JKAU: Med. Sci., **Vol. 17** No. 4, pp: 59-72 (2010 A.D. / 1431 A.H.)
DOI: 10.4197/Med. 17-4.5

Congenital Tracheoesophageal Fistula with or without Esophageal Atresia, King Abdulaziz University Hospital Experience over 15 Years

Osama M. Rayes, FRCSI, FICS

Division of Pediatric Surgery, Department of Surgery
Faculty of Medicine, King Abdulaziz University,
Jeddah, Saudi Arabia
orayes@kau.edu.sa

Abstract. The aim is to study the profile of congenital tracheoesophageal fistula with or without esophageal atresia with the effect of modalities of surgical and postoperative management on their outcome. Retrospective study of the records of all cases that had been diagnosed as congenital tracheoesophageal fistula with or without esophageal atresia treated at King Abdulaziz University Hospital, between 1992 and 2007 inclusive were reviewed. A total of 48 neonates were studied. Forty-three cases (72.8%) had esophageal atresia with distal fistula: ten cases were pure esophageal atresia; one case had esophageal atresia with proximal and distal fistulae. Four cases had H-type of tracheoesophageal fistula. On applying the Waterston prognostic classification to all patients, the survival rate was 94.5%, 76.2% and 22.2% for classes A, B and C, respectively. Incidence of esophageal congenital anomalies in KAUH was 1:3574 life births, with male: female ratio 3:2. The overall mortality rate was 25%**,** whereas the mortality rate among operated cases was 20%. Seven cases developed stricture (15.5%) and 5 had anastomotic leaks (11.1%).

Keywords: Esophageal atresia, Tracheoesophageal fistula, H type, TEF, TOF.

Correspondence & reprint request to: Dr. Osama M. Rayes
P.O. Box 80215, Jeddah 21589, Saudi Arabia
Accepted for publication: 18 July 2010. Received: 14 January 2010.

Introduction

The first recorded case of esophageal atresia (EA) was in 1670, by Durston[1]. He found a blind-ending upper esophagus in one of a pair of female thoracopagus conjoined twins. Gibson[2] in 1697, however, documented the first classic description of an EA with distal fistula. In 1936, Simpson-Smith[3] was the first to attempt a correction of EA at Great Ormond Street Hospital, London, UK. However, Lanman[4] was the first to perform an extra pleural repair in 1936. His patient lived for only 3 hours, and in 1940 he reported his experience with 30 operative cases, all of whom died. He stated that with greater experience, improved technique and good luck, success would soon be reported. Only 3 years later, in 1939, Leven[5] and Ladd[6] independently reported the first survivors, but only after staged reconstruction. Haight and Towsley[7] performed the first successful primary repair in a 12-day-old female neonate, this after 10 previous unsuccessful attempts. Different treatment protocols are available in literature describing variable methodology about management of such congenital anomalies, all of which are discussing the preoperative, operative and postoperative management.

Method

All medical records of patients that had been diagnosed to have EA with tracheoesophageal fistula (TEF) seen at King Abdulaziz University Hospital, Jeddah, Saudi Arabia, between 1992 and 2007 inclusive were reviewed. Retrospective analysis was done for demographic data, preoperative, operative as well post operative details. Furthermore, prognostic factors like prematurity, birth weight, associated congenital anomalies, respiratory status at referral or pre-operatively, and chromosomal abnormalities were studied. All the pediatric surgeon consultants dealing with these anomalies were applying the management protocol (mentioned below) as strict as applicable. Only the cases on which this protocol was applied were included in this study.

Management protocol of the surgeon was applied on all patients as follows: Preoperative diagnosis was built on antero-posterior and lateral neck, chest and upper abdomen X-ray, or baby gram, with nasogastric tube pushed nasally, (as contrast study should not be ordered). ECHO, renal and brain ultra sound was done routinely. Neonatal care, included

antibiotics and ventilation was provided according to patient's need and neonatologist opinion. Patient was nursed supine with head up position if there was lower oesophageal fistula, and in head down if was absence. Nasoesophageal tube (double lumen type) was inserted and connected to low continuous suction.

