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Congenital Duodenal Obstruction: A Collective Review

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1. Abstract

1.1. Background

Intrinsic congenital duodenal obstruction (ICDO) is a common and unique congenital malformation. It is characterized by several important features including its variable presentation and the high incidence of associated anomalies. This is a review of our experience with 130 newborns, infants and children with ICDO outlining aspects of clinical features, associated anomalies, management and outcome.

1.2. Methods

Over a period of 28 years (January 1990 – December 2017), a total of 130 patients with ICDO were treated. Their medical records were retrospectively reviewed for age at diagnosis, sex, gestation, birth weight, clinical features, and associated anomalies, method of diagnosis, treatment and outcome.

1.3. Results

One hundred and thirty cases (66 males and 64 females) with ICDO were treated. Twenty-three of them presented beyond the neonatal period and the remaining 107 presented as newborns. The weight at presentation of the 107 ranged from 1.1 kg-3.8 kg (mean 2.4 kg). The mean maternal age was 25 years (18 years-40 years). Twenty -eight (21.5%) were premature and twenty-nine (22.3%) gave a history of polyhydramnios. Associated anomalies were seen in 74 (57%) of them. Forty-two (32.3%) had Down's syndrome and 31 (23.8%) had congenital heart disease. Twenty-five (19.2%) had rotational abnormalities of the gut including seven with situs inversus. Five (3.8%) had associated esophageal atresia and tracheoesophageal fistula. Intraoperatively, the causes of ICDO were duodenal atresia in 57 (44.9%), duodenal stenosis in 23 (18%) and duodenal diaphragm in 46 (36.2%). In 5 of them the obstruction was precipitated by eating dates with their seeds and the seeds resulted in obstruction of the hole in the duodenal diaphragm. Associated annular pancreas was seen in 27 patients (21.3%). In 18 this was associated with duodenal stenosis and in the remaining 9 it was associated with duodenal atresia. Two patients had congenital pyloric atresia associated with duodenal atresia at the fourth part of the duodenum. One patient was a rare case of ICDO, loss of the third and fourth parts of the duodenum with apple-peel configuration of remaining small bowel and absent superior mesenteric artery. Three patients died preoperatively and 127 were operated on. The different operative procedures were: Duodeno-duodenostomy in 60, excision of duodenal diaphragm and duodenoplasty in 31, Ladd's procedure, excision of duodenal diaphragm and duodenoplasty and appendicectomy in 9, Ladd’s procedure, appendectomy and duodeno-duodenostomy in 7 and duodeno-jejunostomy in 20. Ten had gastrostomy and 8 had trans anastomotic feeding tubes. Two had reduction duodenoplasty. Four required reoperations, 2 because of anastomotic leak and 2 because of duodenal dysfunction. Fifty-nine (46.5%) required total parental nutrition. Eleven patients died postoperatively giving a postoperative mortality of 8.7%. In all the cause of death was the severe associated anomalies and in 2 of them this was complicated by postoperative sepsis.

1.4. Conclusions

ICDO is a common congenital abnormality. The majority of these patients present in the neonatal period but sometimes the presentation of those with duodenal diaphragm with a hole is

delayed and this must be kept in mind. Rotational abnormalities of the gut including situs inversus are among the rare but interesting anomalies associated with ICDO. The presence of malrotation does not exclude the possibility of an associated ICDO which should be looked for intraoperatively. This is to obviate the possibility of a further subsequent operative treatment. The prognosis of ICDO is excellent but prematurity and severe associated anomalies continue to contribute to the prognosis.

2. Introduction

Embryo logically, ICDO is a primary malformation that results from errors in recanalization of the duodenum in early gestation. This is in contrast to other intestinal atresia which result from in utero vascular accidents. ICDO is a relatively common anomaly with an estimated incidence of 1: 3000 to 1:5000 live births [1-4]. Over the years, the prognosis of infants and children with ICDO has improved markedly. Several factors, however, still affect the overall outcome including prematurity, and a high incidence of associated anomalies [5-9]. It is also not uncommon for ICDO to be associated with other anomalies mainly congenital heart disease and Down's syndrome [5-11]. The clinical presentation of ICDO is variable depending on the site, type of obstruction and the presence of associated anomalies. These factors make ICDO a spectrum rather than a single anatomical malformation.

