Congenital duodenal obstruction in neonates: a decade's experience from one center

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Original article

Background: **Congenital duodenal obstruction (CDO) is one of the most common anomalies in newborns, and accounting for nearly half of all cases of neonatal intestinal obstruction. This study aimed to review our single-center experience in managing congenital duodenal obstruction while evaluate the outcomes.**

Methods: **We conducted a retrospective analysis of the records of all neonates dianogsed with congenital duodenal obstruction admitted to our center between January 2003 and December 2012. We analyzed demographic criteria, clinical manifestations, associated anomalies, radiologic findings, surgical methods, postoperative complications, and final outcomes.**

Results: **The study comprised 287 newborns (193 boys and 94 girls). Birth weight ranged from 950 g to 4850 g. Fifty-three patients were born prematurely between 28 and 36 weeks' gestation. Malrotation was diagnosed in 174 patients, annular pancreas in 66, duodenal web in 55, duodenal atresia or stenosis in 9, preduodenal portal vein in 2, and congenital band compression in 1. Twenty patients had various combinations of these conditions. Presenting symptoms included bilious vomiting, dehydration, and weight loss. X-rays of the upper abdomen demonstrated the presence of a typical double-bubble sign or air-fluid levels in 68.64% of patients, and confirmatory upper and/or lower gastrointestinal contrast studies were obtained in 64.11%. Multiple associated abnormalities were observed in 50.52% of the patients. Various surgical approaches were used, including Ladd's procedure, duodenoplasty, duodenoduodenostomy, duodenojejunostomy, or a combination of these. Seventeen patients died postoperatively and 14 required re-operation.**

Conclusions: **Congenital duodenal obstruction is a complex entity with various etiologies and often includes multiple concomitant disorders. Timely diagnosis and aggressive surgery are key to improving prognosis. Care should be taken to address all of the causes of duodenal obstruction and/or associated alimentary tract anomalies during surgery.**

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Introduction

Congenital duodenal obstruction (CDO) is one of the most common anomalies in newborns, affecting approximately one in 2500 to 10 000 live births, and accounting for nearly half of all cases of neonatal intestinal obstruction.[1] Several intrinsic and extrinsic congenital lesions can lead to complete or partial obstruction of the duodenum, including embryologic defects in the development of the foregut, disturbances in recanalization or rotation, and abnormal embryologic relationships with neighboring anatomical structures.[2,3] Intrinsic duodenal obstruction can be caused by duodenal atresia, stenosis, or diaphragm, whereas extrinsic duodenal obstruction can be caused by annular pancreas, malrotation, or preduodenal portal vein.[4] Advances in surgical management, intensive care medicine, and postoperative nutritional support over the last decades have contributed greatly to improved survival of newborns with CDO. However, CDO still carries significant morbidity and mortality because of low birth weights, associated complex anomalies, and postoperative complications.[2,5] It is imperative to make a timely diagnosis and adopt an aggressive therapeutic strategy to maximize the outcome.

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We reviewed our experience in managing CDO in 287 neonates over 10 years, with a focus on characterizing the clinical, radiologic, and prognostic findings.

Methods

We conducted a comprehensive retrospective review of the clinical data of all infants with a diagnosis of CDO admitted to Children's Hospital of Zhejiang University Medical School between 2003 and 2012. Patients with abdominal wall defects (omphalocoele, gastroschisis) and diaphragmatic hernias accompanied by malrotation were excluded from the study. We recorded the history of the mother's pregnancy and the neonates' sex, birth weight, clinical manifestations, associated anomalies, details of preoperative examination, intraoperative findings, type of operative intervention, length of postoperative hospitalization, short- and midterm complications, and final outcomes. The follow-up period ranged from 6 months to 5 years. The project was approved by the Ethics Committee of the Hospital.

All data were collected, tabulated and compared, and numerical data were presented as mean±SD. Statistical analysis was completed using the Statistical Program for Social Sciences (SPSS, version 13.0, Chicago, IL) to perform analysis of variance (ANOVA) or the chi-square tests; $P<0.05$ was considered to be statistically significant.

