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REVIEW PAPER

Oesophageal atresia in newborns: diagnosis, treatment, and perioperative management: a systematic review

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ABSTRACT

Oesophageal atresia (OA) is one of the most common and severe congenital defects of the oesophagus, often accompanied by the occurrence of the tracheoesophageal fistula (TEF). The process of determining the risk factors and the direct causes that contribute to its development is not yet completely understood. It is considered that multiple genes play a significant role in promoting this phenomenon, with most cases leading to TEF formation. The authors specify the importance of prenatal and postnatal diagnosis to determine the presence of OA and promptly stabilise haemodynamically newborns, enabling surgery to be performed as soon as possible. The thoracoscopic method has been recognized as a routine procedure for the treatment of OA. Additionally, both preoperative and postoperative management are extremely crucial to prevent any further complications in newborns.

KEY WORDS:

thoracotomy, oesophageal atresia, thoracoscopy, perioperative care.

INTRODUCTION

Oesophageal atresia (OA) is one of the most prevalent congenital defects of the oesophagus, primarily characterised by its discontinuity (atresia), which may occur with or without the presence of a pathological fusion with the trachea, known as a tracheoesophageal fistula (TEF) [1, 2]. Recent statistics reported an overall European prevalence of 2.43 OA cases per 10,000 births. Furthermore, approximately 70–90% of congenital OA cases are accompanied by TEF. Of 1222 cases, the majority (88.7%) were live births, 3.5% were foetal deaths, and 7.8% ended in pregnancy termination [1, 3, 4].

In approximately 55% of cases, individuals born with OA have other functional impairments or comorbidities such as VACTERL association (10% of cases), CHARGE association (1% of cases), heart anomalies, growth retardation, genitourinary malformations, ear pathologies, and among chromosomal abnormalities: Edwards syndrome (6% of cases) or Down syndrome (< 3% of cases) [1]. Studies have shown that the presence of additional congenital syndromes or chromosomal pathologies increases the mortality rate, reducing survival rates to as low as 51%. In comparison, cases of isolated OA have demonstrated improved survival rates over the past decade, reaching 91–98% [4, 5]. However, despite the significant number of associated anomalies, the rate of pregnancy termination remains relatively low [3].

According to the Gross classification, six distinct types of OA depending on TEF presence and type can be identified, labelled A through F (Figure 1). The majority of cases involve TEF type C, accounting for 82–86% of cases [1, 2, 5].