The operative procedure included right dorsolateral thoracotomy, extra pleural approach, ligation and trans-section of the azygos vein, then ligation, trans-section and repair of TEF using a 5/0 non-absorbable monofilament suture. The upper pouch was located, dissected and anastomosed to the lower esophagus, using a 5/0 non-absorbable monofilament suture, then an inter-costal chest tube / under water system was inserted. If dealing with long gap, bridging was overcome in the following order; upper pouch dissecting was done, with or without Livaditis circular myotomy, at one, two or three levels. Then, the lower esophageal dissection with Livaditis myotomy was also done, only if it was needed.

Results

Fifty-nine neonates were found with different esophageal congenital anomalies and managed by the same permanent consultant pediatric surgeons at the centre. The studied 48 cases were diagnosed as TEF with or without EA. All of whom were treated following the management protocol mentioned above.

A total 57.9 % had associated congenital malformations, the details of which are listed in Table 1. Cardiac malformations affected 28% of the cases, 4 had cleft palate, skeletal anomalies 9% of cases, genitourinary 3%, and isolated renal anomalies affected 2%. Furthermore, 8% VATER, 4% had VACTERL (Vertebral, Anal, Cardiac, Tracheoesophageal, Renal or Radial, Limb) associations, while one had chromosomal abnormality as a case of Edward's syndrome. Two cases had conal atresia and one had congenital diaphragmatic hernia. Then, the different types were studied separately.

EA with Distal TOF (43 cases)

Forty-three cases had EA with distal TOF, 33/43 cases (76.7%) were delivered by SVD, and were full term, while 10/43 cases (23.3%) were delivered preterm with a mean gestational age of 33 weeks. Only 15/43

cases (34.9%) were delivered at our center. Birth weight ranging from 0.63 – 4 kg (mean 2.75 kg). An APGAR score at 1 minute ranged 1-9 (mean 6.7) and a score at 5 minute ranged 5-10 (mean 8.9). In Table 2, details of hospital course of cases with EA and distal TEF according to age at surgery are illustrated. The number of neonates presented with drooling was 32/43 cases (74.4%), and 8/43 cases (18.6%) presented with cyanosis. All patient diagnosed by chest X-ray, which showed a coiled NGT in upper esophagus and gas in the stomach; 8/43 (18.6%) of them had preoperative contrast study before referral which supported the diagnosis.

Table 1. Shows congenital abnormalities of different types of esophageal anomalies.

Congenital Anomalies	EA with distal TEF cases N = 43		H-type Fistula N = 4	
Cardiac	14	32.5%	0	
Skeletal	5	11.62%	0	
Imperforated Anus	4	9.3%	0	
Renal and Genitourinary	3	6.97%	0	
VATER	4	9.3%	1	25%
Chromosomal	1	3.2%	0	
VACTERL	2	6.5%	0	
Cleft palate	2	6.5%	2	50%
Coanal atresia,	2	6.5%	0	
Diaphragmatic hernia	1	3.2%	0	

Table 2. Shows details of hospital course of cases with EA & distal TEF according to age at surgery.

Parameter	< 72 Hr N = 30	< 14 Day N = 9	Late N = 1
TAT out (days) (mean ± SD)	15.6 ± 5.21	18 ± 3.2	35
CT out (days) (mean ± SD)	14.2 ± 3.51	12.5 ± 3.24	35
Ba swallow (days) (mean± SD)	11 ± 3.21	12.6 ± 2.15	10
Leak (n)	3 (Livaditis in 1)	2	1
Ventilation (days) (mean ± SD)	3.7 ± 1.2	3.9 ± 1.9	6
NGT feed (days) (mean ±SD)	6 ± 2.2	5.2 ± 1.8	
Oral feed (days) (mean ± SD)	14 ± 5.2	14.6 ± 4.2	23
Last F/U (years) (mean ± SD)	3.7 ± 1.3	4 ± 2.2	
Stricture (n)	3	3	1
Dilatation (n)	2 cases twice &1 case x 4	2	1 case x 10 times
Hospitalized (years) (mean ± SD)	26 ± 6.3	26 ± 4.3	30 ± 3.3
Deaths (n)	9	0	0

Thirty (69.8%) cases were operated upon within 72 hours of birth; twenty nine cases had thoracotomy, fistula ligation and esophageal anastomosis, while only one of them had Livaditis myotomy. All except one had extra pleural approach. One case had only laparotomy (gastrostomy and colostomy); no thoracotomy was done.

