Congenital Esophageal Stenosis

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Abstract
Congenital esophageal stenosis (CES) is a rare condition. There are three pathological types of CES: with tracheobronchial remnants in the esophageal wall, fibromuscular thickening of the esophageal wall, and membranous mucosal diaphragm or web. Congenital esophageal atresia is the most common associated anomaly. Symptoms of CES include vomiting or regurgitation, dysphagia, recurrent respiratory tract infections, and growth retardation. Esophagograms of stenosis exhibit tapered or abrupt narrowing of the esophagus with various degrees of dilatation of its suprastenotic portion. Esophagoscopy, manometric study, and pH monitoring are helpful tools for differential diagnoses in distinguishing CES from achalasia and secondary esophageal stricture due to gastroesophageal reflux (GER). Balloon dilatation is the first choice of treatment in CES. When patients fail to respond to repeated attempts of bougienage, surgical intervention should be considered. Resection of the stenosis followed by end-to-end esophageal anastomosis is a general surgical treatment. Good outcome of circular myectomy has been also reported. Complications, including an iatrogenic esophageal perforation following bougienage and leakage after segmental resection and reconstruction of the esophagus, have been reported. While good prognosis has been reported following dilatation and/or surgery when needed, it was also reported that dysphagia occurred frequently regardless of the therapeutic option at follow-up. Close, long-term follow-up is highly recommended.

Keywords
Congenital esophageal stenosis • Esophageal atresia • Tracheobronchial remnant • Fibromuscular thickening • Dilatation • Endo-end esophageal anastomosis • Circular myectomy • Esophageal perforation

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Congenital esophageal stenosis (CES) is a rare condition. Gross (1953) reviewed 38 cases of CES from the records of the Boston Children’s Hospital in 1953 and reported that repeated dilation provided complete relief in most cases; however, he later stated that CES cases that did not respond after six attempts at dilation should be strongly considered for surgical resection or revision of the strictured area. Since then, a number of treatment modalities have been devised, but there is no consensus regarding which treatment modality could be the best. Although the efficacy of the conservative treatment varies on the case-by-case basis, the surgical treatment, which is expected to provide prompt relief from symptoms, carries certain risks of postoperative complications including leaks, stricture, and gastroesophageal reflux (GER). The surgeons should consider the differential diagnosis as well as understand the pathological basis of CES to employ the most appropriate treatment strategy.

Pathology

CES is defined as an intrinsic stenosis of the esophagus caused by congenital malformation of the esophageal wall. There are three pathological types of CES: with tracheobronchial remnants in the esophageal wall (Frey and Duschl 1936; Bergmann and Charnas 1958; Kumar 1962; Paulino et al. 1963; Ishida et al. 1969; Rose et al. 1975; Ohkawa et al. 1975; Ibrahim and Sandry 1981; Deiraniya 1974; Fekete et al. 1987; Sneed et al. 1979; Spitz 1973; Briceno et al. 1981; Tubino et al. 1982; Shoshany and Bar-Maor 1986), fibromuscular thickening of the esophageal wall (Fekete et al. 1987; Bonilla and Bower 1959; Mahour et al. 1971; Tuqan 1962; Vidne and Levy 1970; Todani et al. 1984), and membranous mucosal diaphragm or web (Ohkawa et al. 1975; Fekete et al. 1987; Beatty 1928; Huchzermeier et al. 1979; Overton and Creech 1953; Schwaetz 1962; Grabowski and Andrews 1996; Takayanagi et al. 1975; Sarihan and Abes 1997; Komuro et al. 1999; Roy et al. 1996).

Stenosis due to tracheobronchial remnants is the most common type of this anomaly and is localized at the distal esophagus (Sneed et al. 1979). Fibromuscular thickening is observed in the middle or lower portions of the esophagus (Fekete et al. 1987; Todani et al. 1984), and membranous webs are observed in the upper or middle levels of the esophagus (Fekete et al. 1987; Grabowski and Andrews 1996; Takayanagi et al. 1975; Sarihan and Abes 1997; Komuro et al. 1999; Roy et al. 1996). The stenotic area in cases with tracheobronchial remnants is usually localized, and the area of fibromuscular stenosis varies from one to several centimeters in length with circular thickening of the esophageal wall. In cases of membranous web, single webs were observed in children (Grabowski and Andrews 1996; Sarihan and Abes 1997; Roy et al. 1996), whereas plural webs, termed as multiple tracheal-like rings, were observed in young adults (Younes and Johnson 1999; Katzka et al. 2000; Pokieser et al. 1998).