This report describes our experience with 130 infants and children with CDO outlining their clinical features, diagnosis, variable presentations, associated anomalies and outcome.

3. Patients and Methods

Over a period of 28 years (January 1990 – December 2017), a total of 130 patients with ICDO were treated. Their medical records were retrospectively reviewed for: age at diagnosis, sex, gestation, birth weight, history of polyhydramnios, presentation, associated anomalies, method of diagnosis, type of treatment and outcome.

3.1. Results

One hundred and thirty patients with ICDO were treated between 1990 to 2017. There were 66 males and 64 females. Twenty-three of them presented beyond the neonatal period and the remaining 107 presented as newborns. The weight of those who presented as newborns ranged from 1.1 kg-3.8 kg (mean 2.4 kg). The mean maternal age was 25 years (18 years-40 years). Twenty-eight of them were premature and twenty-nine gave a history of polyhydramnios.Associated anomalies were seen in 74 (57%) of them. Forty-two (32.3%) had Down's syndrome and 31 (23.8%) had congenital heart disease. Twenty-five (19.2%) had rotational abnormalities of the gut including seven with situs inversus. Five (3.8%) had associated esophageal atresia and tracheoesophageal fistula as shown in Table 1. All our patients were operated on except 3 who died preoperatively as a result of prematurity and multiple associated anomalies. The remaining 127 patients were operated on. The site of duodenal obstruction was located in the second part of duodenum in 124 (97.6%) of them. In two, the site of obstruction was at the fourth part of duodenum while in the third it was located at the third part of duodenum.Intraoperatively, the causes of ICDO were duodenal atresia in 57 (44.9%), duodenal stenosis in 23 (18%) and duodenal diaphragm in 46 (36.2%). One of our patients had duodenal atresia with apple-peel deformity. Associated annular pancreas was seen in 27 patients (21.3%). In 18 this was associated with duodenal stenosis and in the remaining 9 it was found to be associated with duodenal atresia (Figure 1). Forty-six were diagnosed to have congenital duodenal diaphragm and their age at presentation ranged from 1 day to as late as 3.5 years of age. Those with duodenal diaphragm who presented early had a complete duodenal diaphragm diagnosed intraoperatively and the remaining 22 were found to have congenital duodenal diaphragm with a central hole. In 5 of them the obstruction was precipitated by eating dates with their seeds and the seeds resulted in obstruction of the hole in the duodenal diaphragm (Figures 2a an 2b).Twenty-five of our patients had rotational abnormalities of the gut including 7 with situs inversus (Figures 3a and 3b). The diagnosis of malrotation was made intraoperatively but in those with situs inversus the abdominal radiographs showed the classic double-bubble sign but the stomach was on the right side of the abdomen and no air was found distally in 3. In the remaining 4 there was minimal air in the bowel distally. The diagnosis of situs inversus was confirmed by a preoperative ultrasound which showed the liver on the left side and the stomach and spleen on the right side. Preoperative echocardiogram showed a normally placed heart in all of them but in one there were features of Fallot's tetralogy. Intraoperatively, the diagnosis of situs inversus was confirmed and 3 of them showed complete duodenal atresia while in the remaining 4, the cause of duodenal obstruction was duodenal diaphragm with a hole.Two of our patients had congenital pyloric atresia associated with duodenal atresia at the fourth part of the duodenum. This resulted in a closed duodenal loop with pyloric atresia at one end and duodenal atresia at the other end with accumulation of biliary and pancreatic secretions. This resulted in duodenal perforation in one of them. In both, the diagnosis of associated duodenal atresia was made intraoperatively. One of them was operated on as a case of pneumoperitoneum most likely secondary to perforated necrotizing enterocolitis but intraoperatively was found to have congenital pyloric atresia with distal duodenal atresia forming a closed duodenal loop with duodenal perforation. The other patient had congenital pyloric atresia associated with duodenal atresia, jejunal atresia and a duplication cyst. This patient also had a closed duodenal loop because of pyloric atresia proximally and duodeno-jejunal atresia distally, but there was no duodenal perforation.One of our patients was an unusual and rare case of ICDO, loss of the third and fourth parts of the duodenum with apple-peel configuration of remaining small bowel and absent superior mesenteric artery (Figure 4).

