Management and Outcome of Oesophageal Atresia and Tracheoesophageal Fistula in Pediatrics (Our experience at Queen Rania Al-Abdullah Hospital for Children)

Ahmad Abu Qurah MD; Waseem Al-Meflh, MD*; Amir Al-Ibrahim, MD*; Gaith Khaswneh MD*; Samer karadsha, MD*; Ahmad Alrymoni, MD*.*

ABSTRACT

Objective: To report our experience at Queen Rania Al-Abdullah Hospital for Children (QRHC) in the management of oesophageal atresia (EA) and tracheoesophageal fistula (TEF) regarding prognostic predictors, outcome, complications, survival and death rates.

Method: A retrospective study was conducted by reviewing the medical records of patients with EA-TEF who were admitted to QRHC in the period from March 2017 to November 2019. Total number of cases was 40; 26 males and 14 females with a male to female ratio of 1.86:1. The post-operative follow up period ranged from 8 to 24 months (mean was 18 months). Demographic data, complications, survival and death rates were collected to analyse our outcome.

Results: Out of our 40 patients, 34 (85%) cases were EA Gross type C, 5 (12.5%) were type A and 1 (2.5%) case was type H. Patient mean age was 22.5 hours (ranging from 5 hours to 3 days), mean body weight was 2.1 kg (ranging from 1.1 to 3.6 kg) and mean gestational age was 33.5 weeks (ranging from 28 to 38 weeks). Congenital anomalies were seen in 60% (24 patients); the most common was cardiac, which presented in 50% (12 patients), while the second most common was urogenital anomalies in 33% (8 patients). The VACTERL association was demonstrated in one patient. No chromosomal anomalies were detected.

The survival rate was 80% (32 cases); the 8 patients who died were premature, 7 (87.5%) of whom had a low birth weight (less than 2.5 kg) and 5 (62.5%) of whom had cardiac anomalies. Regarding complications, 27.5% complained of Gastroesophageal Reflux Disease (GERD) (11 cases), 20% developed oesophageal anastomotic stricture (8 cases) and 10% developed anastomotic oesophageal leak (4 cases).

Conclusion: The main prognostic predictors of outcome in the management of EA-TEF were gestational age (GA), birth weight (BW) and associated congenital anomalies, mainly cardiac. The survival rate is improving due to advances in neonatal ICU, anaesthetics and surgical techniques.

Keywords: oesophageal atresia, prognostic predictors, tracheoesophageal fistula.

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INTRODUCTION

EA is a congenital malformation of the oesophagus where the oesophagus does not connect to the stomach and ends as a blind pouch, with or without a connecting fistula to the trachea. [1, 2, 3, 4, 5] It is one of the most common life-threatening congenital anomalies of the oesophagus in paediatrics. [1,6]

* From the Department of Paediatric Surgery, Queen Rania Al-Abdulla Hospital for Children, K.H.M.C, Amman, Jordan.
Correspondence should be addressed to Dr. Ahmad Abu Qurah, King Hussein medical center, Royal medical services, Amman-Jordan,Email: ahmad_abuqurah@yahoo.com
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It results from the failure of tracheoesophageal septum development,[3] and it has an incidence of 2 per 2500-4000 live births with a predominance in males. [2, 3, 7, 8]

It has been shown that 50% of infants with EA-TEF have associated congenital anomalies. The most common are cardiac anomalies (35%); others are renal (25%), gastrointestinal (21%), vertebral (20%) and musculoskeletal (14%). The VACTERL association (vertebral, anorectal, cardiac, tracheoesophageal, renal and limb anomalies) in 20-30%.[2,3,6,8]

ET is classified anatomically by Gross into five types: Type A (7.5%) is characterised by EA without TEF, type B (1%) is EA with proximal TEF, type C (86%) is EA with distal TEF, type D (1%) is EA with both proximal and distal TEF, and type E (4%) is TEF without EA, [3, 8] so nearly 90% of patients with EA have TEF. [9,10]

