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"GASTRIC OUTLET OBSTRUCTION IN INFANCY IN CHILDREN HOSPITAL - BENGHAZI''

انسداد نهاية المعدة وبداية الاثني عشر عند الاطفال اقل من سنة في مستشفى الأطفال - بنغازي

In partial fulfillment of the regulations for the Award of Degree of
MASTER OF SURGERY-PEDIATRIC SURGERY

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وَلَقَدْ خَلَقْنَا الإِنسَانَ مِنْ سُلاَلَةٍ مِنْ طِينٍ (12) ثُمَّ جَعَلْنَاهُ نُطْفَةً فِي قَرَارٍ مَكِينٍ (13) ثُمَّ خَلَقْنَا النُّطْفَةَ عَلَقَةً فَخَلَقْنَا الْعَلَقَةَ مُضْغَةً فَخَلَقْنَا الْمُضْغَةَ عِظَاماً فَكَسَوْنَا الْعِظَامَ لَحْماً ثُمَّ أَنشَأْنَاهُ خَلْقاً آخَرَ فَتَبَارَكَ اللَّهُ أَحْسَنُ الْخَالِقِين(14)

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ABBREVIATIONS

ASA American society of anesthetists

AP Annular pancreas

CDO Congenital duodenal obstruction

DA Duodenal atresia

DW Duodenalweb

GOO Gastric outlet obstruction

ICU Intensive care unit

IHPS Infantile hypertrophic pyloric stenosis

LB Ladd's band

MRI Magnetic resonance image

NEC Necrotizing enterocolitis

OPD Out patient department

OGT Orogastric tube

PUD Peptic ulcer disease

PS Pyloric stenosis

PW Pyloricweb

SHO Senior house office

SPSSStatical package for social sciences

TAT Trans anastomotic tube

TPN Total parenteral nutrition

UGI Upper gastrointestinal image

USG Ultrasonography

ABSTRACT

AIMS: The aim of the study was analysing and reviewing causes of gastric outlet obstruction and its various signs and symptoms also to review various modalities of investigations and treatment and their results.

PATIENTS AND METHODS: All consecutive cases who were admitted to children hospital in Benghazi in a period of 5 years (from July 2008 to June 2013) were included in the study . Medical records of admitting history and clinical examination , admission laboratory values , imaging procedures , follow up sheaths , operative and post-operative notes were analysed . Patients were divided into two groups ; neonatal and infant groups

RESULTS: During the study period 70 patients were admitted with adiagnosis of gastric outlet obstruction. The average age were 8.80645± 7.05 days in neonate group and 45.175± 12.90 days in infants. The patients presented with projectilenonbilious vomiting in 47 patients giving ratio of 67.14%. The majority of patients in this study had pyloric stenosis (n=40 , 57.1 %) , 32 male and 8 female, giving a ratio of 4:1, we notice in our study, the annular pancreas constitutes 5.7 % (n=4) giving a ratio of 3:1.Other causes of GOO has no sex related incidence difference . The double bubble sign, which is very sensitive in detecting the abnormality, with sensitivity 93 % and specificity of 96%.The dilated stomach is seen as the prominent sign during the upper GIT contrast ,with high positive predictive value (93%). Ultrasound study was performed for all patients with clinical diagnosis of pyloric stenosis, the pyloric hypertrophy is the hall mark, the sensitivity of this test is high in detecting the underlying disease is 93 % and specificity 89 %. The types of surgical procedures reveal significant difference between the 2 groups in pyloromyotomy, pyloroplasty and duodenoduodenostomy ($P < 0.05$). No difference was found in the incidence of either postoperative complications between groups ($P > 0.05$).

CONCLUSION: In infants , congenital pyloric stenosis is the commonest cause of gastric outlet obstruction. The majority of patients present early with good general condition. Pyloromyotomy is the most common surgical procedure performed with no significant complications. The X ray , ultrasound and the upper GIT contrast study are highly sensitive in detecting the underlying disease if used in proper context.

Key words: Pyloric stenosis, Pyloric web, Duodenal Atresia, Duodenal Web, Annular Pancreas, Ladd's Band, Pyloromyotomy, Pyloroplasty and Duodenoduodenostomy.