Antenatal abdominal ultrasound showed polyhydramnios and double bubble sign suggestive of congenital duodenal obstruction. Clinically, there was no abdominal distension and the orogastric tube was draining bile-stained aspirate. There were also bilateral corneal opacities and microcephaly. Abdominal x-ray showed dilated stomach with double bubble sign with no air distally and upper contrast study confirmed the diagnosis of duodenal atresia. The patient underwent laparotomy which confirmed the diagnosis of congenital duodenal atresia just distal to the insertion of the biliary and pancreatic ducts. Distal to this there was absence of the remaining parts of the duodenum and superior mesenteric artery. The pancreas was normal and the bile duct was seen entering the duodenum. There was also an apple-peel deformity and jejunal end was found freely mobile in the abdomen. The small intestines were shortened and supplied by a single vessel in a retrograde fashion. An end-to-end duodeno-jejunal anastomosis was done as well as jejuno-colonic anastomosis and appendectomy. Post-operatively, the patient did well and started on feeds gradually which he tolerated.Five of our patients had ICDO associated with esophageal atresia and tracheoesophageal fistula. One of them had dysmorphic features, hydronephrosis, esophageal atresia and tracheoesophageal fistula, duodenal atresia and intraoperatively, he was found to have duodenal atresia, annular pancreas and preduodenal portal vein. The remaining 4 had esophageal atresia and tracheoesophageal fistula associated with ICDO. In all, the diagnosis of esophageal atresia and duodenal atresia was mad by failure to pass a nasogastric tube and the presence of a double bubble sign on abdominal x-ray. All of them underwent thoracotomy first to repair the esophageal atresia and tracheoesophageal fistula followed by laparotomy and duodeno-duodenostomy. In all there was complete duodenal atresia and three of them had gastrostomy.One hundred and twenty-seven of our patients were operated on and the different operative procedures are outlined in table 2. The operative procedures were based on the cause of duodenal obstruction, the site of the obstruction and the presence or absence of associated anomalies. In 10 a gastrostomy was added to the procedure and trans anastomotic feeding tube was placed in 8 patients. These were done in the initial part of the study but subsequently none of our patients had a gastrostomy or trans anastomotic feeding tube. In 2 of these patients, the trans anastomotic tube led to disruption of the anastomosis and these 2 patients underwent reexplanation and repair of the anastomosis. Two patients required reoperation and reduction duodenoplasty for duodenal dysfunction. Their hospital stay ranged from 10 days-28 days (Mean 19.3 days). Fifty-nine (46.5%) required total parental nutrition. Eleven patients died postoperatively giving a postoperative mortality of 8.7%. In all of them the cause of death was the severe associated anomalies and in 2 of them this was complicated by postoperative sepsis.

Table 1: Associated anomalies.

Associated anomaly	No.	%
Down's syndrome	42	32.3
Congenital heart disease	31	23.8
Rotational abnormalities of the gut	25	19.2
Anorectal malformations	8	6.2
Esophageal atresia and tracheoesophageal fistula	5	3.8
Hydronephrosis and other urological abnormalities	11	8.5
Syndactyly, Polydactyly and hypoplastic thumb	3	2.3
Duplication cyst	5	3.8
Meckel's diverticulum	3	2.3
Arterio-venous malformations	1	0.77
Eventration of right diaphragm	1	0.77
Congenital Pyloric atresia	2	1.5
Congenital leukemia	1	0.77
Meconium cyst	1	0.77
Jejuna atresia including apple-peel deformity	3	2.3