Results

Between January 2003 and December 2012, a total of 287 neonates (193 boys and 94 girls) with CDO were treated at our hospital. Patients' age ranged from 1 to 28 days (mean, 8.42±6.54); mean gestational age was 37.84±2.62 weeks (range, 28-41), average birth weight 3048.33±625.40 g (range, 950-4850). Fifty-three patients were born prematurely between 28 and 36 weeks' gestation. Prenatal ultrasound scans revealed a fetal double-bubble signal in 67 patients (Fig. 1A). Neonates diagnosed prenatally were admitted to the hospital and treated much earlier than neonates who were diagnosed postnatally (mean, 3.15±2.11 days *vs.* 7.03±5.71 days, 6.08±4.49 days *vs* 11.36±6.58 days, respectively), and the differences were significant ($F=18.304$, $P<0.001$; $F=33.6$, $P<0.001$). Among the entire cohort, 174 (60.63%) patients were diagnosed with malrotation, 55 (19.16%) had duodenal web (type I duodenal atresia), 66 (23.00%) had annular pancreas, 7 (2.44%) had type III duodenal atresia, 2 (0.70%) were diagnosed with duodenal stenosis, 2 (0.70%) had preduodenal portal vein, and 1 (0.35%) had congenital band compression. In addition, 20 patients (6.97%) had various combinations of lesions, mainly malrotation in combination with duodenal web, annular pancreas, preduodenal portal vein, or duodenal atresia (Table 1).

Vomiting was the first presenting symptom in 86.06% of the patients (247/287), including bilious vomiting in 207 (83.81%). Other symptoms and

Table 1. Patients distribution in different group

Types of anomalies	*n*
Malrotation	154
Annular pancreas	62
Duodenal web	43
Duodenal atresia	5
Duodenal stenosis	2
Congenital bands	1
Malrotation with duodenal web	12
Malrotation with annular pancreas	4
Malrotation with duodenal atreasia	2
Malrotation with preduodenal portal vein	2

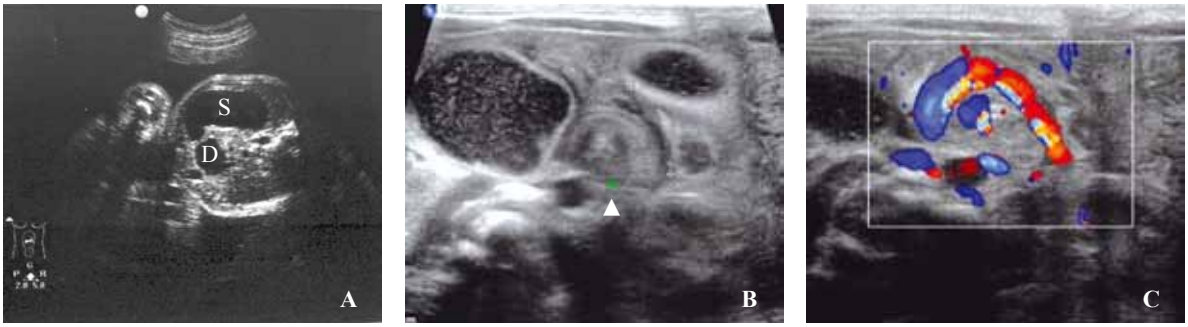

Fig. 1. Ultrasonic examination of duodenal obstruction. **A**: Prenatal sonographic evaluation demonstrated a typical fetal double-bubble signal (S: stomach, D: duodenum); **B:** The transverse sonographic imaging indicated the alternating rings of low and high echogenicity at the base of mesentery due to volvulus (cocentric circle sign); **C:** Color doppler ultrasonograph revealed inversion of the superior mesenteric artery and vein (the whirlpool sign).

signs included epigastric distention in 47 (16.38%), hematochezia in 25 (8.71%), dehydration and acid-base disorder in 85 (29.62%), azotemia in 17 (5.57%), weight loss (>20% birth weight) in 50 (17.42%), hyperbililubinemia (total bilirubin >255 μmol/L) in 88 (30.66%), and aspiration pneumonia in 9 (3.14%). Symptoms appeared within the first 24 hours of birth in 42.51% of the patients and by the third day in 64.46%. Patients with annular pancreas or duodenal stenosis or atresia (including duodenal web) exhibited symptoms earlier than patients with malrotation (mean 1.64±0.21 days *vs*. 3.98±0.54 days and 1.31±0.17 days *vs*. 3.98±0.54 days, respectively).

