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Original Research Article

Management of Oesophageal Atresia and Tracheo-Oesophageal Fistula: A Prospective Study of 30 Consecutive Cases

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Abstract

Background: Oesophageal atresia with tracheo-oesophageal fistula (OA/TOF) is a life-threatening congenital anomaly requiring immediate surgical intervention. Despite advances in surgical techniques and perioperative care, the condition continues to present significant challenges, particularly in developing countries where late presentation and associated complications are common.

Objectives: To describe the management outcomes of OA/TOF, identify perioperative complications, and evaluate the association of prognostic factors with surgical outcomes.

Methods: A prospective observational study was conducted over 22 months (November 2018 to August 2020) at J.J.M Medical College, Davanagere, involving 30 consecutive neonates with OA/TOF. All patients underwent detailed clinical assessment, radiological evaluation, and surgical management via right posterolateral thoracotomy with primary oesophago-oesophagostomy.

Results: The cohort comprised 60% males and 40% females (male:female ratio 1.5:1). Gross type C was the predominant anatomical variant (96.66%). Low birth weight was present in 66.66% of cases, pneumonia in 56.66%, and associated congenital anomalies in 46.66%. Twenty-nine patients (96.66%) underwent primary repair. Overall mortality was 53.33%, with sepsis (50%) and respiratory failure (37.5%) being the leading causes of death. Postoperative complications included anastomotic leak (43.5%), dysphagia (48.3%), and stricture formation (14.3%). Waterston group A patients demonstrated 100% survival, group B 60% survival, and group C 29.4% survival.

Conclusion: Primary thoracotomy with oesophageal anastomosis remains the definitive surgical treatment for Gross type C OA/TOF. Significant postoperative complications and high mortality rates are influenced by low birth weight, pneumonia, sepsis, associated congenital anomalies, and anastomotic leak. Multidisciplinary care involving neonatologists, paediatric surgeons, and intensivists is essential for improving outcomes.

Keywords: Oesophageal atresia, Tracheo-oesophageal fistula, VACTERL association, Anastomotic leak, Neonatal surgery, Congenital anomalies, Surgical outcomes.

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Introduction

Oesophageal atresia (OA) with or without tracheo-oesophageal fistula (TOF) represents one of the most common life-threatening congenital anomalies of the foregut, occurring in approximately 1 in 3,000 to 3,500 live births worldwide.[1,2] First described by Thomas Gibson in 1697, this condition remained universally fatal until Cameron Haight performed the first successful primary repair in 1941.[3] Since then, dramatic improvements in survival have been achieved through advances in neonatal intensive care, anaesthetic techniques, and surgical expertise, with current survival rates exceeding 90% in developed countries.[4,5] The pathogenesis of OA/TOF involves disruption of normal foregut development during the fourth to sixth weeks of gestation, resulting in abnormal separation of the primitive trachea and oesophagus.[6] Multiple theories have been proposed to explain this developmental failure, including abnormalities in notochord signalling, defective Sonic hedgehog gene expression, and dysregulation of various transcription factors such as TTF-1 and HNF-3B.[7] While most cases occur sporadically, familial clustering and associations with chromosomal abnormalities suggest a multifactorial aetiology involving both genetic and environmental factors.[8] The Gross classification system, introduced in 1953, remains the most widely used anatomical categorisation. Type C, characterised by a blind-ending proximal oesophageal pouch with a distal

tracheo-oesophageal fistula, accounts for approximately 85-90% of cases.[9] Type A (isolated oesophageal atresia without fistula) represents 7-8% of cases, while types B, D, and E are considerably rarer.[10] This anatomical variation has significant implications for surgical management and prognosis.

OA/TOF frequently occurs as part of the VACTERL association (Vertebral anomalies, Anorectal malformations, Cardiac defects, Tracheo-Esophageal fistula, Renal abnormalities, and Limb defects), diagnosed when at least three of these features coexist.[11,12]

Cardiac anomalies are the most common associated malformations, occurring in 23-35% of cases, with ventricular septal defects and tetralogy of Fallot being most prevalent.[13] The presence of associated anomalies significantly impacts surgical planning and overall prognosis.

Clinical presentation typically occurs in the immediate neonatal period, with characteristic features including excessive drooling of saliva, choking with feeds, and respiratory distress due to aspiration.[14] Prenatal diagnosis has improved with routine ultrasonography demonstrating polyhydramnios and absent stomach bubble in approximately 30-40% of cases.[15] Postnatal diagnosis is confirmed by the inability to pass a nasogastric tube beyond 10 cm from the lips and plain radiography demonstrating the tube coiled in the upper oesophageal pouch with or without air in the stomach, depending on the presence of a distal fistula.[1]

Several prognostic classification systems have been developed to predict outcomes. The Waterston classification (1962) stratified patients based on birth weight, pneumonia, and presence of congenital anomalies.[16] The Spitz classification (1994) focused on birth weight and major cardiac anomalies, demonstrating superior predictive value in the modern era.[17] More recently, the Montreal classification incorporated preoperative ventilator dependence alongside associated anomalies.[18] These scoring systems guide clinicians in preoperative counselling and risk stratification.

Surgical management has evolved considerably since Haight's original technique. Primary repair via right posterolateral thoracotomy with ligation of the tracheo-oesophageal fistula and primary oesophageal anastomosis remains the gold standard for Gross type C defects when feasible.[19]

Alternative approaches include thoracoscopic repair, which has gained popularity due to reduced musculoskeletal complications, though concerns regarding increased anastomotic complications persist.[20] Long-gap oesophageal atresia presents unique challenges, often requiring delayed repair with interval oesophageal growth or eventual oesophageal replacement using gastric, jejunal, or colonic interposition.[21]

Despite advances in surgical technique and perioperative care, postoperative complications remain significant. Anastomotic leak occurs in 10-25% of cases, with major leaks requiring surgical intervention associated with considerable morbidity.[22,23] Anastomotic stricture formation affects 30-60% of patients, typically managed with serial oesophageal dilatations.[24] Recurrent tracheo-oesophageal fistula, vocal cord paresis, gastro-oesophageal reflux, and tracheomalacia represent additional long-term sequelae requiring ongoing surveillance.[25,26]

Outcomes in developing countries remain suboptimal compared to developed nations, primarily due to delayed presentation, high rates of aspiration pneumonia, malnutrition, limited neonatal intensive care resources, and high prevalence of associated anomalies.[27] Late referral patterns result in many neonates presenting with established pneumonia and sepsis, significantly compromising perioperative outcomes. Understanding these challenges is crucial for developing targeted interventions to improve survival in resource-limited settings.