Nine (20.9%) cases were operated upon during the 2nd week of their life. Seven of them had extra pleural approach; Livaditis was not done in any of the nine cases.

One (2.3%) patient of the 43 cases was operated on the 86th day of life, due to late referral, where at day one gastrostomy and colostomy were done in a different hospital. Repair through extra pleural approach with Livaditis myotomy was performed on the patient.

Almost 50% of our patients were Saudi and 63% were males. Unfortunately, 47% of the patients' studies were delivered as un-booked deliveries, giving an acceptable reason for lacking of some patient's information. Stricture was found in 6 (13.9%) cases, all strictures underwent balloon dilatation; with mean 2.33 dilatations. It was successful in all cases except in one, where gastrostomy was done due to failed dilatation. Leak complicated 5 (11.6%) cases; all were managed conservatively by NPO and antibiotic. It was also successful in all cases, except one case which required re-operation and end-to-end anastomosis. This group mortality (12 cases) were 9 died after definitive surgery and 3 died before definitive surgery (1 Edward's syndrome), while 2 cases had gastrostomy, only due to associated major congenital anomalies. Table 3 describes details of the nine deaths after definitive surgery and Table 4 shows details of the three deaths before definitive surgery.

Table 3. Shows the characters' of the nine deaths after definitive surgery.

	Sex	GA	BW kg	Anomaly	Death	Notes
1	F	FT	1.86	(VACTER +conal atresia)	died 10th day post operative	
2	M	FT	2.65	(CHD)	died 10th day post operative	due to septicemia
3	F	36 wk	2.25	(CHD: dextrocardia)	arrested within 24 h of operation	
4	F	FT	2.45	(Conal atresia)	died 98th day post operative	leak, re-operated at 2 months
5	F	FT	3.19	(Renal anomaly + CHD)	died 14th day post operative	
6	F	35 wk	2		died at the age of 22days	re-operated for fistula closure
7	M	FT	3.03	(Conal atresia + atrophic eyes)	died 44th day post operative	
8	M	FT	2.39	(VATER +CHD)	died 54th day post operative	
9	F	FT	2	(radial anomaly + short neck	died 56th day post operative	Tension at repair

Table 4. Shows the three deaths occurred before definitive surgery.

	Sex	GA	BW kg	Anomaly	Death age	Procedure
1	M	24 wk	0.63	(CHD + NEC)	died at 38th day post operative	Fistula ligation gastrostomy and colostomy, (NEC) bowel resection
2	M	32 wk	1.9	(huge CDH +VATER)	died after laparotomy	No thoracotomy laparotomy only
3	M	FT	2.16	(Edward syndrome)	died after 24 Hrs	Only gastrostomy and colostomy

H-type TOF

Four (7.02%) cases with H-type TOF were treated; 3 (75%) cases delivered by SVD; 3 (75%) cases were FT and 1 (25%) case had GA of 34 wks. All cases were transferred from other hospital. Two (50%) cases presented with recurrent pneumonia, and another 2 (50%) cases presented with cyanosis. All patients were diagnosed by preoperative contrast study which showed communication between the trachea and esophagus. Two (50%) cases underwent extra pleural thoracotomy approach; the other 2 underwent cervical approach. The age at operation ranged from 17 - 1020 days (255 day). Contrast, swallow was done from 7 - 8 days. Three patients were extubated immediately after operation; one patient required long period ventilation. Nasogastric tube feeding was started 1 - 5 days with an average of 3 days. Oral feeding started between 5 - 8 days (6 days). No death occurred. Hospitalization period was 7 - 105 days with an average of 35 days. Last follow-up age was 9 m - 5 years (2.25 years).