X-ray examination and/or ultrasonic evaluation were obtained to confirm the diagnosis. Of the 209 plain film abdominal radiographs, 143 (68.42%) demonstrated a typical double-bubble sign, single-bubble sign, or air-fluid levels in the upper abdomen (Fig. 2A, B). Other nonspecific findings included signs of a dilated stomach, less intestinal gas, and uneven intestinal gas distribution (Fig. 2C, D). Compared with malrotation, there were more positive findings on upright abdominal radiographs for annular pancreas and duodenal atresia/stenosis (87.80 % *vs*. 48.15%, 89.80% *vs*. 48.15%, respectively), and significant differences were observed (X^2=19.33, 24.61, respectively, P<0.001). Upper and/or lower gastrointestinal contrast studies were conducted in 64.11% patients to confirm the diagnosis. Abnormal findings included positioning of the duodenojejunal flexure to the right of the spine, a "coil spring" appearance of the proximal jejunum, and abnormal positioning of the ileocecal junction in patients with malrotation (Fig. 3A-C). Obstruction of the second or third part of the duodenum was present in patients with annular pancreas or duodenal atresia/stenosis (Fig. 3D-F). Among the 174 cases of malrotation, 113 had color Doppler ultrasound imaging, which revealed 81 cases of dilated duodenum with inversion of the superior mesenteric artery and vein (the whirlpool sign) (Fig. 1B, C).

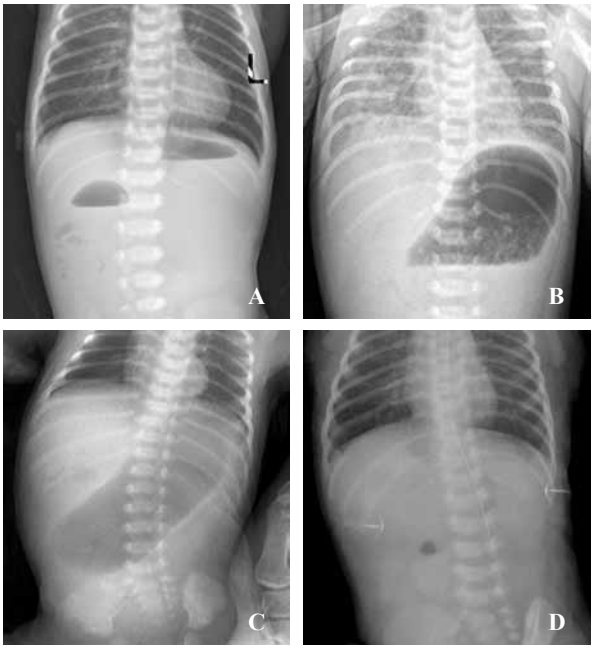

Fig. 2. Various appearance of duodenal obstruction under upright abdominal radiograph. **A**: Classic "double bubble sign" with less gas distributed in the distal intestine; **B**: Markedly dilated stomach fill with air and fluid displayed a "single bubble sign" in the upper abdomen; **C**: Massively dilated stomach without evidence of distal intestinal air; **D**: Uneven gas distribution. Intestinal gas mainly located in the left upper quadrant, and the rectal gas was found.

Half of the patients (145, 50.52%) had one or more associated anomalies. The majority of these were cardiac defects (46, 31.72%), of which atrial septal defect was the most common. Forty-three neonates had associated gastrointestinal malformation, and 22 patients had urogenital system abnormalities. Defects of the neural, orthopedic, vertebral, and endocrine systems were also detected. Nine patients had Down's syndrome.

Surgical repair depended on the type of anomaly. Ladd's procedure with and without intestinal resection was performed in cases of malrotation; duodeno-duodenoduodenostomy or duodenoplasty was performed for annular pancreas (diamond-shaped duodenoplasty) and duodenal atresia; and web resection and duodenoplasty were performed for duodenal web. Laparoscopic surgery was performed in 21 cases (including 10 cases of malrotation, 7 cases of annular pancreas, and 4 cases of duodenal web). Seven patients with megaduodenum (≥5cm in diameter) underwent triangle-tapered duodenoplasty. Combination procedures were performed in patients with multiple gastrointestinal anomalies.