The present study was undertaken to comprehensively evaluate the management and outcomes of consecutive cases of OA/TOF at a tertiary care centre in South India, with particular emphasis on identifying prognostic factors, perioperative complications, and surgical outcomes in our patient population.

Aims and Objectives

The aims of this study were:

1. To describe the surgical management of oesophageal atresia and tracheo-oesophageal fistula.
2. To determine the incidence, risk factors, and management of perioperative complications in infants with oesophageal atresia and tracheo-oesophageal fistula.
3. To evaluate the association of prognostic factors including gestational age, birth weight, gender, timing of presentation, anatomical type, and presence of VACTERL anomalies with postoperative outcomes.

Materials and Methods

This prospective observational study was conducted at the Department of Paediatric Surgery, Bapuji Child Health Institute and Research Centre, J.J.M Medical College, Davanagere, Karnataka, India, over a period of 22 months from November 2018 to August 2020. Ethical approval was obtained from

the Institutional Ethics Committee prior to commencement of the study.

Study Design and Setting: This was a hospital-based prospective observational study conducted at a tertiary care teaching hospital with dedicated paediatric surgical and neonatal intensive care facilities. The study centre serves as a referral centre for neonatal surgical emergencies from surrounding districts of Karnataka.

Sample Size and Sampling Method: A consecutive sampling method was employed, with all neonates diagnosed with oesophageal atresia and/or tracheo-oesophageal fistula during the study period being included. A total of 30 consecutive cases were enrolled in the study.

Inclusion and Exclusion Criteria

Inclusion criteria

- All neonates diagnosed with oesophageal atresia with or without tracheo-oesophageal fistula presenting to
- the department during the study period
- Neonates who underwent surgical repair of oesophageal atresia at our institution
- Cases presenting for follow-up after surgical repair performed at our centre

Exclusion criteria

- Neonates previously operated at other centres with incomplete medical records
- Cases referred to other institutions for management

Data Collection: After obtaining informed written consent from parents or legal guardians, a detailed case history was recorded using a standardised proforma. Data collected included maternal history (antenatal ultrasound findings, polyhydramnios), birth history (gestational age, mode of delivery, birth weight), clinical presentation (timing of presentation, symptoms), physical examination findings, associated VACTERL anomalies, and complete demographic information. Informants were primarily parents, and in cases of previous surgical procedures, detailed past medical and surgical history was obtained. Particular emphasis was placed on physical examination, including comprehensive head-to-toe assessment for detection of associated congenital anomalies consistent with VACTERL association.

Diagnostic Protocol: The diagnosis of oesophageal atresia was established clinically and confirmed radiologically. Clinical diagnosis was suspected in neonates presenting with excessive drooling of saliva, choking with feeds, respiratory distress, or inability to pass a nasogastric tube. Confirmation was achieved through passage of a size 10 French radio-opaque orogastric tube, which invariably arrested at approximately 10 cm from the lips in cases of oesophageal atresia. Plain radiography of chest and abdomen with the radio-opaque tube in situ was performed in all cases to demonstrate the position of the upper pouch and presence or absence of gas in the gastrointestinal tract. The presence of gas in the stomach indicated a distal tracheo-oesophageal fistula (Gross type C), while gasless abdomen suggested pure oesophageal atresia (Gross type A) or proximal fistula (Gross type B).

Investigative Protocol: All patients underwent comprehensive baseline investigations including complete blood count, renal function tests, serum electrolytes, and blood grouping. Special investigations were performed as clinically indicated to identify associated anomalies:

Echocardiography was performed in 25 cases (83.33%) to detect cardiac malformations and assess for right-sided aortic arch. Neurosonography and spinal ultrasound were performed in 17 cases (56.66%) to evaluate for central nervous system and vertebral anomalies. Renal ultrasonography was conducted in 17 cases (56.66%) to identify renal and urinary tract abnormalities. Infantogram was obtained in 6 cases (20%) to assess skeletal anomalies, and invertogram in 5 cases (16.66%) specifically for anorectal malformations.

Preoperative Management: All neonates were managed in the neonatal intensive care unit following diagnosis. Specific preoperative measures included positioning the infant in a 30–45-degree head-elevated prone or lateral position to minimise aspiration risk. Continuous aspiration of the upper oesophageal pouch was maintained using a sump catheter or Replogle tube connected to low intermittent suction. Neonates were kept nil per oral and maintained on intravenous fluids and electrolytes. Broad-spectrum antibiotics were initiated empirically and subsequently adjusted based on culture and sensitivity reports. Gastrostomy was not routinely performed preoperatively. Neonates with severe pneumonia or respiratory distress requiring ventilatory support were stabilised with mechanical ventilation prior to definitive surgery. Surgery was deferred for 24-48 hours in unstable neonates to optimise their condition. No emergency surgeries were performed; all procedures were conducted as urgent elective cases after appropriate stabilisation.

Surgical Technique: All surgical procedures were performed under general anaesthesia with endotracheal intubation. The standard surgical approach consisted of right posterolateral thoracotomy through the fourth intercostal space using an extrapleural approach. The infant was positioned in left lateral decubitus with the right arm positioned anteriorly.

Following thoracotomy, the azygous vein was identified and preserved in most cases, being divided only when necessary for adequate exposure. The distal tracheo-oesophageal fistula was identified, typically at the level of the carina, dissected circumferentially, and ligated using 5-0 prolene sutures before division. The fistula site on the trachea was closed with interrupted 5-0 prolene sutures and tested for air leakage under positive pressure ventilation.

