Alimentary tract duplications in children: report of 26 years' experience

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SUMMARY: Karnak İ, Öcal T, Şenocak ME, Tanyel FC, Büyükpamukçu N. Alimentary tract duplications in children: report of 26 years' experience. Turk J Pediatr 2000; 42: 118-125.

Duplications of the alimentary tract are one of the rare anomalies of the gastrointestinal system. Because of the wide spectrum of the signs and symptoms, preoperative diagnosis frequently cannot be made. A close familiarity with clinical and surgical characteristics provides appropriate management and treatment of duplications. A retrospective clinical study was conducted to evaluate clinical and surgical characteristics and the treatment of duplications of the alimentary tract.

During a 26-year period between 1971 and 1997, 38 patients with duplications of alimentary tract underwent operation at the Hacettepe University Department of Pediatric Surgery. Forty-two duplications in 38 patients (20 male, 53%; 18 female, 47%) were encountered. Sixty-nine percent of the patients were symptomatic under the age of one year, with 24 percent presenting with symptoms in the neonatal period. There were one sublingual, nine intrathoracic (including 2 thoracoabdominal) and 32 intraabdominal duplications. Abdominal mass, abdominal distention, constipation, vomiting and respiratory distress were the most frequently encountered signs and symptoms. Plain thoracic and abdominal X-rays, ultrasonography, and computed tomography of the chest and abdomen were the most commonly used diagnostic radiological methods. Thirty-three duplications (79%) were spherical and nine (21%) were tubular. Multiple duplications were encountered in two patients (5.3%). Fourteen duplications (33%) contained heterotopic mucosa, mostly gastric type. More than one type of heterotopic mucosa in the same duplication was encountered in four duplications (10%). Additional malformations were encountered in 26 percent of patients. Six patients (15.8%) died from unrelated causes.

The signs and symptoms vary among duplications. Signs and symptoms leading to diagnosis and surgery varied according to the age of patient, location of the duplication, type of mucosal lining, duration of disease anad presence of complication. The ideal surgical treatment of duplication is complete excision. However, the other treatment options should be well known.

Key words: alimentary tract, cyst, duplication, gastrointestinal, tubular.

Alimentary tract duplications (ATD) are rare abnormalities in pediatric surgical practice. They are encountered in two or three cases per year, mostly in large pediatric surgical centers. However, this topic interests most pediatric surgeons, and many papers have appeared on this subject in the English language literature. Since they may occur at any location from the mouth to the anus, the spectrum of signs and symptoms is wide.

The management and treatment of ATD require familiarity with their clinical characteristics and anatomy. Therefore, we reviewed our experience on this subject and undertook a retrospective analysis of 38 cases with 42 ATD, treated at our center over a 26 year period from 1971 to 1997.

Material and Methods

During the 26-year period from 1971 to 1997, we encountered 42 ATD in 38 patients. Hospital

records, radiographs, operative reports and the surgical pathology of 38 patients were evaluated retrospectively. In the early years of this study period, ultrasonography (USG) and computed tomography (CT) were not available in our institute.

Results

There were 20 boys and 18 girls with a male to female ratio of 1.1. Nine patients (24%) were admitted to the hospital in the neonatal period. Seventeen patients (45%) were one to 12 months of age and 12 patients (31%) were older than one years. Thus, 69 percent of the patients were symptomatic under the age of one year. The signs and symptoms leading to diagnosis and the locations of duplications are given in Tables I and II.

Table I. Signs and Symptoms Leading to Diagnosis and Surgery

Signs and Symptoms	Number of patients
Feeding difficulty	1
Sublingual mass	1
Respiratory distress	7
Cough	1
Recurrent respiratory tract infections	1
Abdominal pain	5
Abdominal distention	6
Vomiting	10
Constipation	11
Massive rectal bleeding	1
Abdominal mass	10
Failure to thrive, loss of weight	2
Peritonitis	2
Bladder neck obstruction	1

Note: One patient with gastric duplication cyst and one patient with thoracoabdominal duplication were asymptomatic at the time of surgery.