EA with Proximal and Distal Fistulas

Only one (1.75%) case had this diagnosis; a product of SVD. The patient was transferred from another hospital with a BW of 2.0 kg. The patient presented with drooling and cyanosis. Diagnosis reached by preoperative contrast study; chest and abdomen X-ray showed a coiled NGT and gas in the stomach. The patient was operated upon at the age of 7 days through extra pleural approach, and had smooth post operative period with no leak. Although, the patient developed stricture, he responded well to balloon dilatation twice.

Table 5 summarizes the course of different types of the TOF +/- EA, and Table 6 shows plotting of results according to Waterston, Spitz & Montréal classification.

Table 5. Summary of course of different types of the TOF with or without fistula.

	EA with distal TE Fistula N = 43	H-type TOF N = 4	EA with Prox. & Dist. Fistulae N = 1
Birth weight kg Mean ± SD	2.75 ± 2.1	3.1 ± 1.23	2.0
Gestational age Mean ± SD	33 ± 5.9	34 ± 5.1	33
Presentation	Drooling in 32 (85.96%). cyanosis 9	(2)Recurrent Pneumonia (2)cyanosis	With drooling
Diagnosis	Coiled NGT, gastric-gas 8 cases, Preop. contrast study	Contrast Study & Bronchoscopy	Coiled NGT and Stomach Gas Pre-operative contrast
Approach (n)	38 cases with extra-pleural & 3 cases no definitive repair	2 cervical 2 extra pleural	extra pleural
Livaditis(n)	2		
Age at op. days Mean ± SD	4 ± 3.9	140 ± 30.1	7 days
TAT out days Mean ± SD	16.8 ± 7.21		
Ventilation days Mean ± SD	4.04 ± 3.5		
NGT Feeding days Mean ± SD	5.59 ± 4.8	3 ± 2.3	
Oral Feeding days Mean ± SD	14.4 ± 6.5	6 ± 1.2	
BA swallow days Mean ± SD	12.06 ± 4.9	13.7 ± .25	
Leak(n)	5		No leak
Stricture(n)	6		1
Dilatation Mean ± SD	1.74 ± 1.32		2
Hosp. Period days Mean ± SD	27.19 ± 20.41	12.3 ± 5.98	
Death (n)	12	0	0

Table 6. Plotting of the results according to Waterston, Spitz & Montréal classification.

Classification		Patients	Survival Rate
Watterson	W A	18	94.5%
	W B	21	76.2%
	W C	9	22.2%
Spitz	I	34	88.2%
	II	13	53.8%
	III	1	100%
Montreal	2 high risk	6	50%
	1 low risk	42	80.4%

W A - Birth weight > 2500 g and otherwise healthy
W B- Birth weight 2000 – 2500 g and Well or higher weight with moderate pneumonia, and congenital anomaly
W C Birth weight < 2000 g or higher with .sever pneumonia and sever congenital anomaly
Spitz I Birth weight > 1500 g without major congenital heart disease
Spitz II Birth weight < 1500 g OR major congenital heart disease
Sptiz II Birth weight < 1500 g AND major congenital heart disease.

Discussion

Stringer *et al.*[8] showed that in 87 mothers, whose fetus was found to have a small or absent stomach, only 15 fetuses (17%) had EA at birth. However, when the small or absent stomach was accompanied by

hydramnios, the positive predictive value for EA was 56%. There is a well-known incidence of additional congenital anomalies in infants with esophageal atresia. In a cohort of 253 infants, 122 (48%) had 213 associated anomalies[9]. The VACTERL association may be attended by an increased mortality rate or multiple associated malformations. At least 3 components of the VACTERL association were present in 10% of the patients. Of the cardiac anomalies, ventricular septal defect was the most common, followed by clinically significant patent ductus arteriosus and tetralogy of Fallot.

