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Case Study

Duodenal obstruction in an infant a rare presentation

[1]Brig (Dr) S.M. Sharma, [2]Dr. M.S. Chahal, [3]Dr. Sujoy Mukherjee, [4]Dr Arjun Agarwal

[1]Professor Post Graduate Department of Surgery, Rohilkhand Medical College, Bareilly. 243006. UP
[2]Professor Post Graduate Department of Surgery, Rohilkhand Medical College, Bareilly. 243006. UP
[3]Associate Professor PG Department of Surgery Rohilkhand Medical College, Bareilly.UP
[4]Senior Resident, PG Department of Surgery Rohilkhand Medical College, Bareilly. UP

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ABSTRACT

A four and half month old female baby was admitted in our hospital with history of curdy white vomits and fever of seven days duration after normal delivery in a hospital . The parents of the infant observed peristaltic movements from left to right two months after delivery. The baby was kept on breast feeds which was followed by vomiting after each feed, the baby started having forceful projectile vomits after two months resulting in dehydration and passage of small hard stools. Clinical examination revealed an emaciated baby with tachycardia (HR 150/mt), good cry and no evidence of visible peristalsis after feeds. No lump could be palpated in epigastrium. CT abdomen was suggestive of duodenal stenosis resulting in dilatation of stomach. Gastrograffin studies revealed hugely dilated stomach and first part of duodenum. Exploration revealed a hugely dilated stomach and first part of duodenum which was compressed by an extra luminal tight thick band extending from pancreas to porta hepatis. Underneath this thick band intraluminal obstruction of first part of duodenum was found due to severe stenosis and obliteration by a diaphragm. Baby underwent duodeno-duodenal anastomosis after excision of extra luminal obstructing band with uneventful and exceptionally excellent post-operative recovery.

KEY WORDS: - Duodenal atresia, Duodenal stenosis, Duodeno-duodenal anastomosis, Duodenal obstruction.

INTRODUCTION

Duodenal obstruction is uncommonly encountered in neonates and infants. In 1773 Calder described two cases of duodenal obstruction due to duodenal atresia and both died. Fonkalsrud et al reviewed 503 cases of congenital duodenal obstruction treated between 1957 and 1967 [1]. Survival after surgery was recorded in 64% patients. Mortality after surgery was high and was attributed to associated congenital disorders, respiratory complications, prematurity and surgical complications. Survival for such cases in early 20 century was rare. More recent survival rates for infants born with duodenal atresia or stenosis are approximately 90-95 % [2, 3]. Increased survival rates can be attributed to advances in respiratory care, hyperalimentation, improved pediatric anesthesia, improvements in the recognition and management of associated anomalies, and more refined surgical techniques (e.g., the diamond-shaped anastomosis [4]. Common causes of duodenal obstruction are duodenal atresia and duodenal stenosis. Duodenal atresia is more common and occurs in 1 in 2000-5000 live births. Duodenal atresia presents within first twenty hours of life with non-bilious or bilious vomiting depending upon the obstruction being above or below the papilla of Vater. Duodenal stenosis however` may have delayed presentation with vomiting and the neonate becomes dehydrated, develops electrolyte imbalance and fails to thrive and develop normally.

Operative technique for duodenal lesion depends upon the anatomical cause of obstruction. Duodenal atresia and duodenal stenosis are best managed by duodeno-duodenal anastomoses which results in early recovery as compared to duodeno-jejunal anastomosis. The baby must be prepared for surgery by oro gastric aspiration, intra venous fluids, antibiotics and parenteral nutrition through peripherally inserted central venous catheter and when stabilized are taken up for intervention. We present here a case of an infant who presented to us with complete duodenal obstruction due to external

***Corresponding Author Address:** Brig (Dr) SM Sharma, Professor PG Department of Surgery, Rohilkhand Medical College & Hospital, Pilibhit By Pass Road, Bareilly 243006. UP; E-mail surenio31@hotmail.com*

compression by a thick band and severe duodenal stenosis. Infant was successfully managed by modified technique for duodeno-duodenal anastomosis in which anastomosis was achieved by giving longitudinal incision in the duodenum proximal and distal to obstruction and achieving continuity by approximating the openings transversely by interrupted sutures making it a true diamond shaped repair with resultant fastest return of normal gut function in couple of days as compared to delayed recovery among those operated by other techniques. We name it is as Surender's modified technique.