Table II. Locations of Duplications

	Number of patients	%
Intraoral	1	2.6
Sublingual	1	
Intrathoracic	9	2.37
Esophageal	1	
Enteric	6	
Thoracoabdominal	2	
Intraabdominal	28	73.7
Gastric	1	
Duodenal	3	
Jejunal	2	
Jejunoileal	2	
Ileal	10	
Cecal	6	
Colonic*	2	
Rectal	2	

* One patient also had appendiceal duplication.
Note: One of jejunoileal and one of ileal duplication patients had multiple duplications.

Associated malformations were found in 10 patients (26%) (Table III). Six (15.8%) of the 38 patients died. All these deaths were before 1982 when broad spectrum antibiotics, intensive care facilities and new surgical techniques were not available. Four patients died of sepsis and one patient was complicated by midgut volvulus. One patient with multiple urological abnormalities died of chronic end stage renal failure.

Table III. Congenital Anomalies Associated with Alimentary Tract Duplications

	Number of patients
Encephalocele	1
Meningocele	1
Hemivertebra	1
Inguinal hernia	1
Malrotation*	4
Meckel's diverticulum	2
Ileal atresia	1
Bifid ureters	1
Complex urological abnormalities	1

* Two cases presented with midgut volvulus.

Discussion

Alimentary tract duplications can occur anywhere from the tongue to the anus, and have some basic characteristics. They are attached to at least one point of the alimentary tract, they have a well developed coat of smooth muscle and the epithelial lining of the duplication always resembles some part of the alimentary tract[1]. As one exception, intrathoracic duplications may lie adjacent to or distant from the esophagus without sharing a common muscular wall[2]. ATD may be asymptomatic for years or may present with life-threatening complications such as airway obstruction, pneumothorax, intestinal obstruction and perforation, and massive bleeding[3,4].

Numerous theories have been proposed to explain the occurrence of ATD, including the persistent vacuolization theory by Bremer, the presence of mucosal diverticula by Lewis and Thyng, incomplete coalescence of the lacunae that form between the epithelial cells of the solid core of the elongating gut, and faulty separation of the endoderm and notochord early in development proposed by McLetchie et al. and others[1,3]. Each can explain a particular abnormality, but the split notochord theory is an attractive explanation when the duplication is extensive and when vertebral anomalies are present.

Intraoral Duplications

Enteric duplication cysts of the tongue are unusual lesions (only 0.3 percent of the reported ATD) and are not usually associated with extraoral abnormality. During the formation of the lingual enteric cysts, trapping of epithelium by fusing primordia has been proposed[5]. Additionally, the close relation between the primitive stomach and anlage of the tongue in the midneck region at the same stage of development has been proposed for explanation of heterotopic gastric epithelium which is found in the lingual enteric cysts (enterocystoma)[6]. These lesions present with lingual mass that causes symptoms related to feeding difficulties, infection or bleeding. Lingual enteric cysts must be distinguished from dermoid cysts, hemangiomas, teratomas, hamartomas, thyroid remnants, ranulas and cystic hygromas.

We experienced only one case (2.6%) with a lingual enteric cyst at a sublingual location. This case presented with intraoral mass and feeding difficulty, and was cured by total excision without difficulty.

Intrathoracic Duplications

Intrathoracic ATD can be divided into three headings due to the anatomic location of the duplication: esophageal duplications (ED), enteric duplication cysts and thoracoabdominal duplications.

Esophageal duplications occur mostly in the distal third of the esophagus and locate in the posterior mediastinum, frequently in the right side. They have been reported to be the second most frequent location of ATD (20%) and most of the ED are spherical. Tubular ED may communicate with the stomach or proximal part of the small intestine via an esophageal or aortic hiatus[7-9]. Large cysts usually present with respiratory distress. Small ones may be found as asymptomatic masses on chest roentgenograms. Peptic complications such as hematemesis, hemoptysis, pneumothorax or empyema can occur, and rarely during intrauterine life[7] if they contain gastric mucosa.