The CHARGE Association[10] (coloboma of the eye, heart anomaly, choanal atresia, retardation, and genital plus ear anomalies), present in 2% of their cases, carried a high mortality rate (70%), mainly related to the presence of major cardiac anomalies. In the presence of duodenal atresia associated with EA and TEF, ligation of the distal TEF takes precedence. In favorable circumstances it may be combined with primary esophageal anastomosis followed by duodenoenterostomy[11]. Cleft lip and palate were present in 2.6% of cases in one study and were associated with a mortality of 54%. Again, this was due to a severe cardiac anomalies or the presence of multiple associated anomalies[12]. The incidence of congenital esophageal stenosis was estimated at 1:25,000 to 50,000 live births, and the incidence of other congenital anomalies associated with CES ranges from 17% to 33%[13]. There are 3 pathologic/ histological types of CES that have been described in the literature: (1) Fibro muscular thickening, (2) cartilaginous ring, and (3) membranous web[14,15].

The prevalence of 2/10,000 EA with or without TEF, according to this study is close in our area in comparison to other reports of 2.6 and 2.86 in Europe and Hawaii[16-18]. Our population was consistent with those of previous published reports on EA/TEF with regard to anatomic types; male-to-female ratio, associated congenital anomalies, and complications of treatment[19,20]. In this study, 7.0 % had chromosomal abnormality, (1 case had Edward syndrome and 2 other cases had Down syndrome) compared to 10% in Depaepe *et al.*[16] and 8% in Konkin *et al.*[21]. Furthermore, 8% had VATER, and 4% had VACTERL, associations were fewer, in comparison with 37% in other reports[22]. In agreement with Rogers *et al.*[23] and Giadaro *et al.*[24], it was found that mortality was mainly related to the presence of severe multiples associated congenital malformations and severe congenital cardiac

malformations. The results of applying the Waterston prognostic classification to patient population in this study was shown in Table 6. Also, Montreal and Spitz classification survival rate was applied. The survival rate in this series was comparable to other neighboring Gulf countries[25,26], but it was less than the results published by David E. Konkin *et al.*[21]. According to this study, EA cases with distal TOF the overall mortality rate was 25%, 12/48 (12 cases). The mortality rate in operated cases was 20%, 9/45 (9 cases), and 5 cases (11.1%) 5/45 developed localized leakage. This anastomotic leakage rate was comparable to many other modern series[27-30]. Seven cases developed esophageal strictures post operatively making an incidence of (15.5%) in this series, it was slightly lower than that seen by others. This may be attributed to the more homogeneous patient population that had been studied, omitting those with pure EA the likely longer gap disease[20,31].

One of our patients had Livaditis myotomy for long gap. This patient had developed post operative leak and treated conservatively. Distal circular myotomy was very useful, however, delicate procedure that can help solve the problem given by long gap EA[32]. At any rate, tension was a more important risk factor of anastomotic leak of the 2 variables, as it has been reported by others in both clinical and laboratory studies[28,30,33]. This study reported a series of 4 isolated H Type TEF cases (6.7%), in which all of them were diagnosed using preoperative contrast study and bronchoscopy. Two cases (50%) underwent extra pleural approach; the other 2 underwent neck approach. The outcome was favorable in all cases with no major complications. The classical symptoms constitute the triad of Helmsworth and Pryles[34,35], were coughing and aspiration during feeding, abdominal distention and repeated cyanosis and pneumopathy. The initial radiological investigations consisted of a plain chest X-ray and a contrast swallow. The former symptoms may show suggestive signs such as gastro-intestinal and in particular esophageal, and gaseous distension[36-38]. For some authors, a contrast swallow was sufficient to make the diagnosis.

In a series of 30 patients reported by Beasley[39] the initial contrast swallow demonstrated the fistula in 73% of cases and in all patients by the third attempt. In this study, 4 patients with H-type fistula were reported: 2 cases (50%) presented with recurrent pneumonia, and the other 2 cases (50%) presented with cyanosis. All of them were diagnosed preoperative contrast study that showed communication between the

trachea and esophagus. Endoscopy enables direct visualization of the TEF. This examination has become more reliable and less dangerous with the improvements in anesthetic techniques and endoscopic equipment. A tracheoscopy performed under general anesthesia during spontaneous ventilation allowed the fistula to be visualized directly and its origin with respect to the carina or the vocal cords to be accurately established. This determined whether a cervical approach was appropriate for surgical treatment of the fistula or not. In the reported series, the postoperative mortality was not negligible. Chavrier[40] reported a frequency of 6:66, Bedard[41] 4:20 and Yazbeck 1:16. In all of these cases, mortality was due to respiratory problems (infection, respiratory distress, recurrence or inhalation), in particular when a thoracotomy was performed[36,40,41]. None of our patients died, and none developed stricture or leakage. It is important to notice that the incidence of this disease in our centre, according to total number of life birth was 1:3574.