The objective of highlighting this case is delayed presentation of duodenal obstruction in an infant with rare occurrence of duodenal obstruction by both luminal lesion and extra luminal tightly compressing band. The modified technique of fashioning duodeno-duodenal anastomosis over longitudinal duodenotomy on both sides of obstruction has definite advantages over other procedures.

CASE REPORT

A four months and nine days old first born female infant was admitted to our hospital with history of onset of fever four hours after birth and non-projectile vomiting after feeds six to seven times a day. Earlier the infant was admitted to a hospital where she was treated for fever and vomiting which reduced the frequency of vomits to two to three times a day. Mother observed baby having occurrence of peristaltic waves from left to right after feeds one month after birth. Before reporting to our hospital the baby was treated in a few hospitals for unrelenting vomiting and was diagnosed having gastro-oesophageal reflux disease. Four months after birth, baby had increased frequency of projectile vomits twenty to twenty two times a day. The vomits were curd like to begin with but subsequently became coffee ground in colour accompanied with offensive smell. The infant passed scanty hard stools once in a day only.

On examination the infant was found to be poorly nourished and emaciated with a birth weight of 3.9kg, head circumference 37 cm, heart rate 132 beats per minute, respiratory rate 34 per minute with no clinical evidence of associated clinical congenital abnormality. The baby was found to be irritable, crying and hungry and could accept more than 100 ml of top feed in one sitting which was vomited out after every feed. During hospitalization in our center no visible peristalsis after feeds was seen. No visible or palpable lump was noticed in epigastrium since birth. No associated congenital abnormality was detected.

Investigations revealed Hb 11.8 gms/%, TLC 12400 cu/mm, DLC P 64%, L 30%, E 02%, M 04%, Platelets $243x10^3$/Ul, BT $2^{/}$, CT $4^{/}.30^{//}$, Serum Na^+ 124 meq/L, K^+5.8 meq/L. on admission and Na^+ 135 meq/L and K^+ 4.3 meq/L two days after admission and resuscitation. Ultrasound abdomen was noncontributory. CECT abdomen was suggestive of Hypertrophic pyloric stenosis with marked dilatation of contrast filled stomach and pyloric lumen narrowed. Gastrograffin studies revealed contrast filled dilated stomach and duodenum with minimal to little contrast in distal gut. The gastrograffin study revealed double contrast shadow in stomach and duodemum.

Baby was resuscitated with intravenous fluids, oxygenation, antibiotics and was taken up for surgery under GA four days after admission as the baby was being managed by paediatricians for gastro oesophageal reflux disease. A five cm long transverse incision was given at right upper quadrant of abdomen one cm above the umbilicus and extending laterally. The stomach was found to be hugely dilated. Kocherisation of duodenum and upper jejunum was performed and entire gut screened. First part of duodenum was tightly obstructed by a thick tubular looking band approximately 4 mm wide extending from head of pancreas to sub-hepatic region. Aspiration of this structure was negative for bile or blood. Head of pancreas was mobilized and thick band was separated from underlying compressed duodenum, the band was divided safely away from duodenal wall and sent for histopathological examination. Underneath the band an intra luminal firm thick obstruction was palpable between the thumb and index finger in first part of duodenum. A small 1.5 cm long incision was made in the first part of duodenum proximal to obstruction anteriorly along the long axis. The feeding tube could not be negotiated through the intraluminal obstruction of duodenum into its distal part. Saline and air could not be pushed beyond the obstruction into the distal duodenum due to complete obstruction. Another 1.5 cm long longitudinal incision was made in the duodenum anteriorly in its long axis beyond the intraluminal obstruction. Anastomosis between the proximal and distal duodenal openings was established in diamond shaped fashion comfortably transversely without tension with interrupted 3/0 RB Vicryl in single layer. Efficacy and patency of anastomosis was checked with injection of free flow of fluid through anastomotic site into straightened distal gut thus excluding existence of distal atresia or stenosis. Abdomen was closed with mass ligatures and skin closed with subcuticular absorbable 3/0 Vicryl. Patient was transfused 200