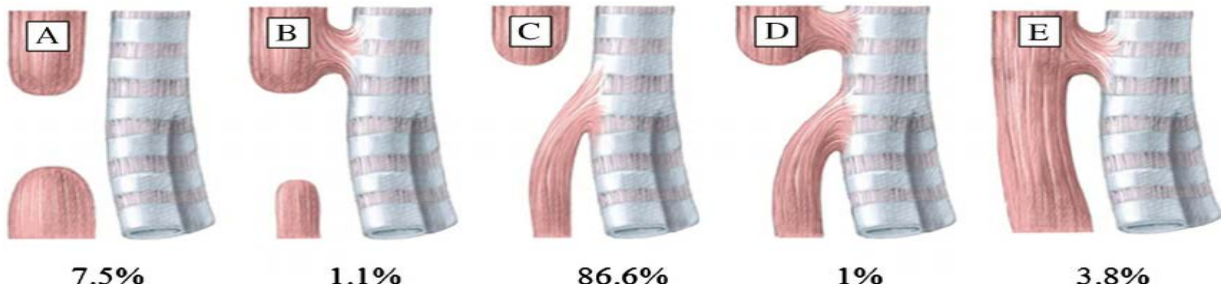

Figure 1 : types of esophageal atresia.

Modified from Puri M, Höllwarth ME. Pediatric Surgery. Springer Eds, 2006.

There are several prognostic classifications for EA related to the prognostic factors that affect operative repair, predict outcome and survival. Of these, the Waterston classification which is based on birth weight, the presence of pneumonia and congenital anomalies, where the survival is 100% for patients with a BW more than 2.5 kg and otherwise healthy while it is 65% for those with a BW less than 2 kg and otherwise well or higher weight with sever associated cardiac anomaly, another classification is the Spitz classification which is based on birth weight and major cardiac anomalies, where the survival is 97% for patients with a birth weight more than 1.5 kg without major congenital heart disease while it is 22% for those with a birth weight less than 1.5 kg associated with congenital heart disease. [7, 8, 11, 12, 13]

Prenatal diagnosis of EA is rare because prenatal ultrasound (U/S) findings of polyhydramnios and absence of a small gastric bubble are non-specific and not always present. In addition, the pouch sign of a dilated proximal oesophagus that appears as an anechoic area in the middle of the foetal neck is technically difficult to identify (observation requires foetal swallowing over time),[1,4,8,14] so most cases of EA are diagnosed postnatally when the patient presents with drooling, inability to swallow, coughing, choking and cyanosis at the first feeding. The diagnosis is confirmed by coiling of a nasogastric tube (NGT) in the proximal oesophagus on chest x-ray, but still surgical exploration is the definitive method to prove the type of oesophageal atresia. [1, 8]

Operative repair of EA-TEF is done after a period of one to two days of stabilising the patient, optimising the pulmonary status and detecting any associated anomalies. [6,8] Repair is performed by thoracoscopy or open thoracotomy with ligation of the TEF and primary anastomosis of the oesophageal ends in type C. [6,15]

For a long gap EA (defined as an oesophageal gap more than 3 cm or spanning more than two vertebral bodies), a staged approach is used, with either gastrostomy and oesophageal elongation and delayed primary repair or gastrostomy with oesophagostomy, then oesophageal replacement later on. [8]

METHODS

A retrospective study was performed at QRHC in the period from March 2017 to November 2019 by searching the medical records of patients with EA-TEF who were admitted to the QRHC paediatric ICU after being born there or transferred from other hospitals.
Data extracted include date of birth, birth weight, gestational age, age at presentation, mode of delivery, presence of maternal polyhydramnios, associated congenital anomalies, type of EA, date and type of operation, complications (leak, stricture and GERD), length of hospital stay and mortality. The post-operative follow up period ranged from 8 to 24 months (mean was 18 months).