Conclusion

The continue usage of management protocol is recommended. Incidence of esophageal congenital anomalies in KAUH was 1:3574 life births, with male: female ratio 3:2. The frequency of different sub-types seen in our centre was as follows: (72.8%) had EA with distal fistula; 16.9% were pure esophageal atresia; the H-type of tracheoesophageal fistula was 6.7%. While 1.7% had EA with proximal and distal fistulae, and only 1.7% had congenital esophageal stenosis. The overall mortality rate was 25%, 12/48, whereas the mortality rate in operated cases was 20%, 9/45. Anastomotic stricture was seen in 15.5% and the leak was 11%.

References

[1] **Durston W.** A narrative of a monstrous birth in Plymouth, October 22: Together with the anatomical observations taken thereupon by William Durston, Doctor of physic and communication to Dr. Tim Clerk. *Philos Trans R Soc* **16705**: 2096-2098.

[2] **Gibson T.** *The Anatomy of Human Bodies Epitomized.* 5th ed. London: Awnsham and Churchill. 1697. 21.

[3] **Simpson-Smith A.** Quoted by Williams DI In: *A Tradition of Surgery.* Simpson Smith Memorial Lecture, Institute of Child Health, London, England, 1981.

[4] **LANMAN TH.** Congenital atresia of the esophagus. *Arch Surg* 1940; **41**: 1060-1083.

[5] **LEVIN NL.** Congenital atresia of the esophagus with tracheoesophageal fistula. *J Thorac Cardiovasc Surg* 1941; **10**: b 648-657.

[6] **LADD WE.** The surgical treatment of esophageal atresia and tracheoesophageal fistulas. *N Engl J Med* 1944; **230**: 625-637.

[7] **HAIGHT C, TOWSLEY HA.** Congenital atresia of the esophagus with tracheoesophageal fistula; Extrapleural ligation of fistula and end-to-end anastomosis of esophageal segments. *Surg Gynecol Obstet* 1943; **76**: 672-688.

[8] **Stringer MD, McKenna KM, Goldstein RB, Filly RA, Adzick NS, Harrison MR**. Prenatal diagnosis of esophageal atresia. *J Pediatr Surg* 1995; **30**(9): 1258-1263.

[9] **Chittmittrapap S, Spitz L, Kiely EM, Brereton RJ.** Oesophageal atresia and associated anomalies. *Arch Dis Child* 1989; **64**(3): 364-368.

[10] **Kutiyanawala M, Wyse RK, Brereton RJ, Spitz L, Kiely EM, Drake D, Blake K.** CHARGE and esophageal atresia. *J Pediatr Surg* 1992; **27**(5): 558-560.

[11] **Spitz L, Ali M, Brereton RJ.** Combined esophageal and duodenal atresia: experience of 18 patients. *J Pediatr Surg* 1981; **16**(1): 4-7.

[12] **Spitz L, Yang X, Pierro A, Kiely EM, Drake D.** Adverse association of oesophageal atresia and cleft lip and palate. *Br J Surg* 2003; **90**(6): 716-719.

[13] **Rowe MI, O'Neill JA, Grosfeld JL.** *Essentials of Pediatric Surgery.* St Louis, MO, Mosby, 1995. 959-961

[14] **Nihoul-Fékété C, De Backer A, Lortat-Jacob S, Pellerin D.** Congenital esophageal stenosis: A review of 20 cases. *Pediatr Surg Int* 1987; **2**: 86-92,

[15] **Cloud DT.** Anastomotic technic in esophageal atresia. *J Pediatr Surg* 1968; **3**(5): 561-564.