In CES with tracheobronchial remnants (mature or immature cartilages), the seromucous tracheobronchial glands and ciliated epithelium were usually reported during microscopic examination of the stenotic esophageal wall (Fig. 1) (Paulino et al. 1963; Fekete et al. 1987; Murphy et al. 1995). On the other hand, in fibromuscular stenosis, circumferential proliferation of smooth muscle fibers with moderate fibrosis has been observed (Fig. 2) (Fekete et al. 1987; Todani et al. 1984; Murphy et al. 1995). Singaram et al. (1995) reported a significant reduction of myenteric nitrinergic neurons and fibers in the muscle layer of two young adults diagnosed with fibromuscular variant of CES. Lack of submucosa (Takayanagi et al. 1975), loose vascular
connective tissue, and diffuse lymphocytes (Grabowski and Andrews 1996) have been observed microscopically in specimens of mem-branous web.

**Epidemiology/Etiology**

CES occurs in 1 in 25,000–50,000 live births (Fekete et al. 1987; Bluestone et al. 1969). Although the reason is unknown, the incidence of CES is higher in Japan than elsewhere (Ohkawa et al. 1975; Nishina et al. 1981). Nevertheless, there is no sex-associated predisposition.

Regarding the etiology of CES, stenosis caused by tracheobronchial remnants and fibromuscular thickening is believed to be a developmental disorder during the formation and separation of the primitive foregut into the trachea and esophagus by the end of the first fetal month. The membranous mucosal diaphragm or web is believed to be a result of incomplete reformation of the esophageal lumen upon recanalization of the esophagus between the sixth and eighth week of gestation.

The incidence of anomalies associated with CES has been reported as 17–33% (Fekete et al. 1987; Nishina et al. 1981); among these, congenital esophageal atresia is the most common (Ibrahim and Sandry 1981; Deiraniya 1974; Fekete et al. 1987; Mahour et al. 1971; Tuqan 1962; Overton and Creech 1953; Nishina et al. 1981; Sheridan and Hyde 1990; Yeung et al. 1992; Neilson et al. 1991; Kawahara et al. 2001; Takamizawa et al. 2002; Amae et al. 2003), and 4.9–14% tracheoesophageal fistula (TEF) cases are associated with CES (Kawahara et al. 2001; Newman and Bender 1997; Vasudevan et al. 2002). Surgeons should be aware of the high incidence of association of congenital esophageal atresia with distal CES with tracheobronchial remnants. Moreover, cardiac anomalies (Fekete et al. 1987; Overton and Creech 1953), intestinal atresia (Fekete et al. 1987; Huchzermeyer et al. 1979), anorectal malformation (Deiraniya 1974; Nishina et al. 1981), and chromosomal anomalies (Rose et al. 1975; Fekete et al. 1987; Todani et al. 1984; Huchzermeyer et al. 1979) can be also associated with CES.

**Symptoms**

Symptoms of CES include vomiting or regurgitation, dysphagia, recurrent respiratory tract infections, and growth retardation.

Although the etiology of CES usually has congenital origins, the symptoms rarely develop in newborns (Schwaetz 1962). Characteristically, the onset of regurgitation coincides with the introduction of semisolid and solid foods around the
age of 6 months in patients with tracheobronchial remnants (Murphy et al. 1995). In some patients, a foreign body in the esophagus might be the first symptom observed (Bluestone et al. 1969). When esophageal peristalsis is preserved, the presentation of CES might be delayed until adulthood (McNally et al. 1990).

Patients with CES associated with esophageal atresia are sometimes diagnosed at the time of surgical repair or during the postoperative course of esophageal atresia before presenting any symptoms of CES.

**Diagnosis**

Difficulties in determining the differential diagnoses of CES to distinguish it from achalasia and secondary esophageal stenosis and particularly from a stricture due to reflux esophagitis (Bluestone et al. 1969) have resulted in various clinical problems during its treatment.

First, achalasia, inflammatory esophagitis, and stenosis caused by tumor or extrinsic compression of the esophagus should be excluded. The localization of the stenosis varies with the type of pathology, as we mentioned in the earlier section.