Management protocol

Patients with EA-TEF were admitted to the surgical paediatric ICU for preoperative preparation and stabilisation by keeping the patient NPO (nil per os) with intravenous fluid (IVF), and by optimising pulmonary status by preventing pneumonitis from aspiration of saliva or gastric juice by positioning the patient in a way to decrease gastric reflux through a distal TEF (upright position or prone with head up). Frequent oropharyngeal suctioning and administration of intravenous antibiotics was also applied.
Chest x-ray and abdomen x-ray were performed to confirm coiling of an NGT in the proximal oesophagus and to assess the presence or absence of gas in the abdomen to determine the type of EA-TEF (Figure 2).


A


B

Figure 2: A – coiled NGT with absence of gas in the abdomen (Type A). B- coiled NGT in the oesophagus with gas in abdomen (Type C)

Echo, renal U/S and spinal x-ray were requested to evaluate the presence of any associated congenital anomalies.
Signed informed consent was obtained from parents after informing them about the procedure in detail and its complications. After a period of preparation and stabilisation (24-48 hours), the patient was sent to the theatre for surgical repair.

In our centre, for patients with Type C, right posterolateral thoracotomy was performed through the fourth intercostal space with either an intrapleural or extrapleural approach, followed by identification and ligation of the distal TEF using non-absorbable proline 4/0 sutures, then mobilising and anastomosing both oesophageal ends (using absorbable Polydioxanone sutures PDS 5/0) over NGT size 6 Fr. Finally, a chest tube is inserted and closure of the wound is done.
The patient is kept fully relaxed on a mechanical ventilator for 4-5 days. Parenteral nutrition is started on the first day and feeding via an NGT is started on the 4th to 5th post-operative day if there is no evidence of leak and progresses gradually until the patient can tolerate oral feeding.
For type A, we perform gastrostomy and oesophagostomy and later on oesophageal replacement via gastric pull up when the patient had grown up with adequate body weight and stomach size.
In our center we are looking forward to start thoracoscopic repair for EA- TEF type C in the near future.

RESULTS

The study included 40 patients; 26 were male and 14 were female with a male to female ratio of 1.86:1. Out of our 40 patients, 34 (85%) cases were EA Gross type C, 5 (12.5%) were type A and 1 (2.5%) case was type H. Patient age on admission ranged from 5 hours to 3 days with a mean age of 22.5 hours. Body weight birth weight ranged from 1.1 to 3.6 kg with a mean coiled NGT in the oesophagus with gas in abdomen (Type C) of 2.1 kg; 10 patients had a BW less than 1.5 kg, 20 patients were between 1.5 and 2.5 kg and 10 patients had a coiled NGT in the oesophagus with gas in abdomen (Type C) greater than 2.5 kg. The mean gestational age was 33.5 weeks with gestational age ranged from 28 to 38 weeks; 33 cases were premature (less than 37 weeks). 12 cases were delivered by caesarean section and the remaining 28 by normal vaginal delivery. 18 patients (45%) had maternal polyhydramnios on prenatal U/S. The duration of hospital stay ranged from 7 days to 25 days with a mean of 9.5 days.
Congenital anomalies were seen in 60% (24 patients). The most common were cardiac (Atrial Septal Defect (ASD), Ventricular Septal Defect (VSD, Patent Ductus Arteriosus (PDA), which presented in 50% (12 patients), while the second most common was urogenital anomalies (hydronephrosis, undescended testes, hypospadias), which were seen in 33% (8 patients). The VACTERL association was demonstrated in one patient. No chromosomal anomalies were detected. Demographic and clinical data of our EA-TEF patients are summerized in table I,II,and III.

Table I

total number of patients	40
sex	26 (65%) male , 14 (35%) females
birth weight	mean BW 2.1kg (ranged from 1.1-3.6kg) 10 patients <1.5 kg, 20 between 1.5-2.5kg, 10 >2.5 kg
gestational age (GA)	mean GA 33.5 week (range from 28-38 week) 33 patients <37 week(premature), 7 patients are term
mode of delivery	normal vaginal delivery :28 patient , C/S : 12 patients
presence of maternal polyhydramnios	18 patients (45%)