INTRODUCTION

Gastric outlet obstruction (GOO, also known as pyloric obstruction) is not a single entity; it is the clinical and pathophysiological consequence of any disease process that produces a mechanical impediment to gastric emptying. Clinical entities that can result in GOO generally are categorized into two well-defined groups of causes: benign and malignant. This classification facilitates discussion of management and treatment.

As part of the initial workup, exclude the possibility of functional non mechanical causes of obstruction, such as gastroparesis. Once a mechanical obstruction is confirmed, differentiate between processes because definitive treatment is based on recognition of the specific underlying cause. Carry out diagnosis and treatment expeditiously, because delay may result in further compromise of the patient's nutritional status.

Delay will also further compromise edematous tissue and complicate surgical intervention. Orient initial management to identification of the primary underlying cause and to the correction of volume and electrolyte abnormalities. Plain X-Ray , barium swallow studies and Ultrasonography are the main tests used to help make the diagnosis. Intrinsic or extrinsic obstruction of the pyloric channel or duodenum is the usual pathophysiology of GOO; the mechanism of obstruction depends upon the underlying etiology.

Patients present with intermittent symptoms that progress until obstruction is complete. Vomiting is the cardinal symptom. Initially, patients may demonstrate better tolerance to liquids than solid food. In a later stage, patients may develop significant weight loss due to poor caloric intake. Malnutrition is a late sign, In the acute or chronic phase of obstruction, continuous vomiting may lead to dehydration and electrolyte derangement of hypokalemic hypochloremic metabolic alkalosis . [1, 2] When obstruction persists, patients may develop significant and progressive gastric dilatation. The stomach eventually loses its contractility. Undigested food accumulates and may represent a constant risk for aspiration pneumonia.

Within the infancy population, pyloric stenosis constitutes the most important cause of GOO. It is more common in males than in females and also is more common in first-

born baby. Pyloric stenosis is the result of gradual hypertrophy of the circular smooth muscle of the pylorus. The incidence of pediatric gastric outlet obstruction is approximately 2–5 per 1,000 . [2] While a detailed history and physical examination might suggest the etiology of gastric outlet obstruction, imaging is virtually always performed to confirm diagnosis before definitive treatment commences.

In infants, gastric outlet obstruction is most often the result of idiopathic hypertrophic pyloric stenosis (IHPS). The exact incidence of gastric outlet obstruction in the older child is not well-established . [3. 7] One Taiwanese report documents that11 of 142 patients admitted with gastric outlet obstruction were from non-IHPS causes, including other congenital anomalies [8]

AIMS OF THE STUDY

It was intended to carry out a retrospective study of gastric outlet obstruction with the following aims:

- TO ESTABLISH OCCURRENCE ENCOUNTERED CAUSES OF GASTRIC OUTLET OBSTRUCTION.
- TO DESCRIBE VARIOUS SIGNS AND SYMPTOMS OF GASTRIC OUTLET OBSTRUCTION.
- TO IDENTIFY THE VARIOUS MODALITIES OF INVESTIGATIONS , TREATMENT AND OUTCOME

MATERIALS AND METHODS

STUDY DESIGN

Clinical-epidemiological retrospective study

STUDY SITTING

This study is conducted in the Department of Pediatric Surgery at Benghazi children hospital in a duration of 5 years (between July 2008 and June 2013) .

Pediatric surgery department one of the medical departments in the children hospital in Benghazi which cover big geographical area extend from Tobrok city in the east up to Serit city in the west and Elkofra city in the south .

The pediatric surgical department provides medical service in emergency and cold surgery for ages from first day of delivery up to 14 years .

The pediatric surgical department covers the medical service science around 35 years 24 hourly .

The pediatric surgical department consists of :

- Surgical units (2 units) with 40 beds
- Neonatal surgery unit with 10 incubators
- Intensive care unit (ICU) with 10 beds
- Operation unit with 4 operation theaters (one of them is septic) and 1 recovery room
- 2 Dressing rooms
- Outpatient clinic and follow up clinic

The patients were seen either in surgical OPD or transferred from medical department in the children hospital or from hospitals inside Benghazi or outside it .

The admitted patients were seen by SHO and registrar doctors who take history and examination and give provisional management then seen by specialist to confirm the diagnosis and gives further management after that seen by consultant who gives final decision .