Postoperative parenteral nutritional support was achieved over a period of 9.32±3.45 days. Patients started enteral feeding after an average of 8.51±3.17 days, and mean postoperative hospital stay was 14.87±5.60 days. Postopative hospital stay was significantly longer for annular pancreas compared with malrotation (mean, 15.02±4.67 days *vs*. 12.76±4.07 days; F=34.4, P<0.001), and for duodenal atresia compared with malrotation (17.38±6.22 days *vs*. 12.76±4.07 days, F=44.4, P<0.001).

Seventeen patients died postoperatively. The cause of death included prematurity, sepsis, pneumonia, brain hemorrhage, extensive bowel gangrene, short bowel syndrome, and other serious anomalies. Eight patients died of serious life-threatening abnormalities or of premature and its sequelae, such as intracranial hemorrhage and respiratory distress syndrome; 8 patients died mainly as a result of volvulus and bowel

Table 2. Postoperative causes of death

Diseases	Causes of death	*n*=17
Malrotation		
	Volvulus and intestinal necrosis, disturbances of electrolyte and acid-base balance, multiple system organ failure	6
	Intestinal atresia (type III & IV), short bowel syndrome	2
	Stomach perforation, peritonitis and sepsis	1
	Complex cardiac anomalies, intestinal aplasia	1
	Hypoxic ischemic encephalopathy and atrial septal defect	1
Duodenal web, atresia or stenosis		
	Complex cardiac anomalies, intracranial hemorrhage, meconium peritonitis	1
	Malrotation, intestinal atresia (apple-peel), pulmonary hemorrhage and disseminated inravascular coagulation	1
	Premature, very low birth weight infants, respiratory distress syndrome (RDS) and intracranial hemorrhage	1
Annular pancreas		
	Complex cardiac anomalies, Hirschsprung's disease and Down's syndrome	1
	Premature, RDS, anastomotic leak and peritonitis	1
	Aspiration pneumonia, sepsis	1