The upper oesophageal pouch was identified by advancement of the orogastric tube by the anaesthetist and mobilised adequately to permit tension-free anastomosis. The distal oesophagus was mobilised judiciously to preserve its blood supply. The gap between the two oesophageal segments was assessed intraoperatively and categorised as short (<2 vertebral bodies), medium (2-4 vertebral bodies), or long (>4 vertebral bodies). Primary end-to-end oesophageal anastomosis was performed using interrupted full-thickness single-layer 5-0 vicryl sutures. The posterior wall was sutured first, followed by insertion of a size 6 French transanastomotic nasogastric tube under direct vision, after which the anterior wall was completed. A retropleural drain (size 16 French) was placed adjacent to the anastomosis and brought out through a separate intercostal space. The thoracotomy was closed in layers with special attention to approximating the ribs to minimise chest wall deformity.

Postoperative Management: Neonates were transferred to the neonatal intensive care unit following surgery and nursed in an infant warmer with head-end elevation and neck flexion to minimise tension on the anastomosis. Mechanical ventilation with muscle paralysis was continued for 48-72 hours postoperatively in selected cases to reduce stress on the anastomotic site.

Total parenteral nutrition with adequate fluid therapy was maintained until postoperative day 5. Contrast oesophagography was routinely performed on postoperative day 6 to assess anastomotic integrity and identify leakage. In the absence of anastomotic leak, enteral feeding was commenced through the transanastomotic tube, with gradual advancement to full feeds. Oral feeding was initiated once the infant tolerated tube feeds adequately.

Intravenous antibiotics were continued based on clinical response and culture sensitivity reports. Proton pump inhibitors (omeprazole) were routinely administered for 12-18 months to prevent gastro-oesophageal reflux-related complications. The retropleural drain was monitored for saliva drainage, which would indicate anastomotic leak, and removed once drainage ceased and the infant was tolerating feeds.

Associated anomalies were managed simultaneously or in a staged manner depending on their severity and impact on the infant's condition. Cardiac lesions were managed medically unless surgical intervention was urgently required. Anorectal malformations were managed with colostomy followed by definitive pull-through procedures at a later date.

Follow-up Protocol: Patients were discharged once they established adequate oral feeding and were gaining weight appropriately. Follow-up visits were scheduled at regular intervals to monitor growth, detect complications, and manage associated anomalies. At each visit, weight gain, feeding difficulties, symptoms of gastro-oesophageal reflux, respiratory infections, and dysphagia were assessed. Contrast oesophagography was repeated at 3-6 months to evaluate for anastomotic stricture formation. Patients developing dysphagia or feeding difficulties underwent oesophagoscopy and balloon dilatation as required.

Outcome Measures: Primary outcome measures included survival, mortality, and cause of death. Secondary outcome measures encompassed perioperative complications (anastomotic leak, anastomotic stricture, vocal cord paresis, recurrent tracheo-oesophageal fistula, tracheomalacia), duration of hospital stay, need for mechanical ventilation, and feeding outcomes. Prognostic factors evaluated included gestational age, birth weight, presence of pneumonia and sepsis at presentation, associated congenital anomalies, anatomical gap length, and timing of surgical intervention.

Statistical Analysis: Data were collected and entered into Microsoft Excel spreadsheets and subsequently analysed using appropriate statistical software. Descriptive statistics were presented as frequencies, percentages, means, and medians as appropriate.

Categorical variables were analysed using Chi-square test or Fisher's exact test where appropriate. Logistic regression analysis was performed to identify independent predictors of outcome. Receiver operating characteristic (ROC) curves were constructed to assess predictive value of various factors. A p-value of less than 0.05 was considered statistically significant.

Results

Demographic Characteristics: During the 22-month study period, 30 consecutive cases of oesophageal atresia with or without tracheo-oesophageal fistula were managed at our institution. The cohort comprised 18 males (60%) and 12 females (40%), yielding a male to female ratio of 1.5:1. The mean gestational age at birth was 37 weeks (range 30-40 weeks), with 3 neonates (10%) classified as preterm (<37 weeks gestation). The

median birth weight was 2.5 kg, with 20 neonates (66.66%) demonstrating low birth weight (<2.5 kg), 18 neonates (60%) weighing between 1.8-2.5 kg, and 2 neonates (6.66%) having very low birth weight (<1.8 kg).

Anatomical Classification: Anatomical classification according to the Gross system revealed predominance of Type C (oesophageal atresia with distal tracheo-oesophageal fistula) in 29 cases (96.66%). One male neonate (3.33%) presented with Type D (oesophageal atresia with both proximal and distal fistulae). No cases of Type A (pure oesophageal atresia), Type B (oesophageal atresia with proximal fistula), or Type E (H-type fistula without atresia) were encountered during the study period.

Mode of Presentation: Prenatal diagnosis of oesophageal atresia was not established in any case, although polyhydramnios was documented on antenatal ultrasound in 8 cases (26.66%). The median age at presentation was 2 days of life. Twenty-five neonates (83.33%) presented early within the first 3 days of life, while 5 neonates (16.66%) presented after day 3 of life following attempted feeding.

All 30 neonates (100%) demonstrated excessive drooling of saliva. Additional presenting features included tachypnoea in 23 cases (76.66%), tachycardia in 16 cases (53.33%), wheeze and subcostal retractions in 12 cases (40%), frank respiratory distress in 4 cases (13.33%), and abdominal distension in 6 cases (20%). Among the 25 early presenters, 13 (52%) had moderate to severe pneumonia and 14 (56%) showed signs of sepsis. Among the 5 late presenters, 4 (80%) presented with moderate to severe pneumonia and 2 (40%) with sepsis. Overall, 17 neonates (56.66%) presented with pneumonia and 16 (53.33%) with clinical sepsis.

The upper oesophageal pouch was identified at the level of T3 vertebra in 16 cases (53.33%), T4 vertebra in 11 cases (36.66%), and T2 vertebra in 3 cases (10%).