ml of blood on table during surgery however there was minimal blood loss. On first post op day the baby had fever of 103^0 F, heart rate 138 beats per minute and oxygen saturation at 95%. On second post op day the baby was transfused 50 ml of packed RBC's and was given intermittent oxygen 2 liters per minute. Aspiration through naso gastric tube was 200 ml on third post op day. On third post op day baby had heart rate between 120-128 per minute and respiratory rate 30 per minute. Baby passed normal stools on third post op day itself and was given 1 ml fluid orally 6 hourly and was simultaneously maintained on intra venous fluids 16 ml per hour. On fourth post op day oral milk feeds were increased to 10 ml six hourly and intravenous Isolyte-P was infused at the rate of 16 ml hourly. The baby continued to receive intravenous Cefotaxime 200 mg, Amikacin and Metronidazole in appropriate doses eight hourly. On sixth post op day oral milk feed was increased to 15 ml three hourly and on eighth post op day the feeds were increased to 25 ml three hourly. Baby started passing yellow coloured normal stools twice a day and was discharged from the hospital after ten days. Post op follow up revealed a healthy baby accepting feeds orally and passing normal stools with well healed operation scar mark.

DISCUSSION

Duodenal atresia and duodenal stenosis are main causes of duodenal obstruction in new born babies and infants. Differential diagnosis of duodenal obstruction also includes malrotation of midgut with volvulus, annular pancreas, pre duodenal portal vein, intestinal atresia, duodenal duplication, congenital bands, foreign body obstruction and gastro oesophageal reflux disease. Duodenal atresia results in complete obstruction and can be detected prenatally in 32-57 of patients. Approximately 30% of cases are associated with Down syndrome, and 23-34% of cases are associated with isolated cardiac defects. Esophageal atresia may be present in 7-12% of patients [5]. A healthy new born baby should have less than 5 ml of gastric aspirate. Congenital intestinal obstruction is associated with gastric aspirates that measure greater than 30 Ml [6]. Such babies should be investigated for duodenal atresia and other causes of intestinal obstruction.

Upper intestinal obstruction results in relentless vomiting after feeds and baby found perpetually crying and hungry for feeds. Baby starts vomiting within 24 hours after birth however it may be delayed for a few days more. Generalized abdominal distension is a feature of lower intestinal obstruction. In upper intestinal obstruction like duodenal atresia and stenosis fullness in epigastrium may be observed due to dilated stomach and duodenum. Duodenal stenosis due to incomplete obstruction may have delayed presentation with recurrent vomiting, aspiration and failure to thrive. Some patients may present in adulthood with gastro-oesophageal reflux disease, peptic ulcer and obstruction proximal to stenosis due to bezoar. The duodenal atresia and stenosis affects the first or second part of the duodenum. The papilla of Vater may open into intraluminal mucosal fold and hence is of great significance during surgery to preserve it. Together, both duodenal atresia and stenosis comprise a frequent cause of intestinal obstruction in the newborn [7].

Pathophysiology: In 1900, Tandler described the traditionally accepted theory on the normal development of the duodenum [8]. Duodenum develops from distal part of foregut and proximal part of midgut. Duodenal maldevelopment occurs due to inadequate endodermal proliferation. During 30-60 days gestation duodenal epithelium proliferates completely packing it with solid cells and occludes the lumen. Between 8-10 weeks duodenum starts getting canalized by a process of vacuolation prompted by apoptosis or programmed cell death. Arrest of vacuolation may lead to duodenal obstruction. Duodenal atresia is also associated with annular pancreas which surrounds duodenum completely and may be result of failure of development of duodenum itself.

Duodenal atresia can take many forms, but proximal and distal intestinal segments always end blindly [9]. Duodenal atresia has been described by Gray and Skandalakis into three types.

Type 1: In this type of deformity duodenum is blocked by mucosal and submucosal tissue which may be elongated further into duodenum and resulting in windsock deformity due to effects of peristalsis. Stomach and duodenum proximal to deformity is dilated.

Type 2: Proximal and distal atretic segments are connected by a fibrous cord.

Type 3: There is a complete separation with a gap between the proximal and distal segments of duodenum. Most of the biliary duct anomalies associated with duodenal atresia are observed in type 3 defects [10].

Lower biliary tract and pancreatic anomalies like stenosis and duplication of distal bile duct, annular pancreas and choledochal cyst are associated with duodenal stenosis and atresia. Air in distal duodenum and gall bladder on plain radiography is suggestive of bifid common bile duct. Double duodenal atresia or stenosis is less frequently reported [11].