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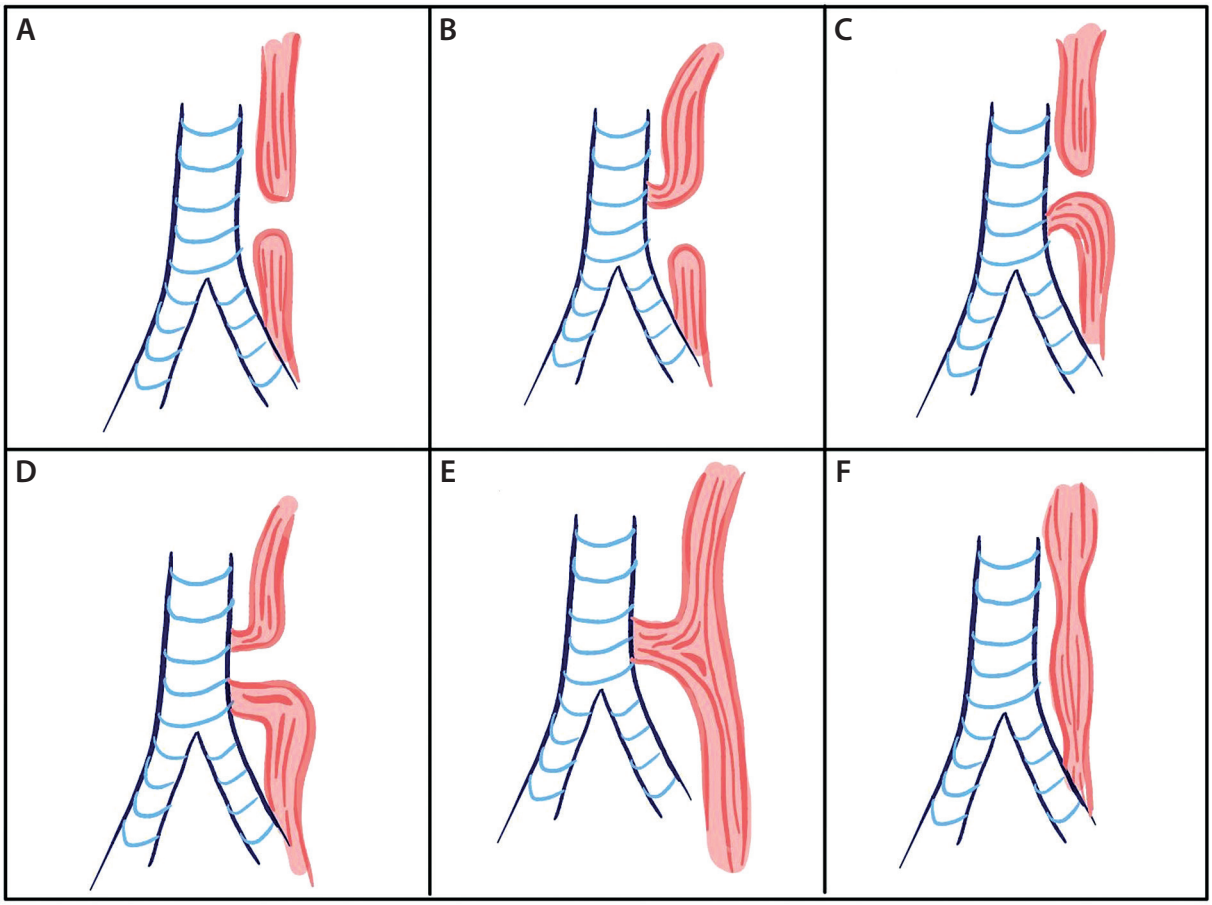

FIGURE 1. A) Oesophageal atresia (OA) without an associated fistula, with a blind proximal oesophagus, the distance between the two oesophageal pouches being 5–8 vertebrae, with a prevalence of 7%. **B**) OA with a proximal tracheoesophageal fistula (TEF) located in the posterior wall of the trachea, with a prevalence of 2%. **C**) OA with a distal TEF, the proximal end of the oesophagus is enlarged and dilated. **D**) EA with proximal and distal TEF not connected, with a prevalence of < 1%. **E**) Congenital pure TEF characterised by a proximal TEF and distal TEF that are connected, usually located at the level of the first thoracic vertebra within the upper thoracic outlet, with a prevalence of 4%. **F**) Distinguished by some authors, it is characterised by congenital oesophageal stenosis primarily affecting the distal oesophagus

Source: prepared based on [2]

AETIOLOGY AND RISK FACTORS

The aetiological cause of OA, with or without an associated TEF, is failure of the foregut to properly separate or fully develop [1, 6]. The underlying mechanisms remain unspecified and are considered multifactorial and polygenic. However, some studies on genetic embryo models in mice associate the abnormal expression of the *Sonic hedgehog* (*Shh*) gene with the pathogenesis of tracheoesophageal malformations. The Shh glycoprotein and its signalling pathway, including *Gli2*, *Gli3*, and *Foxf1* genes, play a crucial role in the formation of multiple organs during embryonic development. Therefore, mutations in the *Shh* and *FOX* genes may contribute to the failure of tracheoesophageal separation and play a key role in the development of OA and TEF in newborns [1, 7, 8].

Other studies have highlighted the role of Nkx2.1 (a respiratory marker) and SOX2 (a gastrointestinal marker) in foregut development. Their signalling pathways involve both the activation and inhibition of transcription, contributing to the dorsoventral patterning of the foregut. Any disruption in the specific expression patterns of Nkx2.1 and SOX2 can result in failure of tracheoesophageal separation [1]. Moreover, multiple other genes, such as *CHD7*, *MYCN*, *FANCB*, and the *Nrf2*/*Keap1* pathway, have been implicated in the development of OA [6, 9].