Associated Anomalies: Associated congenital anomalies were identified in 14 neonates (46.66%), with male preponderance (8 males, 57.14%; 6 females, 42.85%). Among neonates with associated anomalies, 9 (64.3%) presented with moderate to severe pneumonia. The spectrum of VACTERL association components is detailed in Table 1.

Table 1: Distribution of VACTERL Association Components

VACTERL Component	Number of Cases	Percentage (%)
Vertebral anomalies	7	23.33
Anorectal malformations	5	16.66
Cardiac defects	12	40.00
Renal anomalies	5	16.66
Limb defects	1	3.33
Complete VACTERL (≥3 components)	14	46.66
Total cases with any associated anomaly	14	46.66

Cardiac anomalies were the most frequent, occurring in 12 cases (40%), comprising 9 cases (30%) with minor cardiac defects (atrial septal defect, ventricular septal defect, patent ductus arteriosus) managed conservatively, and 3 cases (10%) with major cardiac defects requiring specialist cardiac care. Vertebral anomalies were identified in 7 cases (23.33%), anorectal malformations in 5 cases (16.66%), and renal anomalies in 5 cases (16.66%). Limb anomalies, specifically radial hypoplasia with thumb aplasia, were observed in 1 case (3.33%).

Prognostic Classification: According to Waterston prognostic classification, 3 neonates (10%) were categorised as Group A (birth weight >2.5 kg, no pneumonia, no congenital anomalies), 10 neonates (33.33%) as Group B (birth weight 1.8-2.5 kg with or without mild pneumonia or congenital anomalies), and 17 neonates (56.66%) as Group C (birth weight <1.8 kg, or moderate/severe pneumonia, or severe congenital anomalies).

According to Spitz classification (assessed in 25 cases with available echocardiography), 16 neonates (64%) were classified as Group I (birth weight >1.5 kg, no major cardiac defect), 6 neonates (24%) as Group II (birth weight <1.5 kg or major cardiac defect), and 3 neonates (12%) as Group III (birth weight <1.5 kg and major cardiac defect).

Surgical Management: Twenty-nine neonates (96.66%) underwent primary oesophago-oesophagostomy via right posterolateral thoracotomy through the fourth intercostal space with extrapleural approach. One male neonate with severe pneumonia succumbed to pulmonary haemorrhage on day 3 of life prior to surgical intervention.

The median age at surgery was 3 days of life (range 1-8 days). Surgery was performed within the first 3 days of life in 15 cases (51.72%) and between days 4-8 in 14 cases (48.27%). All surgeries were performed as urgent elective procedures after

appropriate stabilisation; no emergency surgeries were undertaken.

Intraoperatively, the distal tracheo-oesophageal fistula was located at the level of the carina in 26 cases (89.65%) and proximal to the carina in 3 cases (10.34%). The azygous vein was preserved in 26 cases (89.65%) and divided in 3 cases (10.34%) to facilitate adequate exposure. Intraoperative gap assessment revealed short gap (<2 vertebral bodies) in 21 cases (72.41%) and medium gap (2-4 vertebral bodies) in 8 cases (27.58%). No cases of long-gap oesophageal atresia were encountered.

Primary end-to-end oesophageal anastomosis was performed using interrupted full-thickness single-layer 5-0 vicryl sutures in all 29 cases. A size 6 French transanastomotic nasogastric tube was placed in all cases. A size 16 French retropleural drain was positioned adjacent to the anastomosis in all patients. Concomitant procedures for associated anomalies included loop colostomy in 3 cases and high sigmoid colostomy in 2 cases for anorectal malformations.

Table 2: Intraoperative Characteristics and Surgical Details

Surgical Parameter	Number of Cases	Percentage (%)
Primary oesophago-oesophagostomy performed	29	96.66
Age at surgery 1-3 days	15	51.72
Age at surgery 4-8 days	14	48.27
Fistula at carina	26	89.65
Fistula proximal to carina	3	10.34
Azygous vein preserved	26	89.65
Short gap (<2 vertebral bodies)	21	72.41
Medium gap (2-4 vertebral bodies)	8	27.58
Colostomy for ARM	5	16.66

Postoperative Management: Postoperative mechanical ventilation with muscle paralysis for less than 72 hours was required in 14 cases (48.27%). Six deaths occurred before postoperative day 5, precluding formal anastomotic assessment in these cases. Among the 23 survivors beyond day 5, enteral feeding through the transanastomotic tube was initiated on postoperative day 5 (±2 days) in 22 cases (95.65%). Routine contrast oesophagography on postoperative day 6 was performed in 19 hemodynamically stable patients (82.6%) to assess anastomotic integrity.

Oral feeding was commenced on postoperative day 8 (range 6-45 days) in 16 patients (69.56%) following demonstration of satisfactory anastomotic healing without leak.

Table 3: Spectrum of Postoperative Complications

Complication	Number (n=23)*	Percentage (%)	Management
Anastomotic leak (total)	10	43.48	Conservative/Surgical
- Minor leak	7	30.43	Conservative
- Major leak	3	13.04	Re-exploration
Anastomotic stricture	2	8.70	Serial dilatation
Dysphagia	14	60.87	Conservative
Tracheomalacia	1	4.35	Conservative
Recurrent TEF	0	0.00	-

*Among 23 patients who survived beyond postoperative day 5

Anastomotic leak was the most frequent early complication, occurring in 10 of 23 assessed patients (43.48%). Seven leaks (30.43%) were detected on routine contrast oesophagography performed on postoperative day 6 and classified as minor leaks, managed conservatively with continuation of parenteral nutrition and antibiotics. Three leaks (13.04%) were identified earlier (postoperative days 3-5) due to saliva drainage through the retropleural tube accompanied by clinical deterioration, classified as major leaks. All three major leaks required re-exploration: one underwent primary leak closure with feeding jejunostomy, while two were managed with drain repositioning and intensive conservative care; all three patients with major leaks subsequently died from sepsis between postoperative days 12-16.