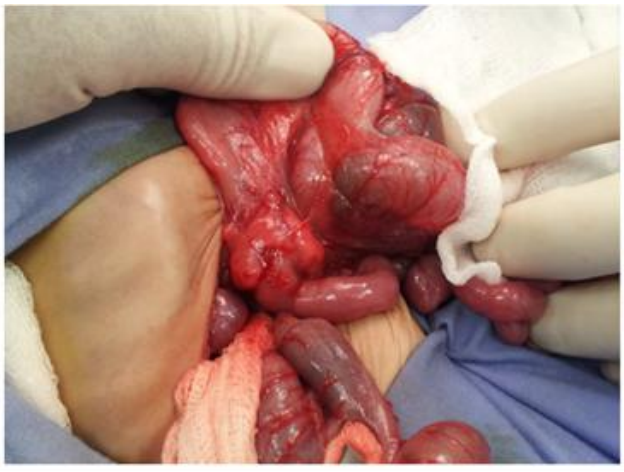
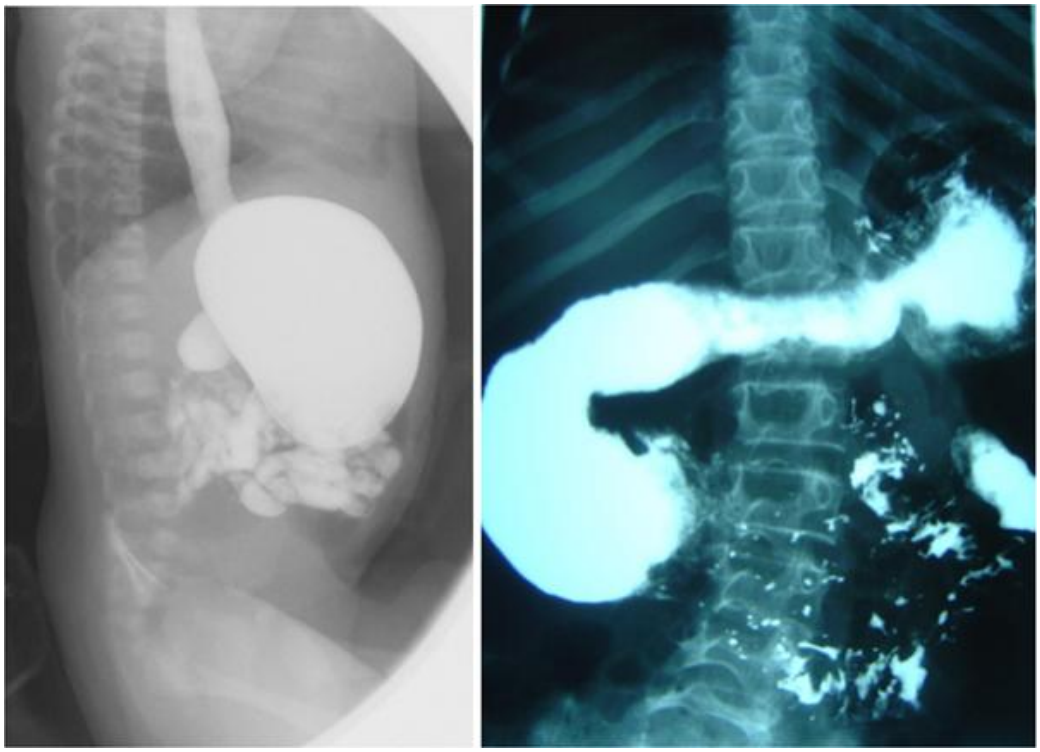


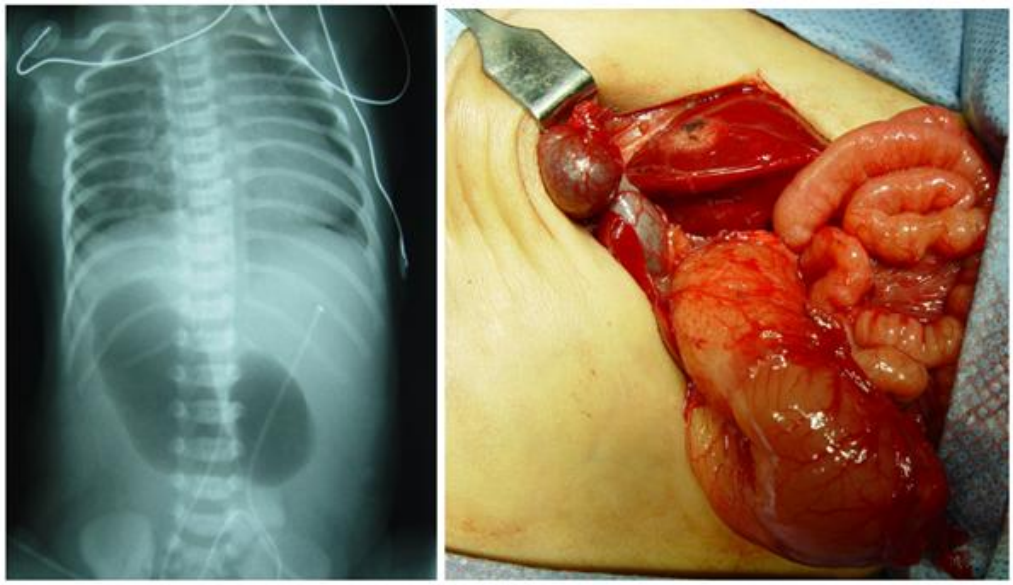
Figure 1: Intraoperative photograph showing annular pancreas associated with intrinsic duodenal obstruction.