One of the most common risk factors for OA is the presence of chromosomal and genetic abnormalities. Among them, trisomy 21, trisomy 18, and deletion of chromosome 13q have been identified as significant contributors [7]. Additionally, VACTERL association (vertebral, anal, cardiac, tracheoesophageal, renal, and limb anomalies), CHARGE syndrome (coloboma, heart defects, choanal atresia, growth and developmental delay, genital hypoplasia, and ear abnormalities), Potter's syndrome (renal agenesis, lung underdevelopment, and distinctive facial features), and SCHISIS association (omphalocele, cleft lip and/or palate, and genital hypoplasia) are also considered significant risk factors for OA [1, 7]. Familial and syndromic cases of OA are exceptionally rare. However, the condition is two to three times more

frequent in twins, and the overall recurrence risk for siblings of an affected child is approximately 1% [7, 10].

Moreover, the number of pregnancies and maternal age also play a significant role. Women having their second or third child have over a 30% lower risk of OA compared to first-time mothers. Children born to mothers aged 35–40 years are twice as likely to develop OA, while those born to mothers over the age of 40 years face a threefold increased risk, compared to children born to mothers under 20 years old [11]. Furthermore, white women have a 60% higher risk of giving birth to a child with this congenital defect compared to women of other racial backgrounds [12].

Another factor that may influence the risk of OA in the foetus is maternal diabetes during pregnancy. Infants born to women with diabetes have a 70% greater risk of developing OA compared to those born to non-diabetic women [12, 13]. Oesophageal atresia may also be associated with maternal exposure to harmful internal or external factors during the early stages of pregnancy, such as medications (e.g. methimazole), exogenous sex hormones, infectious diseases, maternal phenylketonuria, or contact with herbicides and insecticides [12, 14, 15].

DIAGNOSIS

PRENATAL DIAGNOSIS

Prenatal screening in Europe identifies 40% of major structural defects [16]. Abdominal wall defects, with a prenatal detection rate of about 90%, are the most frequently identified birth defects [17]. However, only 10–40% of OA cases are detected prenatally. Additionally, 50% of cases are associated with genetic or chromosomal abnormalities, which may impact the overall prognosis [18]. The diagnosis is typically not made until the third trimester of pregnancy, and a high rate of false-positive results is observed [19].

It is challenging to examine the foetal oesophagus using prenatal ultrasound. Due to its small size and collapsed state, the foetal oesophagus is difficult to differentiate from other fluid-filled structures in the chest, such as the aorta and trachea, during prenatal imaging [20]. Prenatal ultrasound findings suggestive of OA most commonly include polyhydramnios, a small or absent foetal stomach, and the pouch sign (blind-ending dilated upper oesophageal pouch).

In recent years, magnetic resonance imaging (MRI) and amniotic fluid biochemistry have emerged as potential second-line diagnostic tools for confirming a prenatal diagnosis [18]. Magnetic resonance imaging is valuable because it accurately identifies the pouch sign [5, 21]. Moreover, a dynamic sequence provides better visualisation of the amniotic sac than a standard sequence, because it captures the moment of foetal swallowing [18]. In recent decades, many imaging sequences have been developed for data acquisition, employing different tissue contrasts and post-processing analyses [22].

In the case of OA, γ-glutamyl transpeptidase (GGTP) and α-fetoprotein (AFP) levels in amniotic fluid are elevated [23]. Nevertheless, the non-invasive nature of MRI is advantageous compared to AF analysis.

According to the last meta-analysis, the sensitivity of ultrasound was 31.7%, MRI was 94.7%, while the sensitivity of the AF index OA>3 (AFP multiplied by GGTP) was 89.9% [5].

The clear benefit of prenatal diagnosis is reduced risk of aspiration and optimised postnatal care by facilitating delivery at a specialised centre equipped for neonatal intensive care and paediatric surgery [19]. Furthermore, it provides an opportunity to counsel and prepare parents for the birth of an affected newborn [23].