Patients who develop symptoms such as vomiting and dysphagia should be administered a barium swallow test to obtain esophagograms. Esophagograms of stenosis exhibit tapered or abrupt narrowing of the esophagus with various degrees of dilatation of its suprastenotic portion (Fig. 3a, b). Most of the stenoses due to tracheobronchial remnants are visible on esophagograms as abrupt esophageal narrowing, whereas fibromuscular stenosis usually exhibits as tapered esophageal narrowing (Kawahara et al. 2001; Amae et al. 2003). Following surgery of esophageal atresia with or without TEF, an esophagogram should be evaluated with great care as it is easy to overlook a narrowing at the mid-distal esophagus.

To evaluate the exact site and extent of the stenosis, fluorography using a balloon catheter can be employed. The balloon catheter is inserted through the esophagus. The location and shape of the inflated balloon provides a clear image of the esophageal stenosis (Figs. 4 and 5a, b).

Esophagoscopy, manometric study, and pH monitoring are helpful tools for differential diagnoses in distinguishing CES from achalasia and secondary esophageal stricture due to GER. Esophageal endoscopy can directly evaluate not
only the stenotic area and the site of the gastro-esophageal junction but also the presence and the severity of esophagitis. The mucosa distal to the stenosis is normal in CES. In addition to esophagoscopy, endoscopic ultrasonography has been recently employed to evaluate the fine structure of CES and is useful in choosing the treatment strategy: balloon dilatation or surgical treatment (Takamizawa et al. 2002; Kouchi et al. 2002; Usui et al. 2002).

In cases of CES, preoperative esophageal manometry demonstrates a normal pattern of the lower esophageal sphincter and a small high-pressure zone in the resting pressure profile, which corresponds to the stenotic area of the esophagus. This small high-pressure zone disappears after the corrective surgery. Moreover, an esophageal motility study would reveal a peristalsis corresponding to the stenosis. Further, pH monitoring can reveal a significant positive reflux, which is not observed in patients with CES, unlike in patients with GER.

CES associated with esophageal atresia is often overlooked at the time of the initial esophageal

Fig. 4 Fluorography using a balloon catheter. A low-compliance balloon catheter (Rigiﬂex™ II, Boston Scientiﬁc LTD, UK) is placed at the stenosis and gradually inﬂated, and then the location and the length of the stenosis are clearly visible

Fig. 5 (a) is an esophagogram of a patient with abrupt narrowing. A Foley catheter inserted through the esophagus is inﬂated and pulled up to locate the stenosis (b)
surgical repair and diagnosed sometime later. Ibrahim et al. (2007) recommended taking a surgical specimen routinely for histopathological studies from the tip of the lower esophageal pouch during primary repair of esophageal atresia.

**Treatment**

The principal aims of CES treatment are the alleviation of symptoms and maintenance of antireflux mechanism of the gastroesophageal junction. This condition can be treated either conservatively or by surgical intervention. Nonsurgical methods include balloon dilatation and endoscopic treatment (Dall’Oglio et al. 2016; Terui et al. 2015).

**Nonsurgical Treatment**

Balloon dilatation is the first choice of treatment in CES. This method should be employed not only for patients with tapered esophageal narrowing but also with abrupt esophageal narrowing when a balloon catheter can be passed safely through the stenotic area. Low-compliance balloon catheters are commonly used (Amae et al. 2003). It has been reported that the effect of balloon dilatation varies with the type of pathology and that cases with membranous web of the esophagus and some cases of fibromuscular stenosis can be treated by suitable dilatation (Fekete et al. 1987; Grabowski and Andrews 1996; Sarihan and Abes 1997; Komuro et al. 1999). It has been also believed that the efficacy of balloon dilatation in CES with tracheobronchial remnants is limited. However, it is difficult to precisely assess the efficacy of balloon dilatation in CES of each type of pathology, because histological findings of the stenotic lesions of the patients who respond to dilatation are not confirmed. Currently, the responsiveness of CES with tracheobronchial remnants to dilatation is uncertain.

Recently, successful treatment of membranous web with the use of endoscopic instruments (electrocauterization, high-frequency-wave snare/cutter) has been reported (Nose et al. 2005; Chao et al. 2008).