Lab investigations: Routine haematological investigations including haematocrit which indicates the oxygen carrying capacity. Blood urea and serum electrolyte studies are mandatory as the neonates lose large amount of fluids and electrolytes in gastric aspirate and vomiting. Blood glucose levels as the neonates with duodenal obstruction are associated with premature delivery. Premature babies have limited glycogen stores and are prone to become hypoglycaemic. Blood grouping and cross matching.
Karyotype analysis. Duodenal atresia is associated with trisomy 21 in 30% of cases [12].

Imaging studies: Prenatal ultrasonography to detect duodenal obstruction, gastric and duodenal dilatation. Prenatal diagnosis of duodenal obstruction helps the parents and the surgeon to manage the baby appropriately after birth. Plain abdominal radiography shows air in dilated stomach and first part of duodenum, the typical double bubble gas shadow. Insufflation of air after aspiration makes the double sign more prominent.In duodenal atresia no gas distal to obstruction is visible.

Contrast study is helpful in diagnosing duodenal stenosis, malrotation, volvulus, annular pancreas, duplication of duodenum and duodenal web. Small amount of scattered air distally denotes duodenal stenosis and absence of gas in distal gut is diagnostic of atresia.
Echo cardiography to find out cardiac lesions.
Ultrasonography of abdomen to exclude renal lesions.

Surgery: Duodenal atresia and duodenal stenosis are managed by duodeno-duodenal ansatomosis which gives excellent results as compared to duodeno-jejunal anstomosis which results in complications and delayed restoration of gut function. Type 1 duodenal atresia can be managed simply by duodenotomy and web excision. Various techniques have been adopted to fashion the duodeno-duodenal anastomoses to operate upon cases with duodenal atresia and duodenal stenosis.

Pre OP assessment: Baby should be examined and investigated thoroughly at the earliest for duodenal obstruction and associated co-morbidities involving heart, lungs and kidney. Correction of tracheo-oesophageal fistula should be done prior to rectify duodenal obstruction. Restoring duodenal continuity in a case of duodenal obstruction before managing trachea-oesophageal fistula will result in failure of anastomoses with leakage.
Prior to surgery orogastric aspiration should be achieved and baby resuscitated with appropriate intravenous fluids, parenteral nutrition and antibiotics. Fluid and electrolyte imbalance must be corrected and normalized and baby exhibited 1 mg Vit K. Baby should be kept warm and hypothermia avoided throughout the period of hospitalization. Improved anaesthetic techniques have reduced the mortality and morbidity due to surgery in neonatal group.

Abdomen is opened through a right upper quadrant incision starting 2 cm above the umbilicus and extending 5 cm laterally. Abdominal muscles are divided transversely. Liver retracted superiorly and abdominal cavity is searched for associated abnormalities. Duodenum and jejunum are easily mobilized by Kocher maneuver to make tension free duodeno-duodenal anastomoses. Orogastric tube is advanced through proximal duodenotomy wound and gets arrested at the site of obstruction. Proximal duodenum and stomach are thickened and dilated, the distal gut is contracted. Most commonly adopted procedure now a days is fashioning diamond shaped anastomoses between the two segments of duodenum proximal and distal to obstruction. Viscera are examined for other anomalies including mal-rotation, annular pancreas, anterior portal vein. Windsock abnormality due to duodenal web forms an indentation which marks the site of origin of web. Papilla of Vater can be identified through duodenotomy after pressing the gall bladder and detecting flow of bile.

In creating diamond shaped anastomoses a transverse incision in duodenum proximal to obstruction and another incision of equal length in duodenum distal to obstruction is made in longitudinal direction. Duodeno-duodenal ansatomoses is fashioned by anastomosing transverse proximal duodenotomy wound with distal longitudinal duodenotomy. Anastomoses is accompliahed with four zero Vicryl sutures. During the diamond-shaped anastomosis, the midpoint of the proximal incision is approximated to the end of the distal incision [13]. In simple terms the anastomoses is fashioned by approximating midpoint of distal leaf of proximal transverse incision to midpoint of proximal leaf of distal incision in duodenum and suturing it transversely in a diamond shaped fashion till the corners followed by approximating the proximal leaf of proximal duodenal segment with distal leaf of distal duodenotomy wound. Abdomen is closed by 4 0 Vicryl including peritoneum and anteriror and posterior sheath. Skin is closed with 5 0 subcuticular suture. A direct duodeno-duodenostomy is believed to result in a relatively earlier recovery of anastomotic function when compared with results following duodeno-jejunostomy [14]. Duodeno-duodenostomy creates