POSTNATAL DIAGNOSIS

Symptoms in infants with OA typically appear shortly after birth [24]. Key clinical indicators for OA include hypersalivation, feeding difficulties characterised by choking and coughing, cyanosis, and respiratory distress. A common diagnostic marker for OA is the unsuccessful passage of an orogastric tube [24, 25]. Chest X-ray imaging will show the orogastric tube unable to advance into the stomach, with a characteristic coiled appearance proximal to the OA [24]. In cases of TEF, the catheter tip terminates in the upper oesophageal pouch, and gas is present in the intestines. In cases of isolated OA, the intestines are devoid of gas [25]. Additional diagnostic information can be obtained through oesophagoscopy or bronchoscopy [24]. It is essential to assess neonates with OA and TEF for the presence of concomitant defects. Cardiac defects occur in 53% of patients, musculoskeletal defects in 33%, renal anomalies in 30%, and craniofacial lesions in 16% [26]. A comprehensive evaluation includes a cardiac echocardiogram, radiographs of the limbs and spine, renal ultrasound, and a detailed physical examination of the anal and genital regions to identify any abnormalities [24, 26] (Figures 2, 3).

PREOPERATIVE MANAGEMENT

As part of preoperative care, the newborn's condition stabilisation, drug treatment, and interdisciplinary surgical planning are crucial.

Stabilisation of the newborn's condition is a key component of preoperative care. Decompression of the upper oesophagus with a Replogle probe enables effective secretion suctioning and prevents aspiration [25, 27]. This is the standard approach in most newborns [28, 29]. Proper positioning is also crucial; the baby should be placed with the head elevated at a 30–40° angle to reduce the risk of reflux and aspiration [7, 25, 27]. Respiratory support should be tailored to the patient's condition – non-

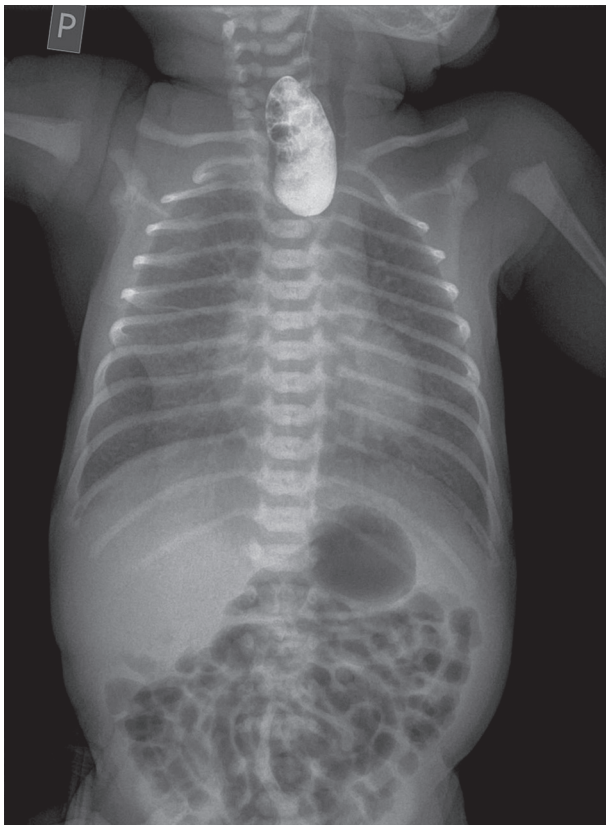

FIGURE 2. The contrast agent used in the X-ray examination reveals a blind-ended upper oesophagus

Source: Archives of the Department and Clinic of Neonatology, Wrocław Medical University