Although chest X-ray, barium esophagogram and computed tomography of the thorax (Fig. 1) give sufficient information, endoultrasonographic diagnosis of ED has also been reported in adults[10].

At operation, the muscular coat of the duplication should be dissected without disturbing the esophageal muscosa[11]. We encountered only one ED (2.6%) of a spherical type. This case was an example for difficult surgery due to the closely attached spherical cyst. Perforation of the esophagus complicated our case. The patient required gastrostomy drainage for prolonged esophageal leakage and for feeding. The patient tolerated oral feeding after three weeks.

The second form of intrathoracic duplication is enteric duplication cysts which are not closely attached to the esophagus or intraabdominal part of the alimentary tract[2]. They are large masses and frequently present with airway obstruction and respiratory tract infection (Fig. 2).

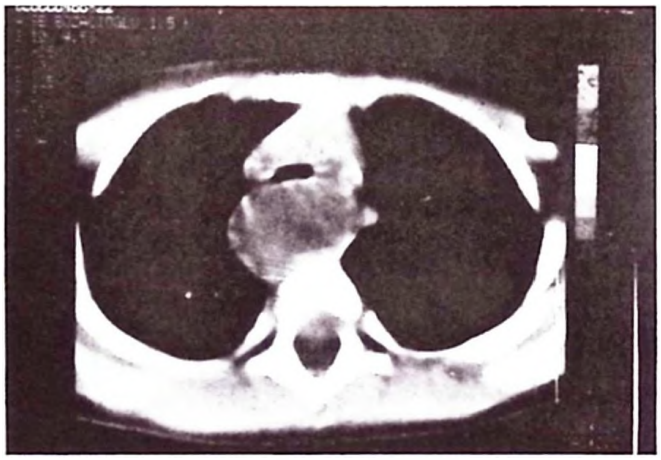

Fig. 1. Chest tomography showing duplication of the esophagus.

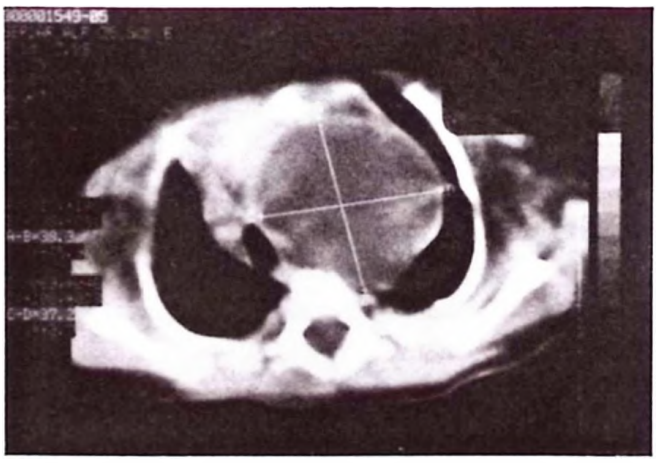

Fig. 2. Huge intrathoracic enteric duplication cyst causing severe mediastinal shift.

We encountered six patients (15.8%) with intrathoracic enteric duplication cysts, all treated by total excision. However, left subclavian artery and left phrenic nerve injury occurred in one patient. Postoperative diaphragmatic eventration was treated by plication one month later.

Thoracoabdominal duplications (TAD) of the alimentary tract are intrathoracic extensions of duplications that originate from the stomach or intestine. They are most likely tubular and are distinct from the esophagus, but may densely attached to the vertebrae and may extend into the neck[1,11]. The symptoms may be related to the thoracic or abdominal part of the duplication. It may be necessary to first excise the symptom-causing part. If there is no acute abdominal picture, the thoracic portion is dealt with first[1]. Any communication with vertebral body must be freed and sutured to prevent postoperative meningitis. If the abdominal procedure was required first, the thoracic procedure should not be delayed.