The round usually done twice daily (morning round and evening round) even in the holidays by the staff , the patients who need surgical intervention discussed with anesthesiologist for any further investigation . Operations done at any time according the urgency of the case .

Post operatively patients stay either in ICU or ward according to the condition and followed regularly until the patient fit for discharge . After discharge the patient follow up surgical OPD regularly

Academic service in pediatric surgical department

- Teaching 4^{th} and 5^{th} medical students
- Training intern doctors
- Training surgery doctors undergo Libyan board and pediatric surgery master
- Supervision thesis in pediatric surgery master

SOURCE OF DATA

Data were extracted from patient's files filled by resident doctors.

Extracted data included the following :

1 - Demographic characteristic : age , gender , residence , nationality , date of admission and discharge

2 – Clinical characteristic : provisional diagnosis , intervention of any , and treatment received

3 – Relevant investigations , different radiological studies , ultrasonography and MRI

4 – Notes describe the surgical interventions

5 – Daily notes regarding clinical status of patient

6 – Length of stay in hospital ,discharge note and death causes if mortality occur

STUDY PERIOD

The study period was 6 months 4 months for data collection and literature review , and 2 months for data analysis and thesis writing

EXCLUSION CRITERIA

- Patients older than 1 year
- Patientswith non mechanical obstruction

METHOD

We identified patients aged from first day of delivery up to one year old with first-admission diagnosis of GOO to our surgical ward between July 2008 and June 2013. Babies with GOO requiring admission were admitted to the pediatric surgery ward in our hospital as our department was responsible for their inpatient care. Case notes were retrieved for the mother or attendants, relevant data extracted and diagnosis of GOO was confirmed.

The patients were divided in two groups . The first group aged from 1 day up to 28 days called neonates . the second group aged from 29 days up to 1 year called infants.

All patients with a clinical diagnosis of gastric outlet obstruction were seen at the Department of Pediatric Surgery at Benghazi children hospital during the study period were consecutively included into the study. Patients with gastroparesis without any mechanical obstruction were excluded from the study. The diagnosis of gastric outlet obstruction was based on clinical presentation and imaging studies,

Preoperatively, all the patients recruited into the study had intravenous fluids to correct fluid and electrolyte deficits, nasogastric suction, urethral catheterization and broad-spectrum antibiotic coverage. They had preoperative anaesthetic assessment using the American Society of Anesthetists (ASA) classification . [3]

Relevant preoperative laboratory investigations included complete blood count, hemoglobin levels, serum albumin, serum electrolytes, urea and creatinine, HIV testing , imaging investigations included plain abdominal X-rays, barium studies and abdominal ultrasound.

Intra-operatively, all patients, under general anesthesia were subjected to exploratory laparotomy through transverse incision. At operation, the diagnosis of gastric outlet obstruction was made by noting a cicatrized first part of duodenum or pylorus with a dilated and thick-walled stomach. The type of surgical procedure was done according to

whether the cause of gastric outlet obstruction was in stomach or in other parts of duodenum.

The operations were performed either by a consultant surgeon or a senior resident under the direct supervision of a consultant surgeon.

Postoperatively patients were kept nil orally till return of bowl sounds and at that time nasogastric tubes were removed. Intravenous antibiotics were used for this period.

The postoperative outcome was monitored; patients in ASA classes IV and V were admitted into intensive care unit after surgery. The study variables included socio-demographic (i.e. age , gender, and area of residence), clinical presentation, HIV status, laboratory, radiological and endoscopic findings, ASA classification, operative findings and surgical procedure performed. The variables studied in the postoperative period were postoperative complications, hospital stay and mortality. Patients were followed up for a period of twelve months.

STATISTICAL ANALYSIS

The statistical analysis was performed using statistical package for social sciences (SPSS) version 17.0 for Windows (SPSS, Chicago IL, U.S.A). The median and ranges were calculated for continuous variables where as proportions and frequency tables were used to summarize categorical variables. Continuous variables were categorized.

Fisher test were used to test for the significance of association between the independent (predictor) and dependent (outcome) variables in the categorical variables. The level of significance was considered as $P < 0.05$.

Multivariate logistic regression analysis was used to determine predictor variables that predict the outcome.