[16] **Depaepe A, Dolk H, Lechat MF.** The epidemiology of tracheo-oesophageal fistula and oesophageal atresia in Europe. EUROCAT Working Group. *Arch Dis Child* 1993; **68**(6): 743-748.

[17] **Robert E, Mutchinick O, Mastroiacovo P, Knudsen LB, Daltveit AK, Castilla EE, Lancaster P, Källén B, Cocchi G.** An international collaborative study of the epidemiology of esophageal atresia or stenosis. *Reprod Toxicol* 1993; **7**(5): 405-421.

[18] **Forrester MB, Merz RD.** Epidemiology of oesophageal atresia and trachea-oesophageal fistula in Hawaii, 1986–2000. *Public Health* 2005; **119**(6): 483-488.

[19] **Poenaru D, Laberge J, Neilson IR, Guttman FM.** A new prognostic classification for esophageal atresia. *Surgery* 1993; **113**(4): 426-432.

[20] **Chittmittrapap S, Spitz L, Kiely EM, Brereton RJ.** Anastomotic stricture following repair of esophageal atresia. *J Pediatr Surg* 1990; **25**(5): 508-511.

[21] **Konkin DE, O'hali WA, Webber EM, Blair GK.** Outcomes in esophageal Atresia and tracheoesophageal fistula. *J Pediatr Surg* 2003; **38**(12): 1726-1729.

[22] **Cano Garci-Nuño A, Solís Sánchez G, Coto Cotallo GD, Cepeda Martínez A, Ramos Aparicio A, López Sastre J, Crespo Hernández M.** Esophageal atresia and associated anomalies *An Esp Pediatr* 1992; **36**(6): 455-459.

[23] **Mee RB, Beasley SW, Auldist AW, Myers NA.** Influence of congenital heart disease on management of esophageal atresia. *Pediatr Surg Int* 1992; **7**(2): 90-93.

[24] **Gidaro GS, Chiesa PL.** [Modern trends in the treatment of esophageal atresia]. *G Chir* 1997; **18**(10): 560-564.

[25] **Nawaz A, Matta H, Shawis R, Jazcobsz A, Kassir S, Al-Salem AH.** Esophageal atresia and tracheoesophageal fistula: success and failure rates in the United Arab Emirates. *Pediatr Surg Int* 1998; **14**(3): 214-217.

[26] **Hassab MH, Swilem MA, Al-Gobair F.** Profile and Outcome of Esophageal Atresia in a Rapidly Developing Area. *Kuwait Med J* 2002, **34**(1): 12-15,

[27] **Chittmittrapap S, Spitz L, Kiely EM, Brereton RJ.** Anastomotic leakage following surgery for esophageal atresia. *J Pediatr Surg* 1992; **27**(1): 29-32.

[28] **McKinnon LJ, Kosloske AM.** Prediction and prevention of anastomotic complications of esophageal atresia and tracheoesophageal fistula. *J Pediatr Surg* 1990; **25**(7): 778-781.

[29] **Engum SA, Grosfeld JL, West KW, Rescorla FJ, Scherer LR 3rd.** Analysis of morbidity and mortality in 227 cases of esophageal atresia and/or tracheoesophageal fistula over two decades. *Arch Surg* 1995; **130**(5): 502-508.

[30] **Tsai JY, Berkery L, Wesson DE, Redo SF, Spigland NA.** Esophageal atresia and tracheoesophageal fistula: Surgical experience over two decades. *Ann Thorac Surg* 1997; **64**(3): 778-784.

[31] **Nambirajan L, Rintala RJ, Losty PD, Carty H, Lloyd DA.** The value of early post operative oesophagography following repair of oesophageal atresia. *Pediatr Surg Int* 1998; **13**(2-3): 76-78.

[32] **Giacomoni MA, Tresoldi M, Zamana C, Giacomoni A.** Circular myotomy of the distal esophageal stump for long gap esophageal atresia. *J Pediatr Surg* 2001; **36**(6): 855-857.