**Surgery**

When patients fail to respond to repeated (four to six times) attempts of bougienage, surgical intervention should be considered. Surgical resection of the stenosis followed by end-to-end esophageal anastomosis is a general surgical treatment for many cases with tracheobronchial remnants (Ishida et al. 1969; Ohkawa et al. 1975; Deiraniya 1974; Fekete et al. 1987; Sneed et al. 1979; Spitz 1973; Briceno et al. 1981; Tubino et al. 1982; Shoshany and Bar-Maor 1986; Nishina et al. 1981; Neilson et al. 1991; Kawahara et al. 2001; Takamizawa et al. 2002; Amae et al. 2003) and some cases with fibromuscular thickening (Todani et al. 1984; Murphy et al. 1995).

Most cases of CES can be operated upon using the thoracic approach. However, when the stenosis is localized in the abdominal esophagus, the abdominal approach might be utilized. In a few cases of CES, a complete resection of the stenotic segment using the thoracoscopic (Martinez-Ferro et al. 2006) or laparoscopic approach (Deshpande and Shun 2009) has been reported.

In the thoracic approach, right thoracotomy is employed for stenosis in the middle esophagus, and left thoracotomy is employed for stenosis in the lower part of the esophagus. After exposing the esophagus, a balloon catheter is inserted from the mouth to locate the stenotic segment. The catheter is then inflated and pulled up to confirm the lower margin of the stenosis. Following the cut in the distal end of the stenosis, a sterile balloon catheter is passed through the stenotic area from the oral stump of the esophagus. The upper margin of the stenosis is determined by pulling down the catheter (Fig. 6a, b) (Amae et al. 2003).

The stenotic area is completely removed, and end-to-end anastomosis is achieved using the single- or double-layer interrupted sutures of absorbable materials. The surgeons should ensure that the phrenic and vagal nerves are not damaged during the surgery.

Other options for surgical treatment are simple excision of cartilaginous remnants, longitudinal myotomy, circular myectomy, and esophageal replacement.
Among these procedures, simple excision of the cartilaginous remnants and subsequent repair of the esophageal wall have been reported (Paulino et al. 1963; Fekete et al. 1987; Amae et al. 2003). However, these methods are rarely employed because they failed to provide sufficient relief from symptoms in most cases of CES with tracheobronchial remnants.

The longitudinal myotomy, which is a standard procedure for esophageal achalasia than for CES, has been employed for fibromuscular stenosis. However, postoperative dilatation is frequently required, and its efficacy is still unconfirmed. Moreover, development of esophageal perforation after myotomy has been reported as well (Takamizawa et al. 2002).

Circular myectomy for CES with tracheobronchial remnants has been described (Maeda et al. 2004; Saito et al. 2008), and it might be employed for fibromuscular stenosis as well. During this surgical procedure, the lower margin of the stenotic segment is determined by pulling up a balloon catheter inserted through the mouth, which is similar to the technique followed during the resection of the stenotic portion. The upper margin is determined by pushing down the balloon catheter inserted through the mouth or by pulling down the balloon catheter inserted through a small incision at the esophageal mucosa into the esophageal lumen. The muscular layer is incised up to the submucosal layer at the lower and upper margins. Subsequently, the abnormal muscular layer is dissected circumferentially using sharp scissors or cautery (Fig. 7). If the incision at the esophageal mucosa is made to evaluate the upper margin of the stenosis, it is closed horizontally with absorbable monofilament sutures, and the muscular layer is closed horizontally with interrupted absorbable sutures. Circular myectomy is expected to prevent postoperative leakage and restenosis of the esophageal anastomosis (Maeda et al. 2004; Saito et al. 2008).

While thoracotomy has been the standard approach for surgical correction, the less invasive approach by thoracoscopy was recently reported (van Poll and van der Zee 2012).

Among various methods of surgical treatment for CES, segmental resection with end-to-end

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**Fig. 6** Resection of the stenotic esophagus using a balloon catheter. A Foley catheter inserted from the mouth is used to determine the lower margin of the stenosis (**a**). After cutting the esophagus at the lower margin, another sterile Foley catheter is inserted from the lower opening of the esophagus and used to determine the upper margin of the stenosis (**b**).

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**Fig. 7** Circular myectomy. The muscular layer is circumferentially removed, keeping the mucosal layer intact.
anastomosis of the esophagus is currently a standard procedure, and circular myectomy is a promising alternative.