Table II

Type of EA-TEF	number of patients	percentage of patients	number of males	number of females
Type C	34	85%	24 (70.6%)	10 (29.4%)
Type A	5	12.5%	2 (40%)	3 (60%)
Type H	1	2.5%	1 (100%)	-----

Table III

Type of congenital anomalies	**number of patients**	**percentage**
cardiac	12	50%
genitourinary	8	33%
gastrointestinal	5	12.5%
VACTERL	1	2.5%

The survival rate was 80% (32 cases); the 8 patients who died were premature with gestational age ranging from 28 to 35 weeks. Seven of them had a low birth weight (3 patients were less than 1.5 kg and the other 4 were between 1.5 to less than 2.5 kg) and 5 patients had cardiac anomalies.

Regarding complications, 27.5% complained of GERD (11 cases), which was diagnosed by paediatric gastroenterologists and treated conservatively with lifestyle, dietary modifications and anti-reflux drugs. As our policy is to refer EA-TEF patients with recurrent respiratory symptoms to the paediatric pulmonologist and gastroenterologist for follow up, 20% (8 cases) developed oesophageal anastomotic stricture, which was diagnosed by oesophagogram for patients with symptoms (dysphagia, foreign body obstruction or aspiration); three of them had oesophageal leak. The earliest stricture was diagnosed two months post-repair. All of these patients were treated successfully with balloon dilatation; the duration to first balloon dilatation ranged from 2 to 14 months after repair and the frequency of balloon dilatation ranged from 3 -5 times. 10% (4 cases) developed anastomotic oesophageal leak, which was confirmed by oesophagogram that is not done routinely in our centre, but is only performed for patients suspected to have an anastomotic leak; 3 cases had a minor leak that was treated conservatively (NPO, intravenous antibiotics, chest drainage and total parenteral nutrition) for about one week while one patient developed a major leak and underwent repeat thoracotomy with repair of the leak.

DISCUSSION

Oesophageal atresia is the most common serious congenital anomaly of the oesophagus. [1, 8] The survival rate is improving dramatically to more than 90% [1, 5, 8, 13, 16, 17] due to advances in neonatal ICU, improvement in anaesthetic and surgical techniques, antibiotics, Total Parenteral Nutrition (TPN) and advancements in the management of associated congenital malformations. [8, 9, 11, 18, 19, 20]

The main poor prognostic factors that are associated with increased mortality and morbidity are low BW, prematurity, congenital heart disease, long oesophageal gap and associated congenital anomalies. [8, 18, 19, 20]

The most common post-operative complications are:

1- Anastomotic leak (15%), which is usually predisposed by ischemic oesophageal ends and anastomotic tension. The majority are minor leaks that close spontaneously by conservative treatment, while major leaks are uncommon and need repeat surgery.[8,19,21]

2- Anastomotic stricture (15-60%), which is an oesophageal narrowing that is detected on oesophagoscopy or oesophagogram and associated with symptoms (dysphagia, foreign body obstruction or aspiration), usually predisposed by anastomotic leak, GERD and poor surgical technique. The majority are treated with balloon dilatation with a good response. [8, 21, 22, 23]