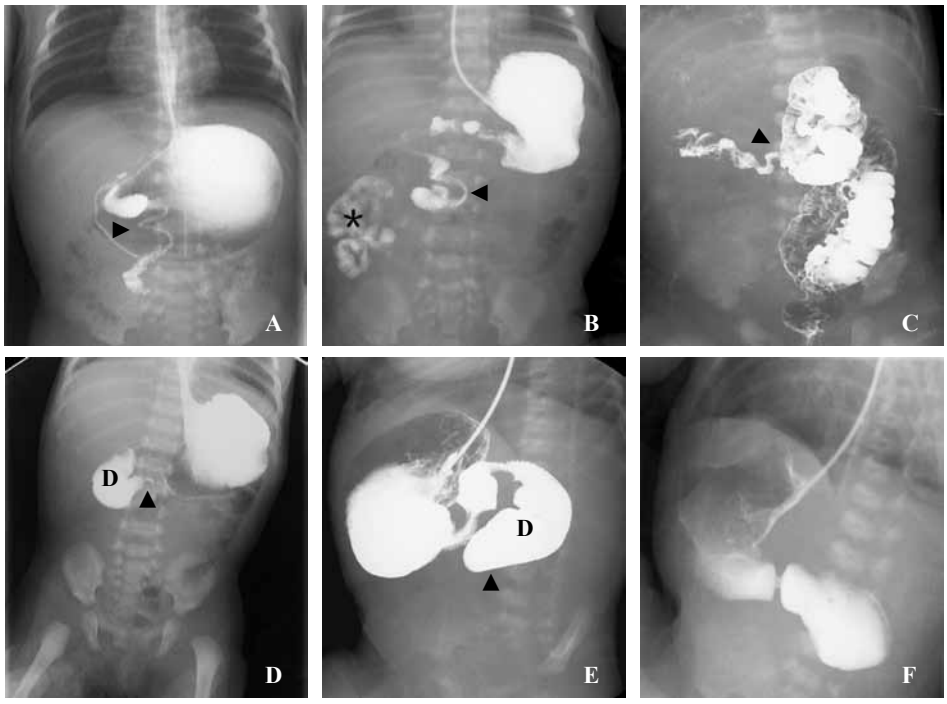

Fig. 3. Imaging of duodenal obstruction by gastrointestinal contrast study. **A&B**: Images on upper gastrointestinal series in a neonate with malrotation showed the position of the duodenojejunal flexure to the right of the spine and the "coil spring" appearance of the obstructed proximal jejunum (black arrowhead), the proximal jejunum located to the right of abdomen (asterisk in **B**); **C**: Barium enema illustrated the abnormal position of the ileocecal junction at the left upper quadrant (black arrowhead); **D**: GI series illustrated the dilated proximal duodenum (D) with a collapsed distal segment, and the location of contrast agent absence indicated the duodenal web with a tiny orifice (black arrowhead); **E**: Lateral view of an annular pancreas on GI series showed the second part obstruction of duodenum with an incisura at the lateral wall (black arrowhead); **F**: The image of markedly dilated proximal duodenum of a megaduodenum patient on the lateral view of upper gastrointestinal radiography.

necrosis, peritonitis and sepsis, aspiration pneumonia, disturbances of electrolyte and acid-base balance, and the complications resulting from delayed diagnosis. One patient died of anastomotic leak and peritonitis (Table 2). Surviving patients were followed up from 6 months to 5 years. Near- and mid-term complications were identified in 23 patients, and 14 patients required re-operation for wound dehiscence, adhesive intestinal obstruction, anastomotic leak, cholestasis, missed diagnosis of duodenal web, or recurrence of malrotation (Table 3).

Table 3. Main postoperative complications and causes of re-operation

Postoperative complications	*n*	Causes of re-operation	*n*
Wound infection	7	Wound dehiscence	3
Wound dehiscence	3	Missed diagnosis of duodenal web	3
Adhesive intestinal obstruction	4	Adhesive intestinal obstruction	2
Missed diagnosis of duodenal web	3	Anastomotic leak	2
Anastomotic leak	2	Malrotation recurrence	2
Malrotation recurrence	2	Cholestasis	2
Cholestasis	2		
Total	23		14

Discussion

CDO is a relatively common abnormality in newborns admitted to the neonatal surgical unit, affecting boys more frequently than girls. Failure to identify duodenal obstruction in the immediate newborn period may result in clinically significant metabolic disturbances, aspiration pneumonia, growth retardation, and even short bowel syndrome in cases of midgut volvulus. Delayed diagnosis is a significant cause of increased morbidity and postoperative mortality.[6,7] Although recent advances in surgical technology, neonatal intensive care unit medicine, respiratory support, and nutritional therapy have dramatically improved CDO outcomes, the relatively high incidence of postoperative re-operation and death still remains a major challenge for pediatric surgeons.[8]

CDO can be detected as early as twenty weeks of gestation, and the obvious benefits of prenatal diagnosis have been wildly recognized.[9] Antenatal diagnosis allows for parent counseling and pre-delivery planning, and enables additional to determine whether associated pathology is present. Adequate preoperative preparation and earlier surgery may help decrease overall morbidity, hospital length of stay, and costs.[10-12] In our group, 67 cases were diagnosed prenatally. The majority of these had serious obstructions, i.e., duodenal web/atresia, annular pancreas, or malrotation associated with intestinal atresia. As compared with those patients diagnosed postnatally, patients diagnosed prenatally tended to to have a decreased gestational age and lower birth weight at delivery, and a higher rate of associated serious congenital anomalies. Despite earlier diagnosis confirmed and definite operation performed, there were no significant differences in total outcomes, and the post-operative hospital stay seems longer. These results were similar to those reported by Cohen-Overbeek's.[13] We reasoned that a higher morbidity and mortality would be observed if the prenatal diagnosis was not achieved.

The postoperative re-operation rate for CDO is reported to be between 4% and 14%.[2,8] The main reasons for re-operation are overlooked combination lesions, anastomotic leak or dysfunction, adhesive obstruction, wound dehiscence, and gastroesophageal reflux disease.

Congenital duodenal obstruction is the result of several embryologic defects in foregut development, canalization, or rotation. Multiple factors likely contribute to this pathogenesis, and it is widely recognized that congenital duodenal obstruction has a high incidence of concomitant anatomic abnormalities. Despite this knowledge, missed lesions during the initial surgery are still the major reason for re-operation, especially in cases of malrotation and duodenal web or multiple weds.[2,8,14] Both duodenal web and malrotation cause incomplete duodenal obstruction and cannot usually be identified using preoperative gastrointestinal contrast. This kind of combination lesion is liable to be missed when performing Ladd's procedure without discovering the web or only removed proximal web but failed to find other webs. Obviously, the number of re-operations could be decreased with more thorough preoperative and intraoperative evaluation of the gastrointestinal tract and more careful attention to operative details. In our experience, we routinely irrigated the distal bowel with warm saline and pulled the nasogastric tube deeply into proximal jejunum during surgery to rule out distal obstruction, and we dissected suspicious sites when necessary. Our reoperative rate decreased dramatically in recent years after we fully realized this phenomenon and strengthened intraoperative exploration.