Both procedures pose substantial risk of developing GER when anastomosis is performed under tension. When the stenosis is situated close to the gastroesophageal junction, and the surgery is performed through laparotomy, an antireflux procedure (e.g., a Nissen fundoplication) might be employed as well to prevent postoperative reflux (Amae et al. 2003).

If the vagal nerve is accidently severed, a pyloroplasty is performed.

Patients with extensive CES, necessitating the resection of more than 3 cm of the esophagus, might require esophageal replacement using the colon, jejunum, or a gastric tube.

Management of Complications Following Treatment

Complications, including an iatrogenic esophageal perforation following bougienage (Deiraniya 1974; Takamizawa et al. 2002; Romeo et al. 2011) and leakage after segmental resection and reconstruction of the esophagus (Fekete et al. 1987), have been reported. The rate of esophageal perforation after dilatation has been reported as 10.6–44% (Kawahara et al. 2001; McNally et al. 1990; Romeo et al. 2011) (Dall’Oglio et al. 2016). Perforations and large leakages might require surgical drainage and/or repair, but minor leakages can be successfully treated by maintaining the patient on total parenteral nutrition.

When GER develops following a simple resection or circular myectomy, an antireflux procedure is required.

Prognosis

Favorable results have been reported following appropriate treatment. Amae et al. (2003) reported the treatment outcome of 14 patients with CES, in which 11 and 3 patients became asymptomatic following surgery and dilatation, respectively. On the other hand, according to the recent report of 61 cases of CSE by Michaud et al. (2013), dysphagia occurred frequently regardless of the therapeutic option, and it remained in 36% of patients at follow-up. Close, long-term follow-up is highly recommended.

Conclusion and Future Directions

CES is very rare condition. It is frequently associated with esophageal atresia but often overlooked at the time of the initial surgery. When dysphagia persists after the surgery for esophageal atresia, association of CES should be suspected and assessed in order to avoid delayed diagnosis. As CES is revealed, dilatation is the first-line therapeutic modality, which is likely to be effective for fibromuscular type of CES. In case repeated dilatation cannot improve the symptoms, employment of surgical treatment should be considered. While surgical resection of the stenotic esophagus and end-to-end esophageal anastomosis is the most common procedure, favorable results of circular myectomy has been reported. Endoscopic surgery for CES was also recently employed.

Good prognosis has been reported following dilatation and/or surgery when needed. However, it was also reported that dysphagia occurred frequently regardless of the therapeutic option at follow-up. Thus, close long-term follow-up is essential.

CES with extensive stenosis or severe esophageal dysmotility may require esophageal replacement, which is highly invasive and associated with considerable risk of morbidity. Due to recent advancement of regenerative medicine (Sommer et al. 2013), tissue-engineered esophagus is expected to be utilized for the most severe type of CES in the near future.

Cross-References

- Caustic Injuries of Esophagus
- Embryology of Congenital Malformation
- Esophageal Atresia
- Esophageal Perforation in Newborn
Foreign Bodies
Pediatric Surgical Endoscopy

References

Ishida M, Tsuchida Y, Saito S, et al. Congenital esophageal stenosis. J Pediatr Surg. 2003;38:565–70.

Beatty CC. Congenital stenosis of the esophagus. Br J Child Dis. 1928;25:237–70.

Bergmann M, Charnas RM. Tracheobronchial rests in the esophagus. J Thorac Surg. 1958;35:97–104.

Bluestone CD, Perry R, Sieber WK. Congenital esophageal stenosis. Laryngoscope. 1969;79:1095–103.

Bonilla KB, Bower WF. Congenital esophageal stenosis; pathologic studies following resection. Am J Surg. 1959;97:772–6.

Briceno LI, Grases PJ, Gallego S. Tracheobronchial and pancreatic remnants causing esophageal stenosis. J Pediatr Surg. 1980;15:637–41.

Chao HC, Chen SY, Kong MS. Successful treatment of congenital esophageal web by endoscopic electrocauterization and balloon dilatation. J Pediatr Surg. 2008;43:e13–5.

Dall’Oglio L, Caldaro T, Foschia F, et al. Endoscopic management of esophageal stenosis in children: new and traditional treatments. World J Gastrointest Endosc. 2016;8(4):212–9.

Deiraniya AK. Congenital oesophageal stenosis due to tracheobronchial remnants. J Pediatr Surg. 1974;9:720–5.