Table 2: The different operative procedures.

Operative procedur	No. of patients
Duodeno-duodenostomy	60
Excision of duodenal diaphragm and duodenoplasty	31
Ladd's procedure, excision of duodenal diaphragm, duodenoplasty and appendectomy	9
Duodeno-jejunostomy	20
Ladd's procedure, appendectomy and duodeno-duodenostomy.	7
Duodeno-duodenostomy and appendicectomy	2
Gastrostomy	10
Trans anastomotic feeding tube	8



Figures 3a and 3b: Plain abdominal x-ray showing the double buble sign with the stomach on the right side of the abdomen. Note the intraoperaive photograph showing the liver on the left side of the abdome, the gallbladder is almost located in the midline and the dialted stomach with duodenum on the right side.



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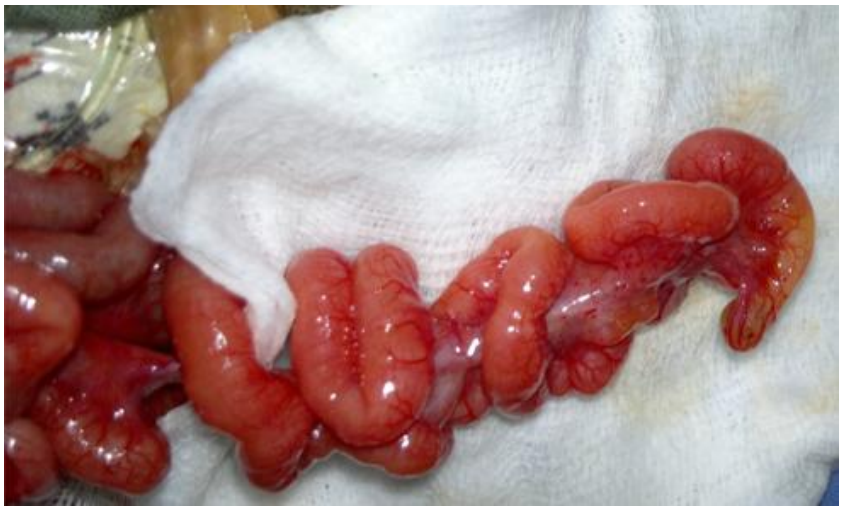

Figure 4: An intraoperative photograph showing apple peel deformity in association with congenital duodenal obstruction.

4. Discussion

Congenital duodenal obstruction is one of the common causes of intestinal obstruction in newborns. There are several causes for ICDO which are broadly divided into intrinsic causes and extrinsic causes. The intrinsic causes include atresia, stenosis, duodenal diaphragm with or without a hole. Extrinsic causes include malrotation with Ladd's bands and midgut volvulus, duplication cyst, annular pancreas and preduodenal portal vein [1,2,12,13]. Annular pancreas is included in the extrinsic causes but in our patients where annular pancreas was found there was an intrinsic cause for congenital duodenal obstruction. Associated annular pancreas was seen in 27 of our patients (21.3%). In 18 this was associated with duodenal stenosis and in the remaining 9 it was found to be associated with duodenal atresia. The presence of annular pancreas does not exclude an associated intrinsic cause of duodenal obstruction and it is best if these patients are treated with a duodeno-duodenostomy [14].An interesting group of our patients was those with duodenal diaphragm. Forty-six (36%) were diagnosed to have congenital duodenal diaphragm. All those with a complete duodenal diaphragm presented early and the diagnosis was confirmed intraoperatively. The remaining 22 were found to have congenital duodenal diaphragm with a central hole. It is this group of patients with duodenal diaphragm who can remain asymptomatic to present subsequently at an older age group or even into adulthood [15,16,17]. Five of our patients with duodenal diaphragm presented beyond 1 year of age with complete duodenal obstruction. The obstruction was precipitated by eating dates with their seeds and the seeds resulted in occlusion and obstruction of the hole in the duodenal diaphragm. This must be kept in mind when evaluating a child beyond the neonatal period with duodenal