ETHICAL CONSIDERATION

Ethical approval to conduct the study was obtained from the Department of Pediatric Surgery at Benghazi children hospital ,joint institutional ethic review committee before the commencement of the study.

LITERATURE REVIEW

Gastric outlet obstruction (GOO, also known as pyloric obstruction) is not a single entity; it is the clinical and pathophysiological consequence of any disease process that produces a mechanical impediment to gastric emptying. [2]

The gastric outlet obstruction in infancy is a relatively rare condition with a incidence of 1 in 100,000 live births when the idiopathic hypertrophic pyloric stenosis**(**IHPS) is excluded . [2,3]

The incidence of IHPS is 1.5 to3 per 1,000 live births.[2]However the conditions like; gastricweb, pyloric atresia, ectopic pancreatic tissue and duplication of pylorus can also produce the signs and symptoms of GOO. The acquired cause of GOO in infants are acid peptic disease, neoplasm and caustic ingestion'[4,5]

1)Idiopathic hypertrophic pyloric stenosis

Hypertrophic pyloric stenosis (HPS) causes a functional gastric outlet obstruction as a result of hypertrophy and hyperplasia of the muscular layers of the pylorus. In infants, HPS is the most common cause of gastric outlet obstruction and the most common surgical cause of vomiting. [9]

Bottle-feeding was associated with an increased risk for HPS in a population-based case-control study of 714 infants. After adjustment for sex, race, maternal smoking status, and other factors, bottle-feeding was associated with an increased risk for HPS (odds ratio [OR], 2.31; 95% confidence interval, 1.81 - 2.95) compared with breast feeding. This effect was most pronounced in the children of older and multiparous mothers.[6 , 7]

Hirschsprung wrote the first complete description of hypertrophic pyloric stenosis (HPS) in 1888. He believed the disease was congenital and represented fetal pyloric development failure. In 1907, Ramstedt described an operation to alleviate this condition. He suggested splitting the pyloric muscle and leaving it open to heal secondarily. This procedure has been used to treat infantile hypertrophic pyloric

stenosis (IHPS) since that time. Although this curious disease is treated easily with surgery, its etiology remains undetermined. Hypertrophic pyloric stenosis is inherited by a multifactorial threshold model, and the generalized occurrence risk for siblings is 5-9%. Associated congenital anomalies are reported in 6-20% of patients with pyloric stenosis. A rare association with developmental delay has also been reported.[8]

HPS occurs secondary to hypertrophy and hyperplasia of the muscular layers of the pylorus, which cause a functional gastric outlet obstruction.HPS occurs secondary to hypertrophy and hyperplasia of the muscular layers of the pylorus, which cause a functional gastric outlet obstruction. [8 , 9]

Recently, genetic studies have identified susceptibility loci for infantile HPS and molecular studies have concluded that smooth muscle cells are not properly innervated in infantile HPS.[9 , 13]

The risk for IHPS was inversely and significantly associated with total cholesterol level, with an odds ratio of 0.77 per 10 mg/dL.[10 , 11]

Typical presentation of an infant with hypertrophic pyloric stenosis (HPS) is onset of initially nonbloody, always nonbilious vomiting at 4-8 weeks. Although vomiting may initially be infrequent, over several days it becomes more predictable, occurring at nearly every feeding. [13]

Careful physical examination provides a definitive diagnosis for most infants with hypertrophic pyloric stenosis. However, some of the classic signs that would lead to diagnosis may be absent due, in part, to the early diagnosis of hypertrophic pyloric stenosis. An enlarged pylorus, classically described as an "olive," can be palpated in the right upper quadrant or epigastrium of the abdomen in 60-80% of infants.[12] .

Ultrasonographyhas become the criterion standard imaging technique for diagnosing hypertrophic pyloric stenosis. It is reliable, highly sensitive, highly specific, and easily performed. An experienced ultrasonographer increases the test's predictive value. Necessary measurements include pyloric muscle thickness and pyloric channel length.