wider stoma and early return of gut function. Duodenal web can be tackled by duodenotomy and removing the web laterally leaving medial end of web intact to avoid damage to papilla of Vater. In case proximal duodenum is dilated disproportionately, antimesenteric reduction duodenoplasty by excising the excess duodenal should be done to avoid megaduodenum and asymmetry of anastomoses. Alternately plication of excess duodenum by interrupted sutures over a dilator is recommended. It is rare to have an anastomotic leak following effective repair made by interrupted 40 sutures. Annular pancreas if present is not divided for fear of creating pancreatic fistula and instead side to side diamond shaped anastomoses is performed anterior to it. In malrotation of gut Ladd procedure is adopted, Ladd peritoneal bands are divided, small mesentery is widened, appendecetomy performed and caecum and colon are placed on the left side. Although gastrostomy tubes were often used in the past, complications associated with their placement and long-term problems with gastroesophageal reflux (following gastrostomy) have prompted the authors to avoid these adjuncts, except in cases where gastrostomy is likely to be needed in the future (ie, an infant with trisomy 21 and complex congenital heart disease) [15].

A trans anastomotic 5 F feeding tube can be negotiated for the purpose of enteral feeding after surgery. The feeding tube may get proximally displaced. Since the baby is likely to have prolonged ileus and would require fluids including pareneteral nutrition for the duration, therefore central venous catheterization should be established. The anomalies and defects can also be managed by laparoscopic method with an umbilical port which is used for gas insufflation and 3mm and 5 mm ports placed in right lower quadrant and left mid quadrant respectively. Another 5 mm port can be placed in right upper quadrant to retract the liver. Surgeon operates from the foot end of the patient. After duodenum is Kocherized diamond shaped anastomoses is established with the help of interrupted sutures or u clips. The distal gut must be screened for co existing atresia or stenosis during laparoscopic intervention.

Post operatively the stomach is kept empty by intermittent orogastric suction till the aspirate falls below 1 ml/kg/hr. Baby is maintained on intravenous fluids and oral fluids are started only when orogastirc aspirate minimizes and the baby has passed stools. Mortality rate after surgery for duodenal defects has fallen due to improvement in paediatric anaesthesia and surgical techniques, improved ventilator methods, better nutritional support and use of surfactants.

The result of operating this infant by modified technique of duodeno-duodenal anastomosis over longitudinal incisions made proximal and distal to obstruction was excellent in terms of its simplicity and recovery of normal function. Baby passed normal stools on third post op day and oral feeds restored.

CONCLUSION

With better techniques neonates and infants with congenital duodenal obstruction have more than 97% chances of survival. Mortality if any is due mainly to co- existent cardiac defects. Babies with duodenal stenoses present with vomiting, dehydration, failure to thrive and marked duodenal dilatation, visible peristalsis and radiological evidence of dilated duodenum and stomach with classical double bubble appearance. Resuscitation with intravenous fluids including parenteral nutrition and antibiotics followed by diamond shaped duodeno-duodenal anastomoses should be undertaken. With adequate stoma the gut function is restored to normal within a few days and baby can be given oral feeds after orogastric aspiration falls to minimum volume and baby has passed stools. Abdomen is opened by right upper quadrant transverse incision. Patient can also be operated by laparoscopic method depending upon the technical expertise and confidence of the surgeon, weight of the baby more than 2.5 kg and absence of cardiac abnormality as pneumoperitoneum can affect the cardio-vascular status of the infant. Excision of duodenal web by duodenotomy preserving the portion at the site of Vater may not be adequate and therefore duodeno-duodenostomy is more appropriate. Gastrostomy in such cases for aspiration or feeding adds to morbidity and may not be fruitful. Advancing the orogastric tube beyond the duodeno-duodenal anastomostic site will splint the ansatomoses and also may be used for feeds and its position should checked radiologically.