obstruction.Congenital duodenal obstruction is a common congenital anomaly with an estimated incidence of in 1 in 5000–10,000 live births (5, 6, 7, 8). Previously we reported an incidence of congenital duodenal obstruction of about 1: 4000 live births from the Eastern province of Saudi Arabia which is similar to that reported internationally [14]. ICDO is known to be highly associated with other anomalies. Among these congenital heart disease and Down's syndrome are the commonest. In our study, 32.3% of our patients had Down's syndrome and 23.8% had congenital heart disease. It is important to document the associated congenital heart anomalies prior to surgery and all our patients with ICDO had an echocardiogram preoperatively not only to document the associated anomalies but also it is essential for the anesthesiologist to know these anomalies as they may also have a prognostic factor when it comes to the outcome [18-20]. It is well known that severe associated cardiac anomalies can have a major influence on the outcome of patients with ICDO [10,12,21,22].In our series, 32.3% of our patients had Down's syndrome which is similar to that reported from Western countries where congenital duodenal obstruction was reported to be associated with Down's syndrome in 30%-50% of patients [1,2,3,4]. This is in contrast to an interesting report from Taiwan which reported racial differences in the clinical characteristics of ICDO as their patients had a higher incidence of pre ampullary duodenal obstruction and lower incidence of associated Down's syndrome [23]. There is also an association between the incidence of Down's syndrome and maternal age. The incidence of Down's syndrome in our series was 32.3% which is lower than that reported by Akhtar and Guiney and the reason for this is the higher proportion of younger mothers in our series when compared to that reported by Akhtar and Guiney [6]. The mean maternal age of our patients was 25 years (18 years-40 years). It is also known that Down's syndrome negatively impacts the management and outcome of patients with ICDO [10,21].Another interesting association in our series was that of congenital duodenal obstruction and rotational abnormalities of the intestines. Twenty-five of our patients had rotational abnormalities of the gut including 7 with situs inversus. Sixteen had malrotation, two had non rotation and 7 had situs inversus. Situs inversus is extremely rare and commonly associated with other cardiac and splenic malformations [14,24,25,26]. The association of congenital duodenal obstruction and situs inversus is extremely rare. The diagnosis CDO in the presence of situs inversus can be made on plain erect abdominal x-ray which shows a double bubble sign but on the right side. Abdominal ultrasound is also valuable to diagnose situs inversus. The diagnosis however can be confirmed by barium meal and follow through. This is more useful in patients with partial duodenal obstruction secondary to a duodenal web with a central hole or duodenal stenosis. The treatment of CDO in association with situs inversus or malrotation is Ladd's procedure, duodeno-duodenostomy and in those with duodenal web, duodenotomy, excision of duodenal web and duodenoplasty. Some surgeons prefer a duodeno-duodenostomy in those with duodenal web to avoid injury to the biliopancreatic ducts because of the close proximity of the web to the ampulla of Vater. The presence of malrotation or non-rotation of the intestines with congenital bands should not exclude the possibility of an associated intrinsic congenital duodenal obstruction which should be looked for intraoperatively after a Ladd's procedure [14]. This is to obviate the possibility of further subsequent operative therapy in case this was missed during the initial surgery. We advocate adding an appendicectomy to the operative procedures in these patients. This should simplify their future management in case they present with acute abdomen as the possibility and confusion with acute appendicitis with its abnormal location is eliminated. One of our patients was operated on for congenital duodenal obstruction and interestingly intraoperatively he was found to have duodenal atresia, loss of the third and fourth parts of the duodenum with apple-peel configuration of remaining small bowel and absent superior mesenteric artery [27]. The concomitant occurrence of CDO and apple peel atresia is extremely rare and only few cases reported so far in the literature [28-31]. The possibility of Strømme syndrome which is rare consisting of apple peel intestinal atresia, ocular anomalies, microcephaly and developmental delay must be kept in mind also (32, 33, 34, 35). Our case is similar to those reported so far. They all had an atretic third and fourth parts of duodenum and proximal jejunum with typical apple-peel configuration of the ileum and total absence of superior mesenteric artery [28-31]. In these patients the pancreatic-biliary ducts drain into the proximal second part of the duodenum. In 1993, Strømme et al described a new syndrome consisting of apple peel intestinal atresia in siblings with ocular anomalies and microcephaly [32]. Our patient had a classic Strømme syndrome (microcephaly, apple peel intestinal atresia and ocular manifestations) but in addition our patient had duodenal atresia with absence of the third and fourth parts of duodenum and absent superior mesenteric artery.An interesting finding in two of our patients was the occurrence of congenital pyloric atresia with distal duodenal atresia. This will form a closed duodenal loop were biliary and pancreatic secretions will accumulate in this closed duodenal loop leading to massive distension of the duodenum. This resulted in duodenal perforation in one of our patients or sometimes the accumulated secretions can reflux back into the biliary tree leading to their dilatation [36,37]. The standard surgical treatment of ICDO is duodeno-duodenostomy [1,3,8,38,39]. This is done through the standard upper abdominal transverse incision, umbilical crease incision, or more recently laparoscopically [40,41,42]. With the recent advances in minimal invasive surgery, laparoscopic duodeno-duodenostomy which was shown to be safe and effective is likely to become the standard treatment of ICDO [43,44,45,46,48,49]. Kimura et al in 1977 described the diamond-shaped duodeno-duodenostomy which was

