Antenatal Diagnosis of Esophageal Atresia

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Abstract

Oesophageal atresia (OA) encompasses a group of congenital anomalies comprising of an interruption of the continuity of the oesophagus with or without a persistent communication with the trachea. OA occurs in 1 in 2500 live births. We report the case of a 33 year old patient referred to our unit for severe Polyhydramnios at 24 week of amenorrhea. Ultrasound examination revealed the absence of stomach bubble. During fetal swallowing we observed a pouch at the upper level of the neck not vascularized at doppler. There was no other associated fetal abnormality and no chromosomal abnormality. We discussed the preened post-natal diagnosis of esophageal atresia, associated malformations should be seeded and management of esophageal atresia.

Keywords: Oesophagial Atresia, Pough Sign, stomach bubble, polyhydramnios.

INTRODUCTION

Oesophageal atresia encompasses a group of congenital anomalies comprising an interruption of the continuity of the oesophagus combined with or without a persistent communication with the trachea.

The first clearly documented case and confirmed at post mortem examination was recorded by Thomas Gibson in 1697 [1], the etiology is multifactorial and remains unknown.

We have reported the case of an esophageal atresia diagnosed prenatally in our obstetrics and gynecology department I at Hassan II CHU in Fez.

OBSERVATION

We report the case of a 33-year-old patient, without significant pathological antecedent, gravida 4 para 3, with three normal vaginal deliveries without incident. The patient was referred to our unit at 24 weeks of amenorrhea with severe Polyhydramnios. Obstetrical ultrasound examination revealed the absence of stomach bubble (Figure-1) with an abdominal circumference below the 10th percentile. During fetal swallowing we observed a pouch at the upper level of the neck not vascularized at doppler (Figure 2 & 3). There was no other associated fetal abnormality and no chromosomal abnormality.
**DISCUSSION**

Oesophageal atresia is a relatively common congenital malformation occurring in one in 2500–3000 live births. The majority of cases of oesophageal atresia are sporadic/non-syndromic. Familial/syndromic cases of oesophageal atresia are extremely rare, representing less than 1% of the total. Oesophageal atresia is 2 to 3 times more common in twins than the total population [2].

The first clearly documented case and confirmed at post mortem examination was recorded by Thomas Gibson in 1697 [1], then Thomas Hill in 1840 reported the second case of esophageal atresia but the first one associated with an anal anomaly [3].

Five major types of tracheoesophageal abnormalities was first described by Vogt in 1929 [4], still used today, and then have been modified by Ladd in 1944 [5], Gross in 1953 [6] and Kluth in 1976 [7]. Vogt described 6 types of esophageal atresia:

- Absence of oesophagus,
- Isolated esophageal atresia (Ladd I, Gross A),
- Esophageal atresia with a fistula connecting the proximal portion of the esophagus with the trachea (Ladd II, Cross B),
- Esophageal atresia with a fistula connecting the distal portion of the esophagus with the trachea (Ladd III, Cross C),
- Esophageal atresia with a double fistula connecting both segments of the interrupted esophagus with the trachea (Ladd V, Cross D),
- A tracheoesophageal fistula without esophageal atresia: H or N type fistula (Cross E).

The third variety is the most common and accounts for 85% to 90% of all cases [8].

The motility of the oesophagus is always affected either due to abnormal innervation by an abnormality in neuropeptide distribution [9] or to vagal nerve damage occurring during the surgical repair. The resting pressure in the whole oesophagus is significantly higher than in normal patients and the closing pressure of the lower oesophageal sphincter is reduced.

The trachea is also abnormal in oesophageal atresia. The abnormality consists of an absolute deficiency of tracheal cartilage and an increase in the length of the transverse muscle in the posterior tracheal wall [10].

The diagnosis of oesophageal atresia may be suspected prenatally by the finding of a small or absent fetal stomach bubble on ultrasound scan performed after the 18th week of gestation. Overall the sensitivity of ultrasonography is 42% but in combination with polyhydraminos the positive predictive value is 56% [11]. Polyhydraminos alone is a poor indication of oesophageal atresia (1% incidence).

Available methods of improving the prenatal diagnostic rate include ultrasound examination of the fetal neck to view the blind-ending upper pouch [12] and to observe fetal swallowing and magnetic resonance imaging [13].

In post-natal, the diagnosis is made using A stiff wide-bore (10–12 French gauge) catheter passed through the mouth into the oesophagus. In oesophageal atresia the catheter will not pass beyond 9–10 cm from the lower alveolar ridge. A plain X-ray of the chest and abdomen will show the tip of the catheter arrested in the superior mediastinum (T2–4) while gas in the stomach and intestine signifies the presence of a distal tracheoesophageal fistula. The absence of gastrointestinal gas is indicative of an isolated atresia. A fine bore catheter may curl up in the upper pouch giving the false impression of an intact oesophagus or rarely it may pass through the trachea and proceed distally into the oesophagus through the fistula. The X-ray may reveal additional anomalies such as a "double bubble" appearance of duodenal atresia, vertebral or rib abnormalities.

There is an increased incidence of associated anomalies in pure atresia (65%) and a lower incidence in H-type fistula (10%). The systems affected are as follows: Cardiovascular 29% (ventricular septal defect and tetralogy of Fallot), anorectal anomalies 14%, gastrointestitinal 113% (duodenal atresia, pyloric stenosis and malrotation), vertebral/skeletal 10%, respiratory 6%, and genetic 4%.

Some associations which may include oesophageal atresia are the CHARGE association (coloboma, heart defects, atresia choanal, retarded growth and development, genital hypoplasia and ear deformities), POTTER'S syndrome (renal agenesis, pulmonary hypoplasia, typical dysmorphic facies), SCHISIS association (omphalocoele, cleft lip and/or palate, genital hypoplasia) and VATER association.
Esophageal atresia is a congenital disorder in which the esophagus is incomplete, resulting in a gap between the upper and lower parts that cannot be surgically connected directly. This gap may be due to a failure in the growth of the esophagus during the embryonic period. The symptoms of esophageal atresia can be severe, including difficulty swallowing and respiratory distress. Treatment typically involves surgical intervention to reconnect the esophagus. The prognosis for survival is directly related to several factors, including the presence of a major cardiac defect, birth weight, and the presence of other congenital anomalies. Management strategies often include the use of suction catheters to maintain a clear airway and to prevent aspiration. The surgical repair of esophageal atresia is a complex procedure that requires careful planning and consideration of multiple factors to ensure the best possible outcome for the child.