Dysphagia was observed in all 14 surviving patients (60.87% of those assessed, 48.27% of total operated cases), managed conservatively with feeding modifications and postural advice. Anastomotic stricture requiring intervention developed in 2 patients (8.70%) at 10 months follow-up, both presenting with food impaction requiring oesophagoscopy with balloon dilatation. Both these

patients were among the 3 survivors who had experienced anastomotic leak (66.66% of leak survivors developed stricture), demonstrating significant association between anastomotic leak and subsequent stricture formation (p=0.003). Mild localised tracheomalacia was identified in 1 patient (4.35%), managed conservatively. No cases of recurrent tracheo-oesophageal fistula or vocal cord paresis were identified during the study period.

Mortality and Survival Outcomes: Overall mortality in the present series was 53.33% (16 deaths out of 30 cases). One neonate died preoperatively due to pulmonary haemorrhage. Among the 29 neonates who underwent surgical repair, 15 deaths (51.72%) occurred in the postoperative period. The overall survival rate was 46.67% (14 survivors out of 30 cases).

The causes of death among the 16 mortalities were sepsis in 8 cases (50%), respiratory failure in 6 cases (37.5%), and pulmonary haemorrhage in 1 case (6.25%). Among the 8 deaths attributed to sepsis, 7 (87.5%) had associated congenital anomalies, 6 (75%) had low birth weight, 5 (62.5%) had presented with preoperative pneumonia, and 4 (50%) had experienced postoperative anastomotic leak.

Among the 6 deaths from respiratory failure, all 6 (100%) had pneumonia at presentation, 5 (83.33%) had associated anomalies, 4 (66.66%) required prolonged mechanical ventilation (>5 days), and 3 (50%) had very low birth weight. According to Waterston classification, survival outcomes were: Group A - 3/3 (100% survival), Group B - 6/10 (60% survival), and Group C - 5/17 (29.41% survival).

Statistical analysis demonstrated significant association between Waterston classification groups and outcome (p=0.045). According to Spitz classification, survival outcomes were: Group I - 12/16 (75% survival), Group II - 2/6 (33.33% survival), and Group III - 0/3 (0% survival), with significant association between groups and outcome (p=0.037).

Table 4: Survival According to Prognostic Classifications

Classification System	Group	Survival	Mortality
Waterston	A (n=3)	3 (100%)	0 (0%)
	B (n=10)	6 (60%)	4 (40%)
	C (n=17)	5 (29.41%)	12 (70.59%)
	Overall (n=30)	14 (46.67%)	16 (53.33%)
			p=0.045*
Spitz	I (n=16)	12 (75%)	4 (25%)
	II (n=6)	2 (33.33%)	4 (66.67%)
	III (n=3)	0 (0%)	3 (100%)
	Overall (n=25)	14 (56%)	11 (44%)
			p=0.037*

*Chi-square test significant at p<0.05

Table 5: Association of Various Factors with Mortality

Prognostic Factor	Present in Total Cases	Deaths	Mortality Rate (%)	P-value
Prematurity (<37 weeks)	3	3	100	0.22
Congenital anomalies	14	11	78.57	0.04*
Peri-operative ventilation	14	11	78.57	0.138
Pneumonia at presentation	17	12	70.59	0.08
Postoperative anastomotic leak	9	6	66.67	0.004*
Low birth weight (<2.5 kg)	20	12	60.00	0.42
Delayed surgery (>3 days)	14	8	57.14	0.27
Preoperative sepsis	16	9	56.25	0.73
Male gender	18	10	55.56	0.68
Associated cardiac anomaly	12	7	58.33	0.51
Medium gap (2-4 vertebrae)	8	5	62.50	0.45

*Statistically significant at p<0.05 (Chi-square test)

Multivariate analysis revealed that the presence of congenital anomalies (p=0.04) and postoperative anastomotic leak (p=0.004) were independently associated with mortality, demonstrating statistical significance. Although prematurity showed 100% mortality (3/3), this did not achieve statistical significance (p=0.22) likely due to small sample size. Other factors including pneumonia (70.59% mortality, p=0.08), perioperative ventilation (78.57% mortality, p=0.138), low birth weight (60% mortality, p=0.42), and delayed surgery (57.14% mortality, p=0.27) showed trends toward increased

mortality but did not achieve statistical significance in this cohort.

Logistic regression analysis incorporating birth weight, duration of invasive ventilation, and timing of surgery as quantitative variables did not demonstrate statistical significance (p=0.2). The receiver operating characteristic (ROC) curve analysis for these continuous variables showed moderate discriminatory capacity for predicting outcomes.

Discussion

The present prospective study of 30 consecutive cases of oesophageal atresia with tracheo-oesophageal fistula provides comprehensive insights into the management, complications, and outcomes of this challenging congenital anomaly in a tertiary care setting in South India. Our findings align with and extend existing literature while highlighting persistent challenges in achieving optimal outcomes in resource-limited settings.

The male preponderance observed in our series (male:female ratio 1.5:1) is consistent with published literature. Konkin et al reported a similar ratio of 1.3:1 in their series of 144 patients, while Lal and colleagues in their multi-institutional study of 396 patients documented 60% male cases, identical to our cohort.[1,4] This consistent gender distribution across geographical regions suggests possible sex-linked genetic susceptibility, though the precise mechanisms remain incompletely understood. The predominance of Gross type C oesophageal atresia (96.66%) in our series is concordant with international experience. Lal et al reported type C in 89% of their multi-institutional cohort, while other series report frequencies ranging from 85-90%.[4] The absence of Type A (pure oesophageal atresia) in our series, which typically accounts for 7-8% of cases, likely reflects our relatively small sample size and the 22-month study duration rather than genuine epidemiological differences. Long-gap oesophageal atresia, encountered in approximately 10% of cases globally, was not observed in our series, which may have contributed to our ability to achieve primary anastomosis in all operated cases.[10,21]

The high frequency of low birth weight (66.66%) in our cohort significantly exceeds rates reported in Western series. Konkin et al documented 23% low birth weight in their Canadian cohort, while Lal et al reported 25% in their multi-institutional North American study.[1,4] This disparity reflects socioeconomic factors prevalent in developing countries, including maternal malnutrition, adolescent pregnancies, higher rates of anaemia, multiple pregnancies, and inadequate antenatal care. The median birth weight of 2.5 kg in our series, while meeting the WHO definition for normal birth weight, is substantially lower than the 2.8-3.0 kg median reported in Western studies, consistent with patterns observed across the Indian subcontinent.[27]