In our first TAD case, we used a transabdominal approach at first because of prediagnosis of a right-sided diaphragmatic hernia. The intrathoracic portion was excised later. The second case with TAD had undergone excision of a retroperitoneal tubular structure in another hospital. However, the intrathoracic extension of the TAD had been overlooked during the first operation. The patient had gone about nine years, interestingly without having symptoms.

Intrabdominal Duplications

Stomach

Gastric duplications (GD) comprise about 3.8 to 9 percent of all ATD. A third present in the neonatal period and most present within the first two years of life with abdominal mass, vomiting, weight loss and abdominal pain. A third of them have been detected in patients more than 12 years of age. Gastrointestinal bleeding was found in older infants and rarely in neonates[8-10,12-18].

Prenatal diagnosis is also possible in GD and the postnatal course may be asymptomatic[12]. According to the location of the GD, it may not be distinguished from choledochal cyst, duodenal atresia, neuroblastoma, congenital diaphragmatic hernia, mesenteric and surrenal cysts and genitourinary anomalies during USG examination[9,12].

The majority of GD are spherical, located along the greater curvature or posterior wall of the antrum and very rarely do they communicate with gastric lumen. Most of them contain gastric mucosa and sometimes pancreatic tissue[8]. Unusual presentations such as hemoptysis, recurring pancreatitis, and rare association with esophageal, duodenal, and jejunoileal duplications have been reported[8,9,14,16,17]. Adenocarcinoma and carcinoid arising from mucosa of GD have also been reported[18].

Ideally GD are treated by total excision. However, in some cases, due to their location or extensive size, subtotal excision with removal of mucosa or cystogastrostomy has been performed[18].

We encountered only one case of GD (2.6%) in whom prenatal diagnosis was made.

Duodenum

Duodenal duplications (DD) account for four to 12 percent of all ATD and occur with a frequency of 1 in 100,000 births[19-24]. In a large series of congenital duodenal obstruction, DD constituted only one percent of the cases[21]. These cysts most frequently develop in neonates or young children, are most often located in the first or second part of the duodenum and fail to comunicate with the lumen. Because of the close relation of these cysts to important structures, the spectrum of symptoms varies from one to another. The most common picture is duodenal obstruction in infancy; occasional patients later on. Unusual symptoms include relapsing pancreatitis and severe ascites, perforation, bleeding resulting from peptic ulceration or segmental portal hypertension, biliary tract obstruction, and hepatic or mediastinal mass[20,23-28]. Upper gastrointestinal series, USG, CT scan and endoscopic retrograde cholangiogpanceratography can be used to determine the location, size and communication to the pancreatic and biliary tract[23,24].

Surgical options in dealing with DD have varied depending on the location of the lesion and its proximity to, and involvement of , the ampulla of Vater, and the pancreatic ducts and biliary system. Such options have included cystectomy with or without duodenostomy or duodenojejunostomy; partial cystectomy and marsupialization to adjacent stomach, duodenum or jejunum, sometimes with removal of the mucosal lining; and pancreaticoduodenectomy (Whipple's procedure) in cases of cysts within the head of the pancreas[26]. Endoscopic removal of duodenal wall cyst and endoscopic drainage have been reported[24,25,29]. However, the exact diagnosis of DD cannot be made during endoscopic drainage procedures. Additionally,

the presence of heterotopic gastric tissue or malignant transformation of the cyst epithelium cannot be excluded.

We encountered three DD cases (7.9%). They presented with abdominal pain or abdominal mass. Total excision was possible in one patient and two were treated by mucosal stripping and cystoduodenostomy. All survived without later problems.