Deshpande AV, Shun A. Laparoscopic treatment of esophageal stenosis due to tracheobronchial remnant in a child. J Laparoendosc Adv Surg Tech A. 2009;19:107–9.

Fekete CN, Backer AD, Jacob SL. Congenital esophageal stenosis. A review of 20 cases. Pediatr Surg Int. 1987;2:86–92.

Frey EK, Duschi L. Der Kardiopasmus. Erge Chirurg Orthopaed. 1936;29:637–52.

Grabowski ST, Andrews DA. Upper esophageal stenosis: two case reports. J Pediatr Surg. 1996;31:1438–9.

Gross RE. The surgery of infancy and childhood. Philadelphia: WB Saunders & Co.; 1953.

Huchzeremeyr H, Burdeiski M, Hruby M. Endoscopic therapy of a congenital oesophageal stricture. Endoscopy. 1979;11:259–62.

Ibrahim NBN, Sandry RJ. Congenital esophageal stenosis caused by tracheobronchial structures in the esophageal wall. Thorax. 1981;36:465–8.

Ibrahim AH, Al Malki TA, Hamza AF. Congenital esophageal stenosis associated with esophageal atresia: new concepts. Pediatr Surg Int. 2007;23:533–7.

Ishida M, Tsucida Y, Saito S, et al. Congenital esophageal stenosis due to tracheobronchial remnants. J Pediatr Surg. 1969;4(3):339–45.

Katzka DA, Levine MS, Ginsberg GG, et al. Congenital esophageal stenosis in adults. Am J Gastroenterol. 2000;95:32–6.

Kawahara H, Imura K, Yagi M, et al. Clinical characteristics of congenital esophageal stenosis distal to associated esophageal atresia. Surgery. 2001;129:29–38.

Komuro H, Makino S, Tsuchiya I, et al. Cervical esophageal web in a 13-year-old with growth failure. Pediatr Int. 1999;41:568–70.

Kouchi K, Yoshida H, Matsunaga T, et al. Endosonographic evaluation in two children with esophageal stenosis. J Pediatr Surg. 2002;37:934–6.

Kumar R. A case of congenital oesophageal stricture due to a cartilaginous ring. Br J Surg. 1962;49:533–4.

Maeda K, Hisamatsu C, Hasegawa T, et al. circular myectomy for the treatment of congenital esophageal stenosis owing to tracheobronchial remnant. J Pediatr Surg. 2004;39:1765–8.

Mahour GH, Jouonston PW, Gwinn JL, et al. Congenital esophageal stenosis distal to esophageal atresia. Surgery. 1971;69:936–9.

Martinez-Ferro M, Rubio M, Piaggio L, et al. Thoracoscopic approach for congenital esophageal stenosis. J Pediatr Surg. 2006;41:E5–7.

McNally PR, Collier 3rd EH, Lopiano MC, et al. Congenital esophageal stenosis. A rare cause of food impaction in the adult. Dig Dis Sci. 1990;35:263–6.

Michaud L, Coutener F, Podevin G, et al. Characteristics and management of congenital esophageal stenosis: findings from a multicenter study. Orphanet J Rare Dis. 2013;8:186.

Murphy SG, Yazbeck S, Russo P. Isolated congenital esophageal stenosis. J Pediatr Surg. 1995;30:1238–41.

Neilson IR, Croitoru DP, Gutman FK, et al. Distal congenital esophageal stenosis associated with esophageal atresia. J Pediatr Surg. 1991;26:478–81.

Newman B, Bender TM. Esophageal atresia/tracheoesophageal fistula and associated congenital esophageal stenosis. Pediatr Radiol. 1997;27:530–4.

Nishina T, Tsucida Y, Saito S. Congenital esophageal stenosis due to tracheobronchial remnants and its associated anomalies. J Pediatr Surg. 1981;16:190–3.

Nose S, Kubota A, Kawahara H, et al. Endoscopic membranectomy with a high-frequency-wave snare/cutter for membranous stenosis in the upper gastrointestinal tract. J Pediatr Surg. 2005;40:1486–8.

Ohkawa H, Takahashi H, Hoshino Y, et al. Lower esophageal stenosis in association with tracheobronchial remnants. J Pediatr Surg. 1975;10:453–7.

Overton RC, Creech O. Unusual esophageal atresia with distant membranous obstruction of the esophagus. J Thorac Surg. 1953;35:674–7.