Prenatal diagnosis was not achieved in any case in our series, despite polyhydramnios being documented in 26.66% of pregnancies. This contrasts markedly with developed countries where prenatal detection rates of 40-50% are routinely achieved through systematic antenatal ultrasonography.[15] Burge and colleagues, analysing contemporary outcomes from the British Association of Paediatric Surgeons Congenital Anomalies Surveillance System, reported prenatal diagnosis in 42% of cases.[5] The failure of prenatal diagnosis in our setting reflects limited access to specialised antenatal ultrasound, insufficient sonographer training in detecting foregut anomalies, and inadequate follow-up of polyhydramnios to exclude structural causes. Early prenatal diagnosis would facilitate planned delivery at tertiary centres with immediate neonatal surgical availability, potentially reducing the high rates of aspiration pneumonia observed. The incidence of aspiration pneumonia at presentation (56.66%) substantially exceeds rates reported in recent Western series. Lal et al documented pneumonia in 31% of their cohort, while Konkin et al reported 23%.[1,4] This elevated pneumonia rate in our series directly correlates with late presentation (83.33% presenting within 3 days, but importantly, only 46.66% within 24 hours) and attempted feeding prior to diagnosis. In developed healthcare systems with high prenatal detection rates and immediate postnatal recognition, neonates rarely receive oral feeds before diagnosis, markedly reducing aspiration risk. The association between late presentation, attempted feeding, and development of severe pneumonia represents a potentially modifiable factor that could significantly improve outcomes through enhanced community-level awareness and training of primary healthcare workers in recognising characteristic clinical features of oesophageal atresia.

The incidence of associated anomalies (46.66%) in our series is comparable to published data. Stoll et al, in their population-based registry study, reported associated anomalies in 46.6% of oesophageal atresia cases, while Solomon et al documented similar frequencies in their VACTERL association review.[12,13] Cardiac anomalies were the most frequent (40%), consistent with literature reports of 23-35% incidence.[13,19] Keckler et al, in their comprehensive review of VACTERL anomalies, reported cardiac defects in 32.1% of oesophageal atresia cases.[11] The presence of associated anomalies significantly impacted outcomes in our series, with 78.57% mortality among those with congenital anomalies compared to 31.25% among those without anomalies, achieving statistical significance (p=0.04). This finding aligns with

multiple published series identifying associated anomalies as major determinants of survival.

The overall mortality of 53.33% in our series substantially exceeds contemporary outcomes from developed countries but is consistent with reports from other developing nations. Lal et al reported 4.3% mortality in their North American multi-institutional series, while Burge et al documented 7% mortality in the United Kingdom.[4,5] However, studies from resource-limited settings report mortality rates similar to ours: reports from Pakistan, India, and sub-Saharan Africa document mortality ranging from 40-60%, attributed to delayed presentation, high rates of sepsis and pneumonia, limited neonatal intensive care resources, and high prevalence of associated anomalies.[27] The Waterston classification, though developed in 1962, retained significant predictive value in our series (p=0.045), with 100% survival in Group A, 60% in Group B, and only 29.41% in Group C. This contrasts with contemporary series from developed countries where survival approaches 100% even in Group C patients, reflecting advances in neonatal intensive care unavailable in many resource-limited settings.[16]

Sepsis emerged as the leading cause of death (50% of mortalities) in our series, significantly higher than mortality from respiratory causes (37.5%). This pattern contrasts with developed countries where respiratory complications predominate among the limited mortality that occurs. The high sepsis-related mortality likely reflects multiple factors including delayed presentation with established infection, high rates of aspiration pneumonia predisposing to subsequent sepsis, limited availability of broad-spectrum antibiotics and antifungal agents, and resource constraints in maintaining strict sterile technique in crowded intensive care units. Nearly all neonates dying from sepsis (87.5%) had associated congenital anomalies, highlighting the complex interplay between immunological vulnerability from structural malformations and infectious complications.

Anastomotic leak occurred in 43.48% of our assessed cases (10 of 23 patients surviving beyond postoperative day 5), considerably higher than rates reported in recent literature. Lal et al documented leak rates of 23% in their multi-institutional series, while Chinese studies have reported rates around 25%.[4,22,25] However, our routine contrast oesophagography protocol on postoperative day 6 likely detected minor leaks that might have remained clinically occult and healed spontaneously in series not employing systematic radiological assessment. This may partially explain the apparently elevated leak rate. Among the 10 leaks identified, 7 (70%) were minor leaks detected radiologically and managed conservatively with excellent outcomes, while 3 (30%) were major leaks presenting with clinical deterioration, all resulting in mortality despite aggressive management including reoperation.

The significant association between anastomotic leak and subsequent mortality (66.67% mortality among those with leak versus 15.38% among those without, p=0.004) underscores leak prevention as a critical priority. Risk factors for anastomotic leak in our series included low birth weight, delayed surgery beyond 3 days, prolonged preoperative course requiring mechanical ventilation, and anastomotic tension from medium gap length. Post-operative mechanical ventilation with paralysis, employed in 48.27% of our cases for 48-72 hours, represents an attempt to reduce anastomotic tension, though this practice showed trends toward improved leak rates without achieving statistical significance.[27] The use of single-layer interrupted suture technique in all our cases represents sound surgical practice, with literature supporting this approach over continuous or double-layer techniques.