Small Intestine

About half of the ATD are located in the small intestine (SI). Pooled data from five large series of ATD revealed an ileal site in 41 percent and a jejunal site in 8 percent[30]. Most of the SI duplications are spherical with a diameter less than 10 cm. Tubular duplications represent five to 10 percent of SI duplications; they are rarely longer than 20 cm.

Intestinal duplications may present with vomiting, constipation, intestinal obstruction or as an abdominal mass. Additional presenting symptoms are chronic anemia, growth retardation, massive bleeding, pain and peritonitis. Combined distal ileal and total colonic tubular duplication associated with transmesenteric hernia, retrograde jejunoduodenal intussusception and melanosis peritonei are the rare presentations of SI duplications[31-33].

Heterotopic gastric tissue and communication with the normal bowel are frequently found. Heterotopic tissue other than gastric, such as squamous, transitional or ciliated mucosa, and pancreatic tissue can be found in SI duplications[34]. Congenital anomalies associated with SI duplications are omphalocele, intestinal atresia or stenosis and malrotation. In our series the most frequently asociated anomaly with SI dupliction was malrotation and Meckel's diverticulum. Malignancies have been reported occasionally in adults with SI duplication[35].

Ultrasonography and CT scan can be used for the diagnosis of spherical SI duplications (Fig. 3). Recently many long tubular duplications containing gastric epithelium have been diagnosed using 99mTc-pertechnetate scintigraphy[36-39]. We also diagnosed such a duplication by scintigraphy in a child presenting with massive rectal bleeding (Fig. 4).

Although intraabdominal tubular SI duplications longer than 50 cm have been reported very rarely[40], we encountered long tubular duplications of 80 cm and 100 cm in two cases.

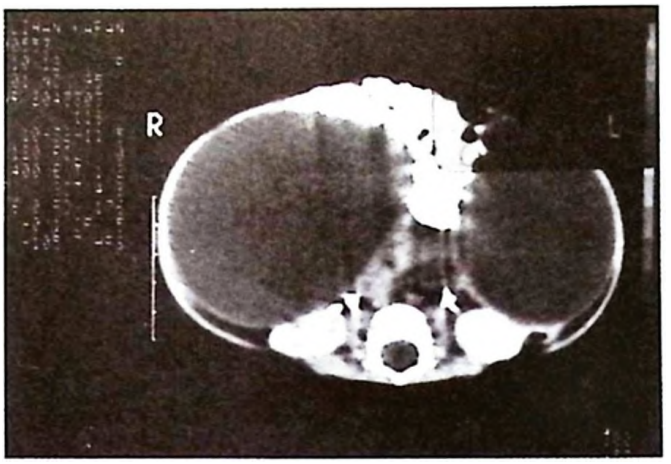

Fig. 3. Duplication cyst of the small intestine presenting with a large intraabdominal mass.

Fig. 4. Scintigraphic visualization of bleeding long tubular duplication.

When tubular duplications are very long, resection may lead to short-gut syndrome. Different surgical techniques have been used to avoid massive intestinal resection: partial resection and marsupialization of the remnant into the alimentary tract, anastomosis of the gastric-linked duplication into the stomach, selective devascularization of the duplication, surgical diversion to the normal bowel for drainage if there is no gastric mucosa, and anastomosis with mucosal stripping if a gastric lining is found, and two-step surgical resection[40]. Tubular duplication may pass in front or behind the mesenteric vessels and may sometimes be adherent to these structures. Very close and gentle dissection of the duplication spares vascular supply to the normal bowel. We were able to dissect long tubular duplications (80 cm and 100 cm) between mesenteric layer

in two cases. The distal 30 and 40 cm of these duplications shared a common wall with a normal bowel. In these cases we tried to dissect the duplicated segment by sacrificing terminal branches of intestinal vessels in only one side of the mesentery. If the duplicated segment cannot be dissected after a point, mucosal stripping should be tried. We did not attempt stripping in these cases but instead resected the terminal 10 and 20 cm of the duplications with the normal bowel and performed end to end anastomosis.