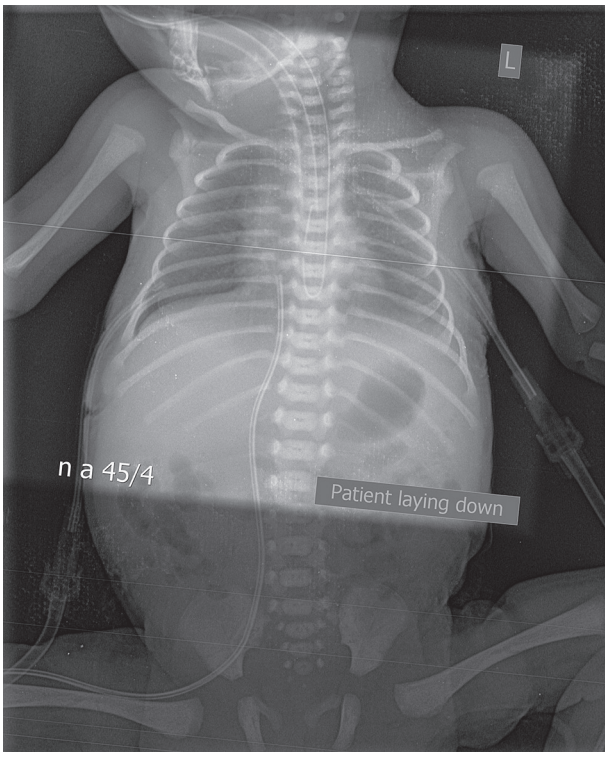

FIGURE 3. The orogastric tube is coiled in the upper oesophagus, indicating a blind-ended proximal oesophagus

Source: Archives of the Department and Clinic of Neonatology, Wrocław Medical University

invasive ventilation is avoided in favour of intubation if necessary [30]. If gastric distension is a concern, a Fogarty catheter can be used to temporarily close the TEF [31]. During transport to a specialised centre, the neonate should be cardiopulmonary stable and continuously monitored for vital signs [7, 32].

Pharmacological treatment plays an important role in preparing the newborn for surgery. Intravenous fluid therapy is used to maintain proper haemodynamic balance [25, 29, 28]. Opioids should be avoided because they may prolong extubation time and impair the swallowing reflex [31]. The use of antibiotic prophylaxis remains controversial; while it is routinely administered in some cases, ERNICA research recommends an individualised approach for each patient [30].

Moreover, surgical preparation includes assessing the length of the oesophageal hiatus, which can be done *via* fluoroscopic examination or with a Hegar dilator in cases of long-standing atresia [30, 31]. The choice of surgical approach depends on the hiatus length and the patient's overall condition. In some cases, primary anastomosis is feasible, whereas in others, staged treatment is required [30, 29]. A multidisciplinary medical team meeting, including a surgeon, anaesthesiologist, neonatologist, cardiologist, and otolaryngologist, is also crucial. Joint management planning enhances the likelihood of a successful surgery and reduces the risk of complications [26, 33].

METHODS OF TREATMENT

The diagnosis of EA in a newborn necessitates surgical intervention. Surgical treatment aims to create a primary anastomosis and is performed using two approaches – classical thoracotomy or thoracoscopy [31]. According to the largest published meta-analysis from 2021, the thoracoscopic method has been recognised as a routine procedure for the treatment of OA in newborns [34]. The ERNICA guidelines also present the thoracoscopic approach as a viable option for surgical treatment, highlighting its many advantages, while at the same time emphasising the necessity of performing it exclusively by specialised teams with appropriate expertise [33].

THORACOTOMY

Classical thoracotomy is performed by making a vertical, horizontal, or U-shaped skin incision, with the most common approach being a right-sided posterolateral thoracotomy. Muscle incision should be avoided whenever possible. If the patient presents with a right-sided aortic arch, a left-sided thoracotomy is recommended. Entry into the thoracic cavity is typically achieved through the fourth intercostal space *via* an extrapleural approach to the posterior mediastinum. Whenever feasible, the azygos vein is preserved. At this stage, any identified TEF is dissected and ligated with the use of a transfixing suture, single suture, or a clip. Subsequently, the upper oesopha-

geal pouch is mobilised and anastomosed with the lower pouch using a single layer of interrupted absorbable sutures to establish oesophageal continuity. A transanastomotic tube is inserted into the oesophageal lumen to prevent secondary stricture formation [31, 33, 35, 36]. According to the ERNICA guidelines, routine thoracic drainage should be avoided [30].

THORACOSCOPY

Thoracoscopy is considered an effective and safe approach, providing similar or better outcomes than classical thoracotomy. The only significant drawback of this method is the higher rate of anti-reflux surgeries performed within the first years of the patient's life due to gastrointestinal reflux (GER) [34].