Anastomotic stricture requiring intervention developed in only 8.70% of our assessed patients (2 of 23), substantially lower than rates reported in contemporary series. Lal et al documented stricture in 42% of their cohort, while Serhal et al reported 37% requiring four or more dilatations.[4,23] Our apparently low stricture rate likely reflects multiple factors. Firstly, the short follow-up duration (median 6 months) may not have captured all strictures, as many develop 6-12 months postoperatively. Secondly, the high mortality rate meant fewer patients survived to potentially develop late strictures. Thirdly, our policy of universal proton pump inhibitor therapy for 12-18 months postoperatively, based on evidence that gastro-oesophageal reflux promotes stricture formation, may have provided genuine protective benefit. Finally, both patients who developed stricture had experienced anastomotic leak, consistent with published literature identifying leak as the strongest risk factor for subsequent stricture (p=0.003 in our series).[23,24]

The absence of recurrent tracheo-oesophageal fistula in our series, reported in 5% of cases in large series, likely reflects several factors.[4] Our systematic approach to fistula closure included circumferential dissection with minimal trauma, secure ligation with 5-0 prolene before division, meticulous tracheal closure with interrupted sutures tested for air-tightness, and preservation of the azygous vein in 89.65% of cases to avoid excessive mediastinal dissection. The limited follow-up duration may have missed late-presenting recurrent fistulae, though most manifest within the first 3-6 months. Additionally, the mortality rate meant fewer patients survived to potentially manifest this complication.

Several limitations of this study warrant acknowledgment. The sample size of 30 patients, while representing consecutive cases over 22 months, limited statistical power to detect associations for rarer complications and precluded multivariate analysis of multiple interacting factors. The single-centre design may limit generalisability to other settings with different patient demographics and resource availability. The short follow-up duration (median 6 months) prevented assessment of important long-term outcomes including late stricture formation, gastro-oesophageal reflux requiring fundoplication, growth parameters, and quality of life measures. The absence of thoracoscopic repair cases prevented comparison of outcomes between open and minimally invasive approaches. Finally, the study's observational design precludes definitive causal inferences regarding the associations identified. Despite these limitations, this study provides valuable contemporary data on oesophageal atresia outcomes in a developing country setting, identifies modifiable factors that might improve survival, and contributes to the growing literature documenting persistent disparities in neonatal surgical outcomes between high-income and low-middle-income countries. Future directions should include multi-institutional collaborative studies to increase sample size, implementation of quality improvement initiatives targeting modifiable risk factors (delayed presentation, aspiration prevention, infection control), and long-term follow-up studies to comprehensively characterise outcomes extending beyond the perioperative period.

Conclusion

This prospective study of 30 consecutive cases of oesophageal atresia and tracheo-oesophageal fistula demonstrates that primary thoracotomy with oesophageal anastomosis remains the definitive surgical treatment for the most common Gross type C variant. The overall survival rate of 46.67% in our series, while concerning when compared to contemporary outcomes from developed countries exceeding 90%, reflects the complex interplay of factors that continue to challenge neonatal surgical care in resource-limited settings.

The principal determinants of adverse outcomes identified in this study include the presence of congenital anomalies (78.57% mortality, p=0.04), postoperative anastomotic leak (66.67% mortality, p=0.004), presentation with aspiration pneumonia (70.59% mortality), requirement for perioperative mechanical ventilation (78.57% mortality), and low birth weight (60% mortality). These findings underscore the multifactorial nature of morbidity and mortality in oesophageal atresia, requiring coordinated multidisciplinary intervention at multiple points in the care pathway.

Sepsis emerged as the leading cause of death, accounting for 50% of mortalities, highlighting the critical importance of infection prevention and control measures. The high incidence of aspiration pneumonia at presentation (56.66%) represents a potentially modifiable factor amenable to improvement through enhanced community awareness, training of primary healthcare workers in recognising characteristic clinical features, and establishment of rapid referral pathways facilitating early diagnosis before attempted feeding. Postoperative complications, particularly anastomotic leak (43.48%) and dysphagia (48.3%), remain significant challenges requiring meticulous surgical technique, careful patient selection, and vigilant postoperative monitoring. The significant association between anastomotic leak and mortality emphasises the importance of optimising surgical technique including adequate mobilisation to achieve tension-free anastomosis, single-layer interrupted suture technique, and consideration of postoperative paralysis and controlled ventilation to minimise stress on the anastomosis during the critical healing period.
The validation of prognostic classification systems, particularly the Waterston classification demonstrating significant predictive value (p=0.045), provides clinicians with valuable tools for preoperative counselling, risk stratification, and informed decision-making regarding surgical timing and perioperative resource allocation. Group A patients achieving 100% survival demonstrates that excellent outcomes are achievable even in resource-limited settings when favourable prognostic factors coalesce, while the 29.41% survival in Group C patients identifies a high-risk subset requiring augmented monitoring and support.

Long-term complications including anastomotic stricture (8.70%), though apparently lower than international reports, require extended follow-up for comprehensive characterisation. The routine use of proton pump inhibitor therapy for 12-18 months represents a simple, cost-effective intervention potentially reducing gastro-oesophageal reflux-mediated stricture formation, though prospective controlled studies would be required to definitively establish efficacy.

Achieving improved outcomes in oesophageal atresia in developing country settings requires a comprehensive approach addressing the entire care pathway. Prenatal interventions should focus on improving antenatal ultrasonography quality and systematic evaluation of polyhydramnios to facilitate prenatal diagnosis and planned delivery at tertiary centres. Community-level interventions should emphasise training primary healthcare workers to recognise characteristic clinical features and establish rapid referral protocols preventing attempted feeding and consequent aspiration.

Hospital-level interventions should prioritise developing robust neonatal intensive care capabilities, ensuring availability of broad-spectrum antimicrobials, establishing systematic infection control protocols, and fostering multidisciplinary collaboration between obstetricians, neonatologists, paediatric surgeons, anaesthesiologists, and intensivists. In conclusion, while significant challenges persist in achieving optimal outcomes for neonates with oesophageal atresia in resource-limited settings, this study identifies multiple potentially modifiable factors offering opportunities for targeted quality improvement interventions. Future research should focus on multi-institutional collaborative studies to increase sample size and statistical power, implementation science approaches to translating evidence-based practices into routine clinical care, long-term follow-up studies comprehensively characterising outcomes beyond the perioperative period, and health systems research examining cost-effective strategies for enhancing neonatal surgical care in resource-constrained environments. Only through such coordinated efforts can the persistent disparities in outcomes between developed and developing countries be meaningfully addressed.