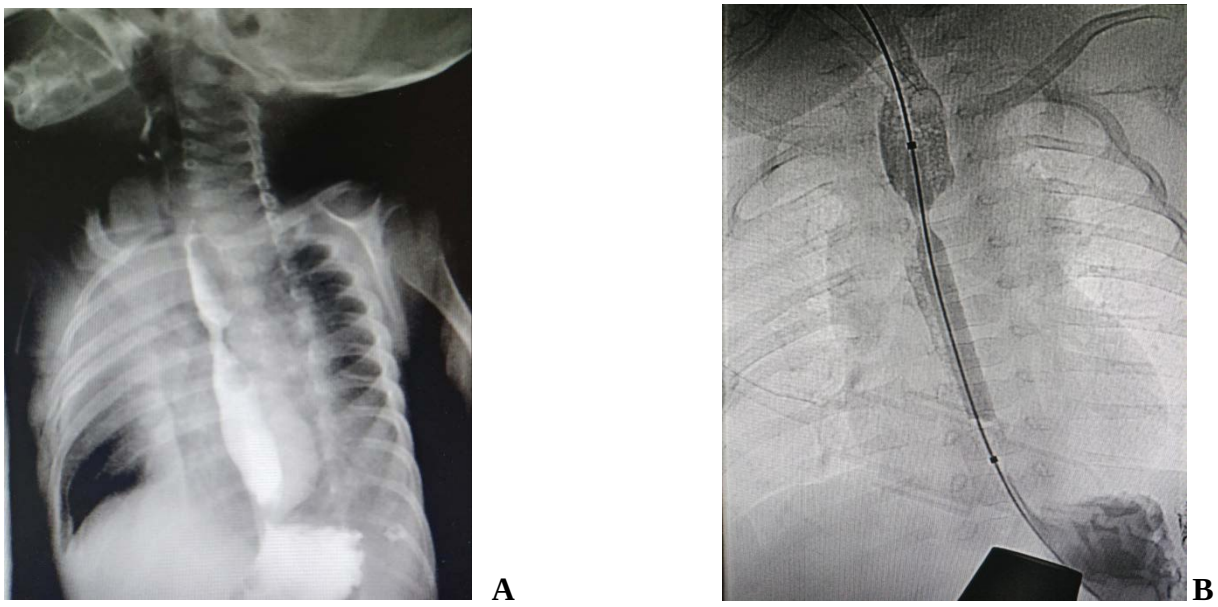

Figure 3: **A:** oesophageal anastomotic stricture appears on oesophagogram, **B:** balloon dilatation for oesophageal anastomotic stricture.

3- Recurrent TEF (5%) secondary to anastomotic leak, which needs surgical repair. [19]

4- GERD (50%) due to oesophageal dysmotility and short intraabdominal oesophagus. It is treated by anti-reflux medications, but if medical treatment fails or refractory oesophageal stricture develops, then anti reflux surgery is performed. This is needed in one quarter of cases.[8]

The survival rate in our study was 80%, which was comparable with studies published by **Ryuta Masuya** [12] in Japan (80.8%) and **Florian Friedmacher** [20] in Austria (84.4%).

In our study, the main prognostic predictors for survival were gestational age, birth weight and associated congenital anomalies, mainly cardiac. This is in contrast to a study by **Robert Peter,**[13] which was conducted in UK, with the main predictors of outcome as low BW, cardiac disease and preoperative pneumonia. **Ryuta Masuya** [12] presented the main prognostic factors as cardiac and chromosomal anomalies.

The post-operative complications in a study by **R. Shan**[24] were oesophageal leak in 3%, oesophageal stricture in 31% and GERD in 39%. Also, in a study by **Miroslav Vukadin,**[19] performed in Belgrade, the complications were leak in 5%, stricture in 28% and recurrent TEF in 3%, comparable to our study (leak 10%, stricture 20% and GERD 27.5%). Table IV summarizes the outcome of EA-TEF repair regarding survival and complication rates in different studies.

Table IV: outcome of EA-TEF repair(survival and complication rates)

study	number of cases	survival rate	complications		
			GERD	*esophageal stricture*	*esophageal anastomotic leak*
our study at QRHC	40	80%	27.5%	20%	10%
Ryuta Masuya [12]	73	80.8%	39%	39%	0%
Florian Friedmacher [20]	109	84.4%	85.5%	71.9%	11.5%
R. Shan[24]	110	100%	39%	31%	3%
Miroslav Vukadin [19]	60	75%	5%	28%	5%

Kiarash Taghavi[25] recommended after a study performed in New Zealand that the routine use of bronchoscopy immediately prior to EA repair facilitates intubation beyond the site of TEF, and moreover detects the site of the TEF and coexisting anomalies such as tracheomalacia and laryngeal cleft. In contrast, in our hospital, we do not perform bronchoscopy routinely before repair except in type (H) TEF to facilitate the localisation of the TEF during dissection by inserting a Fogarty catheter inside it; otherwise it is not done to avoid complications such as desaturation, bradycardia, laryngospasm and airway injury. Still, there is wide variation regarding this issue among centres worldwide.