Thoracoscopy has been associated with many advantages such as shorter hospital stay, earlier postoperative extubation, faster initiation of oral feeding, and minimal blood loss, eliminating the need for transfusion [34, 37]. Moreover, it is a less traumatic technique, requiring the use of fewer analgesics. At the same time, it avoids posterolateral thoracotomy, which increases the risk of severe pain, thoracic deformities, tracheomalacia, and surgical site infection [34].

The risk of complications such as secondary oesophageal stricture, the necessity for stricture dilation, and anastomotic leakage is comparable to that of the classical approach [34]. Similarly, mortality rates remain low, with thoracoscopic surgery showing a mortality rate of 3.2%, primarily attributed to the presence of VACTERL association in affected neonates [38].

The thoracoscopic method can be performed *via* a transpleural or extrapleural approach. The transpleural approach leads to right lung collapse due to pleural insufflation but allows improved visualisation of critical structures, including the superior vena cava, azygos vein, phrenic nerve, and vagus nerve, reducing the risk of iatrogenic injury. In the extrapleural approach, carbon dioxide insufflation expands the operative field. This technique aims to prevent pleural empyema and the leakage of gastrointestinal content into the pleural cavity. It is achieved by dissecting the intercostal muscles and introducing instruments into the extrapleural space [36, 39, 40]. Technical guidelines recommend that intraoperative carbon dioxide insufflation pressure should not exceed 5 mm Hg [33].

OESOPHAGEAL POUCH ELONGATION AND DELAYED PRIMARY ANASTOMOSIS IN LONG-GAP OESOPHAGEAL ATRESIA

Long-gap oesophageal atresia (LGOA) is defined as any case in which the gap between the oesophageal pouches is equal to or greater than three vertebral bodies, or when no air is present in the stomach [30].

The restoration of oesophageal continuity in LGOA differs from standard cases and is typically achieved through oesophageal pouch elongation followed by delayed primary anastomosis. Elongation of the oesophageal segments can occur spontaneously over time or be actively stimulated using traction techniques. According to recent global studies, the most commonly employed approach is spontaneous elongation, in which the proximal oesophagus is stimulated by the swallowing reflex, while the distal segment undergoes lengthening due to increased intraluminal pressure caused by reflux [41–43].

One of the earliest traction-based oesophageal lengthening techniques was described by Foker *et al.* [44]. This approach involved placing multiple small sutures around the oesophageal pouches and securing them to the prevertebral fascia *via* a transpleural approach. After three to four days, a second thoracotomy was performed to assess the effectiveness of the traction. Depending on the results, either additional traction sutures were applied dorsally or a primary anastomosis was attempted. A maximum gap of approximately 1 cm between the oesophageal ends was required for anastomosis [36]. In 2015, van der Zee *et al.* [45] adapted the Foker technique for thoracoscopic application, improving its feasibility and reducing surgical invasiveness [36].

An alternative traction method, based on the Foker technique, was proposed by Patkowski [37]. His thoracoscopic approach involves the use of an internal static traction suture placed between the oesophageal segments. To enhance traction force and prevent tissue tearing or leakage, two clips are applied at the ends of both oesophageal pouches. Typically, primary anastomosis is performed within one to five days following the placement of the traction suture.

According to the ERNICA guidelines, procedures using the Kimura method, which involves extrathoracic elongation of the oesophagus, should no longer be performed [30, 36]. Similarly, gastric division, circular myotomy (Livaditis procedure), and flap techniques are also considered inadvisable [30].

OESOPHAGEAL REPLACEMENT WITH THE USE OF GASTROINTESTINAL SEGMENTS IN LONG-GAP OESOPHAGEAL ATRESIA

The method of oesophageal replacement by using the stomach or jejunum is only performed when other reconstructive techniques fail to achieve the desired outcomes [7, 41, 46]. According to the ERNICA guidelines, the colon should no longer be used for oesophageal replacement [30]. Surgical practice prioritises the use of the native oesophagus for reconstruction whenever possible, to minimise the risk of long-term complications [30, 41, 46].

POSTOPERATIVE MANAGEMENT

When the intervention is complete, the patient is transferred directly from the operating room to the Neo-

natal Intensive Care Unit (NICU) and kept intubated, sedated, and paralysed for the first 24–48 hours. In the case of tension anastomosis, management must be individualised, because this may necessitate extended intubation with administration of muscle relaxants for 5–7 days post-op and a chin-to-chest position [33, 47, 48].