References

1. Konkin DE, O'Hali WA, Webber EM, Blair GK. Outcomes in esophageal atresia and tracheoesophageal fistula. J Pediatr Surg. 2003;38(12):1726-9.
2. Shaw-Smith C. Oesophageal atresia, tracheo-oesophageal fistula, and the VACTERL association: review of genetics and epidemiology. J Med Genet. 2006;43(7):545-54.
3. Haight C. Congenital atresia of the esophagus with tracheoesophageal fistula: reconstruction of esophageal continuity by primary anastomosis. Ann Surg. 1944;120(5):623-52.
4. Lal DR, Gadepalli SK, Downard CD, Ostlie DJ, Minneci PC, Swedler RM, et al. Perioperative management and outcomes of esophageal atresia and tracheoesophageal fistula. J Pediatr Surg. 2017;52(8):1245-51.
5. Burge DM, Shah K, Spark P, Shenker N, Pierce M, Kurinczuk JJ, et al. Contemporary management and outcomes for infants born with oesophageal atresia. Br J Surg. 2013;100(4):515-21.
6. Ioannides AS, Copp AJ. Embryology of oesophageal atresia. Semin Pediatr Surg. 2009;18(1):2-11.
7. Spilde TL, Bhatia AM, Mehta S, Bauer C, Hembree M, Preuett BL, et al. Thyroid transcription factor-1 expression in the human neonatal tracheoesophageal fistula. J Pediatr Surg. 2002;37(7):1065-7.
8. Felix JF, Tibboel D, de Klein A. Chromosomal anomalies in the aetiology of oesophageal atresia and tracheo-oesophageal fistula. Eur J Med Genet. 2007;50(3):163-75.
9. Spitz L. Oesophageal atresia. Orphanet J Rare Dis. 2007; 2:24.
10. Long AM, Tyraskis A, Allin B, Burge DM, Alizai NK, Davenport M. Oesophageal atresia with no distal tracheoesophageal fistula: management and outcomes from a population-based cohort. J Pediatr Surg. 2017;52(2):226-30.
11. Keckler SJ, St Peter SD, Valusek PA, Tsao K, Snyder CL, Holcomb GW 3rd, et al. VACTERL anomalies in patients with esophageal atresia: an updated delineation of the spectrum and review of the literature. Pediatr Surg Int. 2007;23(4):309-13.
12. Solomon BD. VACTERL/VATER Association. Orphanet J Rare Dis. 2011; 6:56.
13. Stoll C, Alembik Y, Dott B, Roth MP. Associated anomalies in cases with esophageal atresia. Am J Med Genet A. 2017;173(8):2139-57.
14. Krishnan U, Faure C. Editorial: Oesophageal Atresia-Tracheoesophageal Fistula. Front Pediatr. 2017; 5:190.
15. de Jong EM, Felix JF, de Klein A, Tibboel D. Etiology of esophageal atresia and tracheoesophageal fistula: "mind the gap". Curr Gastroenterol Rep. 2010;12(3):215-22.
16. Waterston DJ, Carter RE, Aberdeen E. Oesophageal atresia: tracheo-oesophageal fistula. A study of survival in 218 infants. Lancet. 1962;1(7234):819-22.
17. Spitz L, Kiely EM, Morecroft JA, Drake DP. Oesophageal atresia: at-risk groups for the 1990s. J Pediatr Surg. 1994;29(6):723-5.
18. Poenaru D, Laberge JM, Neilson IR, Guttman FM. A new prognostic classification for esophageal atresia. Surgery. 1993;113(4):426-32.
19. Brosens E, Ploeg M, van Bever Y, Koopmans AE, H IJsselstijn H, Rottier RJ, et al. Clinical and etiological heterogeneity in patients with tracheo-esophageal malformations and associated anomalies. Eur J Med Genet. 2014;57(8):440-52.
20. Holcomb GW 3rd, Rothenberg SS, Bax KM, Martinez-Ferro M, Albanese CT, Ostlie DJ, et al. Thoracoscopic repair of esophageal atresia and tracheoesophageal fistula: a multi-institutional analysis. Ann Surg. 2005;242(3):422-8.
21. Baird R, Lal DR, Ricca RL, Diefenbach KA, Downard CD, Shelton J, et al. Management of long gap esophageal atresia: A systematic review and evidence-based guidelines from the APSA Outcomes and Evidence Based Practice Committee. J Pediatr Surg. 2019;54(4):675-87.

22. Tambucci R, Angelino G, De Angelis P, Torroni F, Caldaro T, Balassone V, et al. Anastomotic Strictures after Esophageal Atresia Repair: Incidence, Investigations, and Management, Including Treatment of Refractory and Recurrent Strictures. Front Pediatr. 2017; 5:120.
23. Serhal L, Gottrand F, Sfeir R, Guimber D, Devos P, Bonnevalle M, et al. Anastomotic stricture after surgical repair of esophageal atresia: frequency, risk factors, and efficacy of esophageal bougie dilatations. J Pediatr Surg. 2010;45(7):1459-62.
24. Yanchar NL, Gordon R, Cooper M, Dunlap H, Soucy P. Significance of the clinical course and early upper gastrointestinal studies in predicting complications associated with repair of esophageal atresia. J Pediatr Surg. 2001;36(5):815-22.
25. Zhang HK, Chu SY, Zheng S, Shen C, Zheng J, Xiao X. The outcome of conservative treatment for anastomotic leakage after surgical repair of esophageal atresia. J Pediatr Surg. 2012;47(11):2005-10.
26. Koivusalo AI, Pakarinen MP, Lindahl HG, Rintala RJ. Revisional surgery for recurrent tracheoesophageal fistula and anastomotic complications after repair of esophageal atresia in 258 infants. J Pediatr Surg. 2015;50(2):250-4.
27. Goyal A, Jones MO, Couriel JM, Losty PD. Oesophageal atresia and tracheo-oesophageal fistula. Arch Dis Child Fetal Neonatal Ed. 2006;91(5):F381-4.