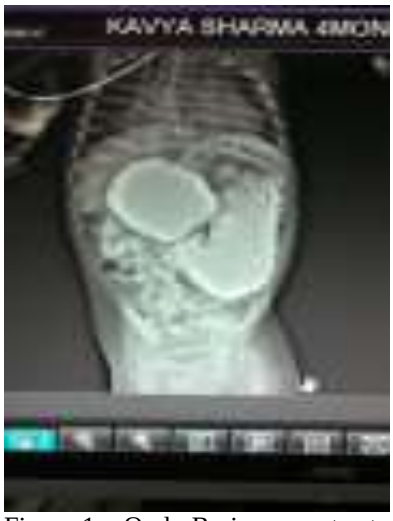

Figure-1: Oral Barium contrast shows dilated stomach and first part of duodenum.

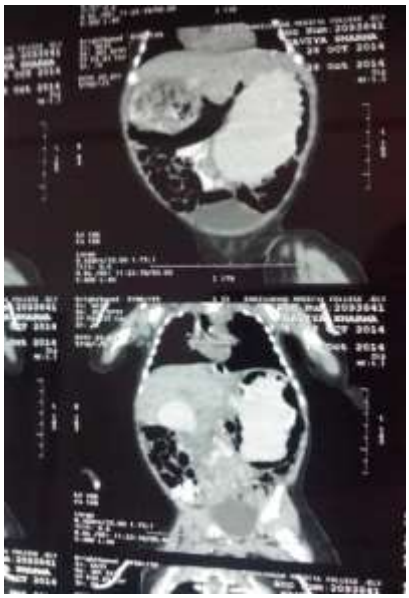

Figure-2: Contrast CT showing dilated stomach and duodenum.

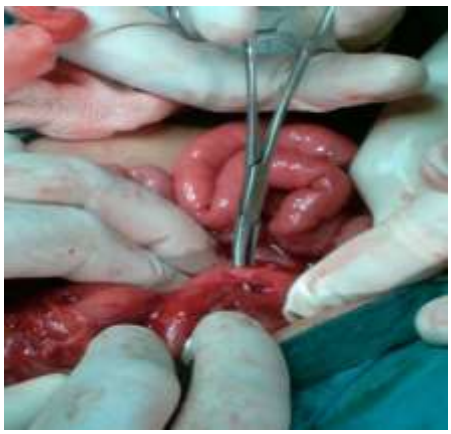

Figure 3: Shows a thick band occluding first part of duodenum which was dissected out and divided.

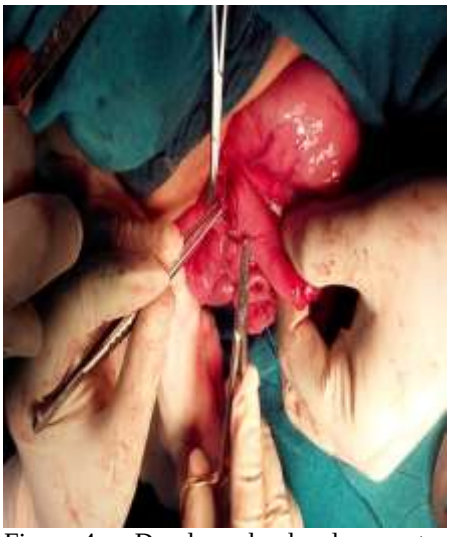

Figure-4: Duodeno-duodenal anastomoses in progress over longitudinal duodenotomy.

Figure-5: Post operatively baby showing excellent recovery.

Figure-6: Abdominal incision in infants and neonates for duodenal obstruction should be sited

Two cms above umbilicus extending to right transversely.

Figure -7: Surender's modification for duodeno-duodenal anastomosis performed over longitudinal incisions proximal and distal to obstruction.

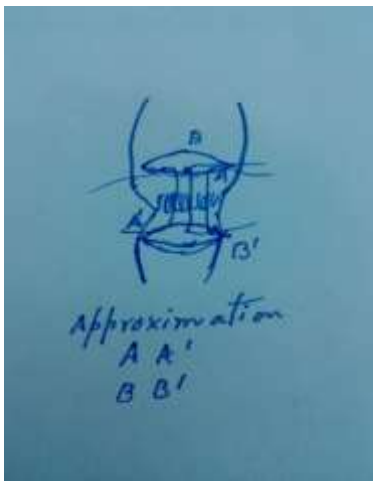

Figure-8: Proximal and distal duodenotomy wounds are sutured transversely.

Figure-9: Anastomoses completed.

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