Paulino E, Roselli A, Aprigliano F. Congenital esophageal stricture due to tracheobronchial remnants. Surgery. 1963;53:547–50.

Pokieser P, Schima W, Schober E, et al. Congenital esophageal stenosis in a 21-year-old man: clinical and radiographic findings. AJR Am J Roentgenol. 1998;170:147–8.

van Poll D, van der Zee DC. Thoracoscopic treatment of congenital esophageal stenosis in combination with...
H-type tracheoesophageal fistula. J Pediatr Surg. 2012;47:1611–3.

Romeo E, Foschia F, de Angelis P, et al. Endoscopic management of congenital esophageal stenosis. J Pediatr Surg. 2011;46:838–41.

Rose JS, Kassner EG, Jurgens KH, et al. Congenital esophageal strictures due to cartilaginous rings. Br J Radiol. 1975;48:16–8.

Roy GT, Cohen RC, Willeams SJ. Endoscopic laser division of an esophageal web in a child. J Pediatr Surg. 1996;31:439–40.

Saito T, Ise K, Kawahara Y, et al. Congenital esophageal stenosis because of tracheobronchial remnant and treated by circular myectomy: a case report. J Pediatr Surg. 2008;43:583–5.

Sarihan H, Abes M. Congenital esophageal stenosis. J Cardiovasc Surg. 1997;38:421–3.

Schwaetz SI. Congenital membranous obstruction of esophagus. Arch Surg. 1962;85:480–2.

Sheridan J, Hyde I. Oesophageal stenosis distal to oesophageal atresia. Clin Radiol. 1990;42:274–6.

Shoshany G, Bar-Maor JA. Congenital stenosis of the esophagus due to tracheobronchial remnants: a missed diagnosis. J Pediatr Gastroenterol Nutr. 1986;5:977–9.

Singaram C, Sweet MA, Gaumnitz EA. Peptidergic and nitrinergic denervation in congenital esophageal stenosis. Gastroenterology. 1995;109:275–81.

Sneed WF, LaGarde MS, Kogutt MS, et al. Esophageal stenosis due to cartilaginous tracheobronchial remnants. J Pediatr Surg. 1979;14:786–8.

Sommer G, Schrieﬂ A, Zeindlinger G, et al. Multiaxial mechanical response and constitutive modeling of esophageal tissues: impact on esophageal tissue engineering. Acta Biomater. 2013;9:9379–91.

Spitz L. Congenital esophageal stenosis distal to associated esophageal atresia. J Pediatr Surg. 1973;8:973–4.

Takamizawa S, Tsugawa C, Mouri N, et al. Congenital esophageal stenosis: therapeutic strategy based on etiology. J Pediatr Surg. 2002;37:197–201.

Takayanagi K, Ii K, Komi N. Congenital esophageal stenosis with lack of the submucosa. J Pediatr Surg. 1975;10:425–6.

Terui K, Saito T, Mitsunaga T, Nakata M, Yoshida H. Endoscopic management for congenital esophageal stenosis: a systematic review. World J Gastrointest Endosc. 2015;7(3):183–91.

Todani T, Watanabe Y, Mizuguchi T. Congenital oesophageal stenosis due to fibromuscular thickening. Z Kinderchir. 1984;39:11–4.

Tubino P, Marouelli LF, Alves E, et al. Choristoma: esophageal stenosis, due to tracheobronchial remnants. Z Kinderchir. 1982;35:14–7.

Tuqan NA. Annular stricture of the esophagus distal to congenital tracheoesophageal fistula. Surgery. 1962;52:394–5.

Usui N, Kamata S, Kawahara H, et al. Usefulness of endoscopic ultrasonography in the diagnosis of congenital esophageal stenosis. J Pediatr Surg. 2002;37:1744–6.

Vasudevan SA, Kerendi F, Lee H, et al. Management of congenital esophageal stenosis. J Pediatr Surg. 2002;37:1024–6.

Vidne B, Levy MJ. Use of pericardium for esophagoplasty in congenital stenosis. Surgery. 1970;68:389–92.

Yeung CK, Spitz L, Brereton RJ, et al. Congenital esophageal stenosis due to tracheobronchial remnants: a rare but important association with esophageal atresia. J Pediatr Surg. 1992;27:852–5.

Younes Z, Johnson DA. Congenital esophageal stenosis: clinical and endoscopic features in adults. Dig Dis. 1999;17:172–7.