To maintain optimal thermoregulation, the newborn is placed in an incubator. An important principle of postoperative care for neonates is 'minimal handling', including avoidance of sudden head movements [47]. Maintaining effective analgesia is crucial during the postoperative period [47, 48]. The issue of administering antibiotics for longer than 24 hours after surgery is not standardised; each case should be considered individually [31, 33].

According to ERNICA, a routine contrast study is not required before initiating oral feeding [33]. However, close monitoring of the patient is essential to promptly detect any anastomotic leaks, as these can occasionally occur and may lead to mediastinitis [49]. Enteral nutrition through the nasogastric tube may be initiated at 24 hours postoperatively and is gradually transitioned to oral feeding. The nasogastric tube can be removed after 8–9 postoperative days, when there is no evidence of stenosis or of leakage at the site of the anastomosis [33, 48]. After surgery, the newborn requires monitoring of basic vital and biochemical parameters for early detection of potential complications and associated risks [47]. A study shows that the overall postoperative morbidity rate is 62%. The most common complications following primary surgery include oesophageal anastomotic stenosis (42.5%), anastomotic leak (22.9%), vocal cord dysfunction (6.7%), recurrent fistula (4.9%), surgical site infection (2.3%), and chylothorax (2.6%). Careful monitoring of biophysical parameters is crucial due to the high mortality rate along with adjusting treatment to control any associated anomalies, including cardiac and circulatory system defects [26].

Oesophageal atresia/tracheoesophageal fistula is closely associated with the development of GER, which has been reported to occur in approximately 43% of patients. Gastrointestinal reflux can manifest as regurgitation or vomiting, growth failure, apnoeic attacks, barking cough, recurrent pneumonia, chronic respiratory tract disease, and recurrent anastomotic stenosis [50]. Acid suppression is an important element in the prevention of GER-related complications, such as Barrett's oesophagus. Further research is needed to determine the optimal duration of antacid therapy [51]. The most commonly used medications are proton pump inhibitors (40%), H2 blockers (37%), or a combination of both (22%) [26]. However, anti-reflux surgery is considered when symptoms persist despite optimised medical management, including postural measures, dietary modification, and appropriate antacids and prokinetic agents [50].

Another very important aspect of managing OA is active cooperation with parents, which encourages them to learn about the illness and prepares them to participate in their child's care at home. Additionally, education should include resuscitation training [31, 33, 47].

CONCLUSIONS

This review aims to familiarise the medical community with the topic of OA and its implications for the patient during the first days and months of life, which can be severe due to the nature of the malformation and frequent associated anomalies. Particular attention is given to the perioperative period and to the comprehensive care provided by neonatologists and paediatric surgeons, which enables newborns to survive and develop properly.

Oesophageal atresia is among the most common congenital oesophageal disorders. In Europe, the prevalence is 2.43 cases per 10,000 births, underscoring the importance of understanding its aetiology, risk factors, diagnostic process, treatment strategies, and perioperative care of affected newborns, who may be encountered by physicians across multiple specialties.

Diagnosis of OA should be initiated as early as possible in order to detect abnormalities or structural defects and to adequately prepare both the parents and the medical team for the specialised care required from the very first hours of the patient's life. Prenatally, OA can be suspected with ultrasonography, MRI, and AF analysis, although detection remains challenging. In Europe, a large proportion of OA cases are detected postnatally, owing to the rapid onset of the newborn's symptoms.

Perioperative care of an infant with OA involves a range of specialised procedures carried out in the NICU, including stabilisation of the general condition, monitoring of vital functions and biochemical parameters, respiratory support, appropriate feeding, and the administration of medications.

Surgical intervention is always required, increasingly performed *via* a thoracoscopic approach. Recent guidelines consider thoracoscopy a safe and effective option, with outcomes comparable to or better than thoracotomy, while not exposing the infant to higher rates of postoperative complications. Experts emphasise its many advantages but note that further specialisation and training are needed to optimise results, particularly in reducing operative time. Comparative randomised long-term studies are needed to determine the true value of this approach.

DISCLOSURES

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