amniotic fluid chloride level is usually diagnostic. A series of amniocentesis’ were performed until 34 weeks. Neonatal and subsequent investigations confirmed the diagnosis of Bartter syndrome. The infant was healthy at birth and now, is at 4 years of age under constant follow-up by pediatricians.

1. Introduction

Antenatal Bartter syndrome (ABS) is a rare autosomal recessive renal tubular disorder. The defective chloride transport in the loop of henle draw leads to fetal polyuria hence resulting in severe polyhydramnios and premature delivery. Early initiation of the symptoms along with unexplained polyhydramnios often challenges the treating obstetrician. Gradually increasing levels of polyhydramnios without apparent placental or fetal abnormalities should at least give raise suspicions about this entity. Biochemical analysis of amniotic fluid is usually diagnostic. Early recognition, maternal treatment with indomethacin, and amniocentesis can help continue the pregnancy (Nakanishi et al., 2005). Polyhydramnios is dominantly present throughout, the pregnancy especially between 24th and 30th weeks of gestation. During antenatal follow-up growth restriction, nephrocalcinosis, polyuria and events of dehydration and hypercalciuria are characteristics regarding postnatal course (Rodriguez, 2004).

The disease is generally diagnosed during the infancy or early childhood period. Prenatal diagnosis of any affected fetus with Bartter syndrome is substantial for several reasons including, the need to treat polyhydramnios at an early onset and to be prepared for possible premature delivery along with
prompt treatment of symptoms such as blood and urine electrolyte instability at an early time after birth which is especially considered to be essential if we want to decrease the pathological severeness and the occasional alleviations of the disease. In terms of treatment Indomethacin is found to be an effective agent against the gradual raise of polyhydrannios anding us against premature delivery in Bartter syndrome (Moise et al., 1988). Here in this report we would like to present a case of Bartter syndrome complicated with severe polyhydrannios and managed with recurrent drainage

2. Case report
A 22-year-old patient (G2P1A0) with 24 weeks of pregnancy who had her previous pregnancy diagnosed with Bartter syndrome was referred to our clinic. According to the history taken from the patient, her other child is currently 4 years old and in good health under regular observation by pediatrics and also the patient revealed that she and her husband are related on 1st degree. When asked about her current pregnancy, patient reveals that a detailed fetal ultrasonographic scan was taken at 20th week showing no significant anomalies other than polyhydrannios with amniotic fluid index more than 32 cm in total with the deepest pool being 15 cm. After admission patient was hospitalized in our clinic administered betametazon for lung maturation and put under close observation including nst (non stres test), biophysical profiling and fetal doppler ultrasonography every 2-3 times a week which were all in normal range.

Series of amniocenteses were carried out in order to control uterine contractions and increasing dyspnea associated with polyhydrannios, respectively at weeks 28, 31 and 33 up to the point where persistent uterine contractions developed at 34th week. The fetus was delivered with cesarean section. A male infant with APGAR scores of 3 at 1st min and 8 at 5th min with a birth weight of 2030 g (10-50 percentile) was delivered. Neonatal course was complicated with hyponatraemia and polyuria. The diagnosis of Bartter Syndrome was confirmed by laboratory findings afterwards. The child is currently doing well, at 4 years of age, since prostaglandin synthetase inhibitors are usually required to control the disease he is receiving Indomethacin therapy and is being followed by pediatricians

3. Discussion
Case reports of pregnancy complicated with Bartter syndrome are rare. This syndrome was primary described in 1962. Onset may be infancy, childhood, in the neonatal period (Amiralok and Dawson, 2000). Polyhydrannios occurs in 1–2.8% of all pregnancies and in some cases the aetiology is not identified. Polyhydrannios usually arises from fetal anomalies (mostly gastro-intestinal), uncontrolled maternal diabetes, chromosomal abnormalities and neural tube defects such as anencephaly. Unexplained severe polyhydrannios is a challenge for obstetricians (Rodriguez, 2004). Consistent with the literature, as in this case, when faced with gradually increasing severe polyhydrannios we would like to emphasise ABS where no other cause can be found. Marked fetal polyuria due to antenatal Bartter syndrome is one of the rarer causes of violent polyhydrannios. Patients with this syndrome usually present in infancy or childhood with chronic pain related to growth restriction or hypokalemia (Tomoyoshi and Toshihide, 2001).

Maternal amniocentesis and indomethacin are the options that consent pregnancy to continue. Indomethacin, by inhibiting prostaglandin synthetase, reduces salt-wasting and maternal administration of indomethacin successfully prevents the advance of polyhydrannios.

However, this therapy might have a risky negative effect. In the fetus, prostaglandin E keeps the patency of the ductus arteriosus. Indomethacin, being a strong inhibitor of prostaglandin synthesis, might bring about constriction of the ductus. Hence, wary clinical and echocardiographic monitoring of the fetus is essential (Bhat et al., 2011). In our case, we didn’t use indomethacin, instead we preferred to control polyhydrannios by series of amnio-drainage to buy us enough time to get to 34th week.

Indomethacin and serial amnio-drainage is an effective viable treatment option and especially more efficient together in terms of preventing premature delivery if used carefully.

In conclusion, we would like to say that Bartter syndrome should be considered in severe idiopathic polyhydrannios cases where no other obvious cause is present.

REFERENCES