reported to be superior to simple duodeno-duodenostomy in term of early tolerance to feeds [50]. In none of our patients Kimura technique was used. A duodenal diaphragm can be managed by duodenotomy and resection of the duodenal web and duodenoplasty by closing the vertical duodenotomy transversely to avoid narrowing and stenosis of the duodenum at the site of duodenotomy closure. Care should be taken at the time of web excision because of the close proximity of the diaphragm to the ampulla of Vater and if in doubt a duodeno-duodenostomy is an alternative procedure. There are reports of successful endoscopic treatment of duodenal diaphragms [51,52, 53]. Currently, a duodenojejunostomy is not commonly performed and should be avoided due to its higher risk of long-term complications, such as delayed return of bowel function and blind loop syndrome. It is less physiological than a duodeno-duodenostomy and associated with more complications but it is a suitable procedure if the duodenal obstruction site is in the third or fourth parts of the duodenum. Duodenojejunostomy was used initially in our series but subsequently we stopped using this procedure. To obviate postoperative functional duodenal obstruction, we advocate reduction duodenoplasty in the initial procedure in those with megaduodenum. This was done in 2 of our patients and in both this was done as a second procedure.The use of trans-anastomotic feeding tubes in the post-operative period is still controversial. Recently, there were reports favoring the use of trans-anastomotic tubes as it shortens the time to full feeds and significantly reduce the need for central venous catheters, TPN and reduce cost [54-57]. Initially we used trans-anastomotic tubes in 8 of our patients but we stopped using trans-anastomotic tubes as our initial experience was not favorable. They tend to recoil and caused anastomotic disruption in 2 of our patients. Gastrostomy was also used in the initial part of the study in 10 of our patients but subsequently, none of our patients had gastrostomy as we felt it prolonged the operative time and was not necessary.In the majority of patients with ICDO, the prognosis is excellent. Over the years, the prognosis of infants and children with ICDO has improved markedly but several factors still continue to affect the overall outcome including prematurity, a high incidence of associated anomalies and reoperations [1,2,4,6,7].The reported survival rates in isolated CDO are 97–98% [6,7,8,20]. In the majority of the cases, mortality is due to associated conditions such as congenital cardiac anomalies and extreme prematurity [8,10,14,58]. In our series, 11 patients died postoperatively giving a postoperative survival rate of 91.3%. In all of them the cause of death was the severe associated anomalies and in 2 of them this was complicated by postoperative sepsis.In conclusion, ICDO is one of the common causes of intestinal obstruction in infants and children and continues to present unique management challenges. The majority of these patients present in the neonatal period but sometimes the presentation of those with duodenal diaphragm with a hole is delayed and this must be kept in mind. Rotational abnormalities of the gut including situs inversus are among the rare but interesting anomalies associated with ICDO. The presence of malrotation with congenital bands does not exclude the possibility of an associated intrinsic cause for CDO which should be looked for intraoperatively. The prognosis of ICDO has improved over the years but severe associated anomalies and prematurity continue to affect the overall outcome.

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