International Surgery Journal
Gadgade BD et al. Int Surg J. 2016 Feb;3(1):318-320
http://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: http://dx.doi.org/10.18203/2349-2902.isj20160251

Research Article

Outcomes of esophageal atresia in a tier II referral centre

Bahubali D. Gadgade[1]*, Venkatesh M. Annigeri[1], Gautam Kabbin[2], Anil Halgeri[1]

[1]Department of Peadiatric Surgery, SDM College of Medical Sciences and Hospital, Sattur, Dharwad, Karnataka, India
[2]Department of Peadiatrics, SDM College of Medical Sciences and Hospital, Sattur, Dharwad, Karnataka, India

Received: 02 January 2016
Revised: 13 January 2016
Accepted: 16 January 2016

***Correspondence:**
Dr. Bahubali Gadgade,
E-mail: bahudg@gmail.com

ABSTRACT

Background: To study the clinical profile, complications and outcomes of neonates with esophageal atresia (EA) and/or tracheoesophageal fistula (TEF) in a tier II referral centre.
Methods: This is a retrospective study of 54 patients with EA and/or TEF from 2008 to 2014.
Results: Fifty four cases of EA/ TEF which were reviewed, 32 (59.26%) were male and 22 (40.74%) were female patients. The mean birth weight was 2.38 kg. 39(72.22%) babies were born at term and 15(27.78%) were preterm. Most common anatomical variant was type C. Most common associated anomaly was cardiac, seen in 8 patients (14.81%). Post operative barium study showed minor leaks in 7 patients. 22 of 54 patients (40.74%) died.
Conclusions: Early recognition of symptoms and early referral to hospitals would improve the outcomes of neonates with EA/TEF. Sepsis remains the leading causes of mortality, and delayed referral with onset of pneumonia contribute to sepsis.

Keywords: Esophageal atresia, Tracheoesophageal fistula, Associated anomalies

INTRODUCTION

Esophageal atresia (EA) is a congenital disconnection of the oesophageal lumen, that may be associated with a connection to the trachea, called as Tracheoesophageal fistula (TEF). It is a common congenital anomaly and incidence ranges from 1 in every 2500 to 4500 live births.[1-3] Cameron Haight in 1941, performed the first successful surgical repair of this anomaly.[2] Over the years, excellent outcomes have been achieved due to early diagnosis, improved surgical techniques, advancement in anaesthesia and good perioperative and postoperative neonatal care. Survival rates as high as 95% have been reported in centers which offers best neonatal care.[4] We aimed at studying the clinical profile, complications and outcomes in neonates with EA/TEF in our hospital which is a tier II referral centre and analysing the determinants of survival.

METHODS

All neonates admitted in SDM College of medical sciences and hospital between 2008 to 2014 was retrospectively reviewed from the medical record department. Neonates born at the authors' institution and those referred from other saller hospitals with diagnosis of esophageal atresia and/or trachea esophageal fistula were included in the study. Parameters like antenatal history, demographics (birth weight, sex, and gestation), referral pattern, associated anomalies, surgical details and mortality rates were studied. All patients underwent standard posterolateral thoracotomy and repair if type C and cervical esophagostomy and gastrostomy if type A. Post surgery, contrast study was done in all cases before starting feeds and on follow up in suspected cases of stricture.

RESULTS

Fifty four cases of EA/ TEF which were reviewed. 32 (59.26%) were male and 22 (40.74%) female patients. Antenatal history of polyhydramnios was present in 12 (22.22%) patients. The mean birth weight was 2.38 kg. 39 (72.22%) babies were term neonates and 15 (27.78%) were preterm. 28 (51.85%) were born in our hospital and 26 (48.15%) were referred from other centres.

Most common anatomical variant was type C, found in 51 cases (94.44%), and followed by type A in 3cases (5.56%). Associated systemic anomalies were found in 15 cases (27.78%). In our study most common associated anomalies was cardiac anomaly (Ventricular septal defects and patent ductus arteriosus) in 8 (14.81%) followed by genito-urinary abnormalities in 4 (7.4%), anorectal malformations in 2 (3.7%) and polydactyly in 1 (1.85) %.