Approximately 20 percent of ATD are multiple[41]. We encountered multiple duplications in two cases (5.3%), one with double and the other with quadruplicate duplications. Therefore, intraabdominal cavity, root of the mesentery and bursa omentalis should be carefully checked for additional duplications.

Retroperitoneal duplications are very unusual. In a series of 78 cases of ATD only two were located in the retroperitoneum[42]. We did not encounter pure retroperitoneal duplication. However, the abdominal part of the duplication in our second TAD case was retroperitoneal.

Colon

Hindgut duplications constitute about 18 percent of all ATD. Twenty-five percent of reported colonic duplications were tubular and three quarters had a proximal, distal, or proximal and distal communication with the adjacent intestine. Those with distal communication tended to drain freely and therefore were associated with fewer symptoms[43,44].

Appendiceal duplication (AD) is a rare abnormality. However, with AD detected in childhood, the patient may have serious associated intestinal or genitourinary malformations[45,46].

Rectal duplications comprise only 2.5 to 3 percent of ATD[42]. Spherical duplications of the distal colon most commnly apear adherent to the posterior rectal wall. Patients may present with transanal prolapse or symptoms secondary to compression.

Duplications that affect the large intestine are commonly associated with genitourinary and anorectal malformations[47-49]. Genitourinary anomalies occur more frequently with tubular duplication of the entire colon. Bladder exstrophy is found 10 to 14 percent, genitourinary duplications are present in 21 to 43 percent, and spina bifida and other vertebral anomalies are seen in 15 percent of these patients[44].

We interestingly encountered cecal duplications (15.8%) as the most frequent location of colonic duplications. All these cecal duplications were spherical and embedded in the wall of the cecum and were very close to the ileocecal valve; dissection was impossible. Therefore, all these cases were treated by ileocecal resection and anastomosis. In one case, the patient presented with palpable mass and with no signs of intestinal obstruction. CT appearance suggested intussusception, and ileocecal intussusception due to cecal duplication was determined at operation (Fig. 5).

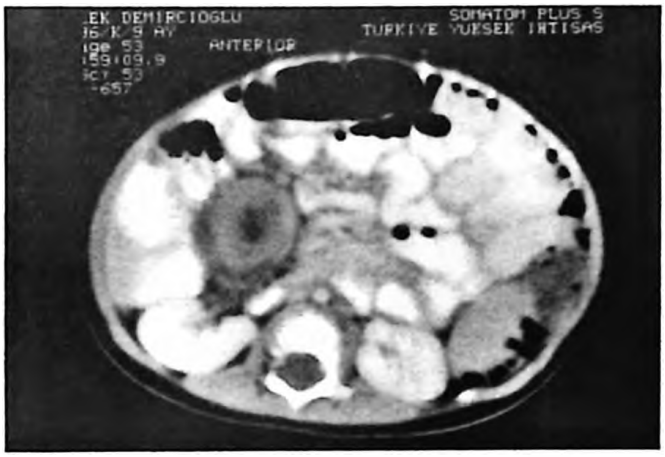

Fig. 5. Ileocecal intussusception due to cecal intramural duplication cyst.

The only case of tubular colonic duplication also had appendiceal duplication and multiple associated abnormalities leading to chronic renal failure. This child was managed by sigmoid colostomy but died of sepsis with end stage renal failure.

The most frequent location of ATD in our series (26.2%) was an ileal site. Intrathoracic enteric duplication cysts (15.8%) and intraabdominal cecal duplications (15.8%) were the second most frequent duplications. ATD present a wide spectrum of signs and symptoms. Although they are rare, one may encounter this abnormality in an unexpected case. Therefore, close familiarity with the anatomy of duplications and adequate knowledge of treatment options, which have to be chosen at operation, are required. Mortalities were encountered in the early years of this series and were closely related to associated abnormalities and sepsis, not to operations.

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