Muscle wall thickness 3 mm or greater and pyloric channel length 14 mm or greater are considered abnormal in infants younger than 30 days. [8 , 9]

Barium upper GI (UGI) study is an effective means of diagnosing HPS when ultrasonography is not diagnostic. It should demonstrate an elongated pylorus with antral indentation from the hypertrophied muscle. The UGI may demonstrate the "double track" sign when thin tracks of barium are compressed between thickened pyloric mucosa or the "shoulder" sign when barium collects in the dilated prepyloricantrum. After UGI barium study, irrigating and removing any residual barium from the stomach is advisable to avoid aspiration. [8 , 9 , 22]

Surgical repair of hypertrophic pyloric stenosis (HPS) is fairly straightforward and without many complications, yet properly preparing the infant for this procedure is vitally important. Most infants with hypertrophic pyloric stenosis do not have complete gastric outlet obstruction and can tolerate their inherent gastric secretions.

RamstedtPyloromyotomy remains the standard of treatment, and outcome is excellent. The best surgical outcome and lowest complications are more likely when the surgeon has specialist pediatric surgical training.[14]

Some authorities report that laparoscopic pyloromyotomyhas a significantly shorter recovery time compared with open pyloromyotomy but that open pyloromyotomy has higher efficacy and fewer complications. However, a small (N=98) 2011 prospective, randomized trial found no difference in operating time, length of stay, or difference in time to full feeds between open and laparoscopic pyloromyotomy. While complication rates were similar between the 2 groups, significantly superior long-term cosmetic results were noted in the laparoscopic group. [15] .

A systematic review of 502 patients echoed these results, finding laparoscopic pyloromyotomy does not lead to significant postoperative complications compared to open pyloromyotomy.[16]

Feedings are usually resumed 6-8 hours after operation. In most instances, gradually increasing the volume and strength of feedings is recommended.[17]

Undetected mucosal perforation: Perform a diligent search for mucosal transgressions at the time of operation and examine the infant again before initiating feedings. In those rare cases where a perforation was not detected, the infant develops fever, tenderness in the abdomen, and abdominal distention. Return to the operating room if perforation is suspected. [14 , 15 , 16]

Bleeding: In most instances, venous oozing from the myotomy site is self-limited and is not a concern in the postoperative period. Reports of continued bleeding are exceedingly rare but can occur, especially in children with undetected coagulopathy.[14 , 15 , 16]

Persistent vomiting: Incomplete pyloromyotomy is rare in the hands of an experienced pediatric surgeon and usually presents as persistent vomiting until after the second week post-surgery. This problem is confounded when repeat studies performed after surgery provide a confusing picture. Patient observation resolves the problem in most cases. [14 , 15 , 16]

2) Congenital duodenal Atresia

Relatively speaking, congenital duodenal atresia is one of the more common intestinal anomalies treated by pediatric surgeons. In 25-40% of cases, the anomaly is encountered in an infant with trisomy 21 (Down syndrome). [18]

Calder published the first report of duodenal obstruction in 1733 when he described 2 children with "preternatural confirmation of the guts." Both infants died, as did subsequently reported infants with this defect. Scattered reports of duodenal obstruction appeared in the European literature over ensuing years. In 1916, the first survivor was reported, yet survival in the early 20th century remained rare. Morbidity and mortality significantly improved only over the last 50 years. [19]

Because of progress in pediatric anesthesia, neonatology, and surgical techniques, survival is about 90% in infants who present with this anomaly. [1]

The standard operative procedure today consists of duodenoduodenostomy via a right upper quadrant incision, although recent advancements have enabled some surgeons to repair the defect by minimally invasive means. [20 , 21].

Duodenal atresia can take many forms, but proximal and distal intestinal segments always end blindly. [23].

The intestine on either side of the defect may be in apposition (type 1), separated by a fibrous cord (type 2), or gap (type 3). Regardless of atresia severity, the proximal intestinal segment is typically dilated and the distal segment empty; these are hallmarks of duodenal atresia. Although obstruction may occur anywhere within the duodenum, it is most common in the vicinity of the ampulla of Vater. [21]

Stenosis may manifest as a stricture or a perforated intraluminal diaphragm. The perforation within the diaphragm is usually singular and centrally located within the lumen of the duodenum, although variations have been reported. A windsock abnormality is a thin diaphragm that has ballooned distally as a result of peristalsis. Together, both duodenal atresia and stenosis comprise a frequent cause of intestinal obstruction in the newborn. [19]