Surgery was performed within 24 to 48 hours of diagnosis. 3 patients underwent gastrostomy and cervical esophagostomy for pure esophageal atresia. Rest of the neonates with EA /TEF underwent postero-lateral thoracotomy, division of fistula and end to end primary anastomosis. 7 patients had minor leaks that were documented by barium swallow (done on post op day 5) and managed conservatively. During follow up, barium study was done in suspected cases of anastomotic narrowing in which 4 patients showed strictures. These were managed by dilatation. The number of neonatal deaths were 22 (40.74%) out of 54 patients (Table 1).

Table 1: Frequency of survival and mortality among neonates.

Characteristics	Survival N (%)	Mortality N (%)
Male	15 (46.87%)	17 (53.15%)
Female	17 (77.27%)	05 (22.72%)
Full term	30 (76.92%)	09 (23.07%)
Preterm	02 (13.33%)	13 (86.66%)
Inborn	20 (71.42%)	08 (28.57%)
Referred	12 (46.15%)	14 (53.84%)
Pneumonia		
Inborn	03 (60%)	02 (40%)
Referred	06 (40%)	09 (60%)
Age at presentation in hours		
< 24	24 (72.72%)	09 (27.27%)
24-48	05 (71.42%)	02 (28.57%)
>48	03 (21.42%)	11 (78.57%)

DISCUSSION

Of 54 cases which were reviewed, EA/TEF was more in males 59.26% as also seen in a study by Bindi et al.[5] Lower incidence among female could be due to reluctance to provide treatment to female newborns with congenital anomalies in our country.[6]

Antenatal history of polyhydramnios and suspicion of upper GI obstruction was seen in only 12 (22.22%). Routine antenatal ultrasonography and screening for anomalies is still not well established in the peripheral hospitals and diagnosis is most often made postnatally following onset of symptoms.

Out of 26 referred cases, 15 had aspiration pneumonia at the time of admission. A delay in diagnosis, improper shifting of such patients, time taken to reach our hospital contributed to its higher incidence. Aspiration pneumonia was seen in 5 inborn cases in the present study. EA and TEF should be suspected if a newborn has difficulty in clearing saliva, repeated episodes of coughing and choking, or transient cyanosis shortly after birth. Infants may also present with a sudden onset of respiratory distress following attempts at feeding.[7] Failure to pass a nasogastric tube with the feeling of distal resistance at the blind end of the upper oesophageal pouch helps in diagnosis (Figure 1). A plain X-ray of the chest and abdomen shows nasogastric tube coiled in the upper pouch, gas-filled intestine and stomach below the diaphragm confirming the diagnosis in EA and TEF.[7] To prevent aspiration pneumonia during transfer to hospital, a suction catheter (double lumen) can be placed in the upper oesophageal pouch to suction secretions and prevent aspiration. A preterm infant with respiratory distress requires endotracheal intubation and mechanical ventilation with position of the endotracheal tube end distal to entry of the TEF and by applying gentle low pressure ventilation which would prevent gastric over-distension and rupture of the stomach.[1]

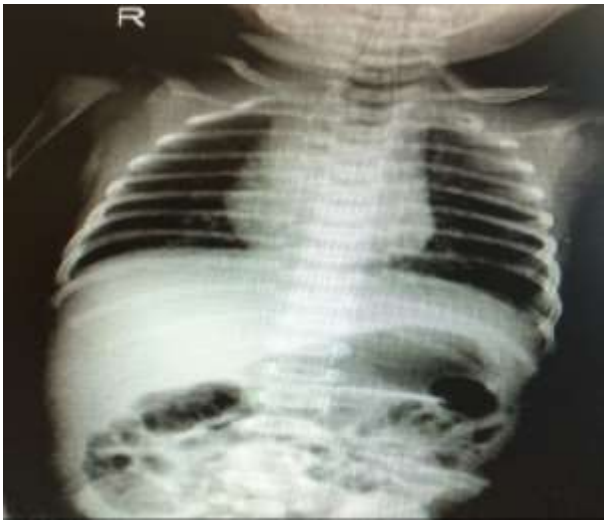

Figure 1: X-ray shows coiling of feeding tube in upper pouch.

Type C was noted in 51 cases (94%) in our study, which is the most common form of EA as reported in most series.[8] In 15 (27.78%) newborns there were associated systemic anomalies along with EA. The most common cardiac anomalies included ventricular septal defect and patent ductus arterosis, renal anomalies such as

hydronephrosis was seen in 3 cases. Hence, a thorough investigation such as echocardiogram and ultrasound of abdomen needs to be done to look for associated anomalies which also has to be addressed.