All the EA-TEF repairs were performed in our study by an open approach, except for Type H by the thoracoscopic approach, but the new trend is to start thoracoscopic repair for EA-TEF type C because of its advantages such as better visualisation and cosmoses, less need for post op narcotics and shorter hospital stay. **Florian Fridmacher**[20] performed procedures by an open approach, and mentioned that thoracoscopic repair should be done by a qualified surgeon and requires very good surgical skills.

Paola Papoff[26] suggested a non-surgical technique for the treatment of respiratory distress that results from gastric distention in EA-TEF type C by inserting an umbilical catheter through the distal TEF to the stomach via a flexible bronchoscope to decompress the stomach and relieve distention. In our study, one patient developed respiratory distress before the operation; he was intubated with low pressure Mechanical Ventilation and underwent urgent thoracotomy with ligation of the fistula and was repaired later on.

We routinely perform oesophageal anastomosis over a feeding tube during EA repair. The advantages of this feeding tube are early enteral feeding, resulting in shorter time to full regular oral feeding, earlier discharge from hospital and avoidance of prolonged TPN with its complications, especially when there is a minor leak. Enteral feeding can be resumed earlier via a feeding tube as part of conservative treatment to decrease TPN, although **Sarath Kumar Narayanan**[15] suggests avoiding a transanastomotic feeding tube, as this will not increase the complication rate and it may decrease the incidence of aspiration pneumonitis.

Gawad[27] reported that the use of a chest tube is not indicated routinely after EA repair because it does not decrease the rate of early post-operative complications or the length of hospital stay. While our policy is the routine use of a chest tube for early diagnosis and the treatment of post-operative complications such as anastomotic leak, the chest tube is removed once there is no evidence of leak and the patient has started feeding.

Yuichi Okata[21] described the importance of performing oesophageal anastomosis under less tension in EA repair to avoid or decrease post-operative oesophageal leak and stricture, Moreover, **Tate Nice**[22] identified the risk factors for oesophageal strictures, which are oesophageal leak, staged repair, GERD and thoracoscopic repair. In our study, 8 patients(20%) developed oesophageal anastomotic stricture; 3 of them had oesophageal leak. The earliest stricture was diagnosed two months post-repair, and all of these patients

were treated successfully with balloon dilatation. The duration to first balloon dilatation ranged from 2 to 14 months after repair and the frequency of balloon dilatation ranged from 3 to 5 times.

In our study, no case developed refractory oesophageal stricture to balloon dilatation that necessitated repeat thoracotomy and resection of the stricture as in the case report by **Azakpa**[23] describing a refractory stricture in a female baby post-repair of an EA type C. After 3 years of lost follow up, when a diagnosis of neglected refractory oesophageal stricture was confirmed by oesophagogram and oesophagoscopy, surgical resection of the stricture with end to end anastomosis was performed after the failure of multiple trials of balloon and bougienage dilatation.

Administration of prophylactic anti-reflux medication (PARM) such as proton pump inhibitors or H2 blockers after EA repair did not decrease the incidence of oesophageal anastomotic stricture as mentioned by **Hiromu Miyake**;[17] in our hospital, PARM is indicated for the treatment of patients with confirmed GERD post-EA repair, not as prophylaxis for stricture because as **Hiromu Miyake** [17]noted, there is still no consensus regarding type, dose and duration of PARM; moreover, its feasibility and safety need more studies. Also, **Pernilla Stenström**[28] observed that proton pump inhibitors did not affect the frequency of balloon dilatation for oesophageal stricture and the overall need for balloon dilatation.

CONCLUSION

The main prognostic predictors of outcome in the management of EA-TEF were gestational age, body weight and associated congenital anomalies, mainly cardiac. The survival rate is improving due to advances in neonatal ICU, anaesthetics and surgical techniques. EA-TEF patients need frequent hospital follow-up care to manage late complications such as oesophageal stricture and GERD, in addition to continuous education of medical staff caring for these patients.

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