Anastomotic leak or dysfunction is another major cause of re-operation. In many CDO cases, the proximal duodenum is massively dilated and hypertrophied and demonstrates leather-like changes, and is always associated with secondary degeneration of ganglion cells and smooth muscle, features similar to those seen in cases of megaduodenum.[15] The dilated proximal duodenum not only affects anastomotic healing, but it also results in ineffective coaptation of the dilated duodenum with a consequent inadequate aboral pressure gradient and functional obstruction.[16] Delayed transit is one of the most problematic issues following duodenal obstruction repair, and is usually associated with a persistently dilated and dyskinetic proximal duodenum. The chronically obstructed duodenum cannot produce effective peristaltic waves, which may require prolonged total parenteral nutrition or surgical revision for persistent abdominal pain and failure to thrive. Previous studies[15,16] have demonstrated that tapering or contouring the duodenum may ameliorate these problems. In recent years, we have routinely performed a tapering duodenoplasty when megaduodenum (≥5 cm diameter) is present. After trimming the length of the duodenum laterally to avoid damage to the main blood supply of the duodenum and the major duodenal papilla, we used full-thickness, interrupted sutures to repair the duodenal wall and create an anastomosis. There were no duodenal leaks or other complications, and postoperative feedings were initiated within 7 days for all patients who had tapering duodenoplasty. Pathological analysis of the dilated proximal duodenal tissue identified vacuolar degeneration of the enteric neuron.

The rapid expansion of laparoscopic surgery in the pediatric population has led to decreased morbidity and a shorter hospital stay for a number of procedures. Most duodenal obstruction operations that were classically performed via open laparotomy can be

done endoscopically.[17,18] There have been several reports indicating that laparoscopic Ladd's procedure and duodenoduodenostomy can be performed with success rates equal to those achieved with laparotomy, without increasing operative time.[19-22] In the current series, we used laparoscopy in 21 patients in the last 3 years. One case required conversion to open laparotomy and 2 cases required re-operation due to postoperative volvulus and missed duodenal web. Although laparoscopic management of duodenal obstruction has proven to be safe and effective, it does have some evident disadvantages. The small size of the neonatal abdominal cavity affects the manipulation of laparoscopic instruments and results in prolonged operation time for beginners. It is difficult to widen the mesenteric base adequately and thoroughly assess the distal bowel for other atretic segments, resulting in a relatively high rate of conversion and re-operation.[23-25] We propose that laparoscopy is preferable for treating duodenal obstruction in select patients, providing that a low threshold for conversion to open surgery is maintained.

The mortality rate of duodenal obstruction has gradually decreased since Ladd published his classic article more than 70 years ago, and the survival rate now exceeds 90%.[26,27] The risk factors for death are complex cardiac anomalies, prematurity, sepsis, pneumonia, and surgical complications such as short bowel syndrome, anastomotic leak, and gastro-duodenal dysfunction.[2,27,28] Multiple congenital anomalies and sepsis are still the main risk factors for mortality, accounting for nearly 80% of all postoperative deaths. In the present study, nearly half of the 17 patients who died postoperatively suffered from intestinal necrosis, aspiration pneumonia, or severe electrolyte and acid-base imbalance when they were admitted to our hospital. These situations could be avoided or minimized if the parents paid more attention to neonatal vomiting and if the primary care physician fully recognized this disease. In our opinion, although multiple congenital anomalies and prematurity are the major factors influencing the prognosis for duodenal obstruction, which is an established fact that can not be altered artificially. Beyond this prerequisite, we should put more emphasis on the importance of early diagnosis and urgent surgical treatment. It is key to improving prognosis and reducing postoperative complications of CDO.

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Competing interest: None declared.

Contributors: Chen QJ wrote the first draft of this paper. All authors contributed to the intellectual content and approved the final version. Shu Q is the guarantor.

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