Even after surgical repair, the trachea retains a wide membranous section with a relatively narrow cartilaginous portion leading to tracheomalacia of varying severity. Abnormal tracheal epithelium and loss of goblet cells can result in susceptibility to repeated chest infections. Anastomotic leak occurs in 11–21% of patients and about 50% of these develop an oesophageal stricture.[7] In our study 7 (12.96%) patients showed minor leaks before starting feeds which settled without intervention and on follow up barium study 4 (7.4%) patients showed strictures and are currently on dilatation. Patients with EA/TEF have reduced neuronal tissue in Auerbach's plexus in the lower oesophagus and gastric plexus. Gastro-oesophageal reflux occurs in 35–58% of patients and is probably the result of intrinsic oesophageal dysfunction.[7]

In our study, mortality rate was 40.74% which was similar to study by Yang CF et al. Mortality rates vary widely. In Kumar P et al's study it was 47.9%.[9,10] Snajdauf reported 20.2%, Calisti reported 9.4% and Marinaccio reported 0% mortality rate.[11-13] In our study the most common cause of death was sepsis with/without pneumonitis leading to cardio-respiratory arrest. Delayed referral from peripheral hospitals, with onset of pneumonia is the main contributor to sepsis. Being a tier II referral centre, the authors' institute caters to a large rural/semi-rural population The observations made in this study highlight the need for creating more awareness about antenatal sonography, symptoms of esophageal atresia, quick and safe transport of the surgical neonate to a tertiary centre so that survival outcomes can improve.

CONCLUSION

In this study, sepsis remains the leading causes of mortality. Delayed referral from peripheral hospitals, with onset of pneumonia is the main contributor to sepsis. Being a tier II referral centre, the authors' institute caters to a large rural/semi-rural population The observations made in this study highlight the need for creating more awareness about antenatal sonography, symptoms of esophageal atresia, quick and safe transport of the surgical neonate to a tertiary centre so that survival outcomes can improve.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. Spitz L. Oesophageal atresia. Orphanet J Rare Dis. 2007;2(24):1-13.
2. Pinheiro PF, Simoes e, Silva AC, Pereira RM. Current knowledge on esophageal atresia. World J Gastroenterol. 2012;18(28):3662-72.
3. Spitz L. Esophageal atresia. Lessons I have learned in a 40-year experience. J Pediatr Surg. 2006;41(10):1635-40.
4. Gupta DK, Sharma S. Esophageal atresia: The total care in a high-risk population. Semin Pediatr Surg. 2008;17:236-43.
5. Bindi G, Chowdhary SK, Rao KL. Esophageal atresia with tracheoesophageal fistula: An audit. J Indian Assoc Pediatr Surg. 2004;9:127.
6. Tandon. Esophageal atresia. J Indian Assoc Pediatr Surg. 2008;13(1):5.
7. Al-Rawi O, Booker PD. Continuing Education in Anaesthesia, Critical Care & Pain J. 2007;7(1):15-6.
8. Osia S, Hadipour A, Moshrefi M, Mirzapour M. Esophageal atresia. 13 years' experience in Amirkola Children's Hospital, north of Iran. Caspian J Pediatr. 2015;1(1):22-4.
9. Yang CF, Soong WJ, Jeng MJ, Chen SJ. Esophageal atresia with tracheoesophageal fistula: ten years of experience in an institute. J Chin Med Assoc. 2006;69(7):317-21.
10. Kumar P, Ojha P. Preoperative Prediction of Survival in Oesophageal Atresia: A New Approach.Indian J of Surgery. 2002;64(6):511-5.
11. Snajdauf J, Kalousova J, Styblova J. Results of treatment of esophageal atresia. Cas Lek Cesk. 2004;143(9):614-7.
12. Calisti A, Oriolo L, Nanni L. Mortality and long term morbidity in esophageal atresia: the reduced impact of low birth weight and maturity on surgical outcome. J Perinat Med. 2004;32(2):171-5.
13. Marinaccio F, Nobili M, Niglio F. Esophageal atresia and tracheoesophageal fistula: personal contribution. G Chir. 2002;23(1-2):29-33.

Cite this article as: Gadgade BD, Annigeri VM, Kabbin G, Halgeri A. Outcomes of esophageal atresia in a tier Ii referral centre. Int Surg J 2016;3:318-20.