No predisposing maternal risk factors are known. Although up to one third of patients with duodenal atresia have Down syndrome (trisomy 21), it is not an independent risk factor for developing duodenal atresia. [18]

The use of modern ultrasonography has allowed many infants with duodenal obstruction to be identified prenatally. [24 , 25]

In a large cohort study of 18 different congenital malformation registries from 11 European countries, 52% of infants with duodenal obstruction were identified in utero. [30]

Duodenal obstruction is characterized by a double-bubble sign on prenatal ultrasonography. The first bubble corresponds to the stomach and the second to the postpyloric and prestenotic dilated duodenal loop. Prenatal diagnosis allows the mother the opportunity to receive prenatal counseling and to consider delivery at or near a tertiary care facility that is able to care for infants with GI anomalies. [31,32]

When duodenal atresia is suspected, erect and recumbent plain radiography of the abdomen should be the first imaging study obtained. A characteristic finding of duodenal obstruction is the double-bubble image of an air-filled stomach proximal to an air-filled first portion of the duodenum. Absence of gas in the remaining small and large bowel suggests atresia, whereas scattered amounts of gas distal to the obstruction suggests stenosis or malrotation/volvulus.Upper GI contrast evaluation in the infant with duodenal atresias is unnecessary unless correction is going to be delayed. [33]

Duodenal atresia and stenosis are treated surgically. In patients with duodenal obstruction, a duodenoduodenostomy (either a side-to-side or a diamond-shaped fashion) is the most commonly performed procedure. A duodenojejunostomy is now uncommonly performed due to its higher risk of long-term complications. Duodenal repair may be performed via a right upper quadrant incision, an umbilical incision, or laparoscopically, depending on surgeon preference. [26]

In patients with a duodenal web, the surgeon can identify the site of the web's origin by passing the OG tube through the pylorus into the duodenum and noting the indentation of the duodenal wall caused by tenting of the web. A duodenotomy can be performed along the site of this indentation. [28 , 29]

In patients with an annular pancreas, pancreatic tissue should not be divided for fear of pancreatic fistula. Instead, a diamond-type or side-to-side duodenoduodenostomy is recommended. Patients who present with associated malrotation should undergo a Ladd procedure at the time of duodenal repair. [20]

One retrospective case series compared the right upper quadrant incision to a laparoscopic repair for duodenal atresia or stenosis. [30]

Fourteen patients were in the open group, and 15 patients were in the laparoscopic cohort. No anastomotic leaks were reported in either group. Patients that underwent a laparoscopic repair were advanced to full feeding quicker (9 d vs 17 d) and were discharged from the hospital sooner (13 d vs 20 d) compared with patients who underwent open repair. [31]

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 by some surgeons 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). [27]

If possible placement a small transanastomotic feeding tube (5F silasticnasojejunal feeding tube) across the anastomosis to facilitate postoperative enteral feeding and always leave an OG tube in place for gastric decompression. One should consider placing a peripheral intravenous central catheter (PICC) or central intravenous catheter at the time of operation because of the expected prolonged ileus and the need for parenteral nutrition. [32 , 33]

Despite improvements in early mortality rates, as many as 22% of children may incur late complications. Late complications include blind-loop syndrome, megaduodenum with altered duodenal motility, gastritis with duodenal-gastric reflux, peptic ulcer, esophagitis and gastroesophageal reflux, pancreatitis, and cholecystitis. Blind-loop syndrome can be corrected by conversion to a duodenoduodenostomy. Megaduodenum with abnormal duodenal motility can be addressed by performing a tapering duodenoplasty. Today, these issues may be addressed at the time of initial operation by performing the duodenoduodenostomy along with duodenoplasty when necessary. [32 , 33 , 34]

The overall mortality rate for infants with duodenal atresia was 33% in a large series published in 1967. Today, the early mortality rate associated with this condition has

declined to approximately 3% in most series. Most deaths occurring in association with duodenal atresia are attributed to the presence of multiple associated anomalies (usually complex cardiac defects). Improvement in survival rates is most likely a result of advances in neonatal care such as high-frequency ventilation, surfactant supplementation, nutritional support, pediatric anesthesia, and sophisticated cardiac surgery. Long-term survival is excellent at rates reported between 86% and 90%.[32 , 33 , 34]