[33] **Brown AK, Tam PKH.** Measurement of gap length in esophageal atresia: A simple predictor of outcome. *J Am Coll Surg* 1996; **182**(1): 41-45.

[34] **Adkins JC.** Congenital malformations of the esophagus. In: *Pediatric Otolaryngology,* C. Bluestone, S.E. Stool, (eds.), 2 ed, Philadelphia: WB Saunders, 1990. 973–984.

[35] **Chavrier Y, Revillon Y.** [Congenital tracheoesophageal fistulas in children]. *Chir Pediatr* 1984; **25**(4-5): 228-233.

[36] **Crabbe DC, Kiely EM, Drake DP, Spitz L.** Management of the isolated congenital tracheo-oesophageal fistula. *Eur J Pediatr Surg* 1996; **6**(2): 67-69.

[37] **Smith WL, Franken EA, Smith JA.** Pneumoesophagus as a sign of H type tracheoesophageal fistula. *Pediatrics* 1976; **58**(6): 907-909.

[38] **Beasley SW, Myers NA.** The diagnosis of congenital tracheoesophageal fistula. *J Pediatr Surg* 1988; **23**(5): 415-417.

[39] **Genty E, Attal P, Nicollas R, Roger G, Triglia J, Garabedian EN, Bobin S.** Congenital tracheoesophageal fistula without esophageal atresia. *Int J Pediatr Otorhinolaryngol* 1999; **48**(3): 231-238.

[40] **Bedard P, Girvan DP, Shandling B.** Congenital H-type tracheoesophageal fistula. *J Pediatr Surg* 1974; **9**(5): 663-668.

[41] **Yazbeck S, Dubuc M.** [Congenital tracheoesophageal fistulas without esophageal atresia]. *Chir Pediatr* 1983; **24**(2): 113-116.

الناسور الخلقي بين الرغام والقصبة الهوائية مع أو بدون انسداد المريء: خبرة مستشفى جامعة الملك عبدالعزيز لمدة ١٥ عام

أسامة محمد ريس

شعبة جراحة الأطفال، قسم الجراحة، كلية الطب، جامعة الملك عبدالعزيز جدة – المملكة العربية السعودية

المستخلص. يهدف هذا البحث إلى تقييم نتائج الأطفال المصابين بالتشوهات الخلقية للمريء، سواء صاحب ذلك ناسور بين الرغام والقصبة الهوائية أم لا. وكذلك دراسة مختلف طرق العلاج والمضاعفات في نتائجهم النهائية. درست جميع ملفات المرضى المشخصين، والذين عولجوا بمستشفى جامعة الملك عبد العزيز بجدة، المملكة العربية السعودية، خلال الفترة يناير ١٩٩٢م إلى نهاية ديسمبر٢٠٠٧م، واستخرجت المعلومات الأساسية والديموغرافية لهم والتشوهات الخلقية المصاحبة وتفاصيل العلاج ونتائجها.

كان عدد المرضى المصابين بمختلف الأنواع من تشوهات المريء هو ٤٨ مريضاً فقط. يمثل النوع المشهور منهم أي انسداد علوي مع ناسور سفلي (٧٢,٨٪) منهم، وتم استبعاد الحالات ذات التشخيص انسداد تام بدون ناسور، لتفادي تكرار النشر، وكانت تمثل ١٦,٩٪ من الحالات. مثلت حالات الناسور الرغامي نوع H ٦,٧٪. وكان هناك حالة واحدة من انسداد المريء مع ناسور علوي. تم تطبيق تقسيم وترسون، وتقسيم سبيتز، وتقسيم مونتريال، واستنتجنا النتائج التالية: احتمالية الإصابة بالمرض هي

٣٥٧٤:١ وولادة طفل حي بنسبة ٣ ذكور: ٢ إناث. تقسيم نسب الأنواع المختلفة هي كما ذكر أعلاه.

النسبة العامة للوفاة كانت ٢٥٪. بينما كانت ٢٠٪ في الحالات التي أجريت لها عمليات جراحية. سبعة مرضى أصيبوا بتضيق بعد العملية (١٥,٥٪) وخمس حالات تسريب المريء(١١,٠٪)