3) Gastric volvulus

Gastric volvulusoccurs when there is rotation of part or all of the stomach by at least 180° from its usual position. Acute gastric volvulus can be life-threatening, necessitating rapid diagnosis and treatment. In organo-axial volvulus, the mostcommon type, occurring in 50–60% of cases, the stomach twists about a longitudinal axis between the pylorus and the gastroesophageal junction. [35]

This might be a transient phenomenon when seen in newborns and in children with neuromuscular disorders. In mesentero-axial volvulus, the stomach twists about an axis perpendicular to its long axis, resulting in the pylorus being located anterior and superior to the gastroesophageal junction . [35 , 36]

Children can present with acute gastric volvulus at any age, although the majority of patients are younger than 1 year . [37 ,38]

Symptoms of pain, vomiting and pneumonia are common, but unlike other causes of gastric outlet obstruction, hypochloremic alkalosis is not present because of gastric inlet and gastric outlet obstruction. Borchardt’s triad of nonproductive vomiting, pain and inability to pass an enteric tube is seen in 70% of patients . [35 , 38]

Gastric volvulus is often associated with other congenital abnormalities, including diaphragmatic or hiatal hernias, diaphragmatic eventration and intestinal malrotation [37 , 38 , 39 ,40]

Definitive treatment is surgical derotation with repair of associated defects is needed, and possible gastropexy. [36 , 39]

Nonsurgical management has been reported with some success. [37 ,40 , 41]
Most cases are diagnosed by UGI, though plain radiography and CT scan also be used . [40 , 41]

On both UGI and radiography, an abnormal configuration of the stomach isseen . Organo-axial volvulus results in a horizontally oriented stomach with the greater curvature located superiorly with respect to the lesser curvature. In mesentero-axial volvulus, the pylorus is closely approximated to and located just anterior and superior to the gastroesophageal junction. The diaphragm might be elevated because of marked gastric distention or congenitally absent associated with an intrathoracic stomach . [41]

4) Gastricantral web

Gastric antral web, also known as prepyloric web or mucosal diaphragm, is a rare cause of outlet obstruction. Histologically the web is composed of normal, non-inflamed mucosal and submucosal gastric mural layers. [42]

This condition presents with varying degrees of nonbilious vomiting and abdominal pain . [43 , 44]

Symptoms usually correlate with apertures of 1 cm or less. [45]

Most children present in the first year of life, although symptom onset in later childhood and even adulthood has been reported . [46 , 47]
Acquired webs can occur, such as are occasionally seen in epidermolysisbullosa. UGI studies are reportedly 90 % sensitive in cases of antral webs . [45]
These mucosal webs are usually seen as a thin lucent band across a contrast-filled antrum, up to 7 cm from the pylorus . [45]

A “double bulb” sign has been described when a thicker web mimics the pylorus, creating an antral chamber . [42 , 46]

US might also show the web and the related gastric outlet anatomy, if facilitated by adjacent fluid and gas . Gastric duplication cysts are the least common of the alimentary duplications, comprising only 4–7 %. They usually present before 1 year of age with symptoms of obstruction, pain, bleeding or ulceration, and are twice as common in girls . [47]

Histologically, they contain smooth muscle within their walls and are lined by alimentary mucosa. Contrary to other alimentary duplications that occur along the mesenteric aspect of the bowel, gastric duplications aremore common along the greater curvature . [48]

Pancreatic tissue is common within gastric duplications; ectopic gastric mucosa can also be found.Theexact etiology of these congenital lesions is unknown, and a variety of pathogeneses has been proposed .[47]

Abdominal radiographs often show typical findings of gastric outlet obstruction but can also be helpful if vertebral body anomalies are identified—present in 5–21% of patients with duplication cysts . [47 , 49]

US is often diagnostic and, as with other duplications, these cysts will show the “muscular rim” sign of a hypoechoic muscular outer layer and hyperechoic mucosal inner layer. CT and MRI can be employed in complicated cases. [48]

5)Intestinal Malrotation and Ladd's band

Intestinal malrotationis a defect that occurs at the 10th week of gestation. During this stage of development, the intestines normally migrate back into the abdominal cavity following a brief period where they are temporarily located at the base of the umbilical cord. As the intestine returns to the abdomen, it makes two rotations and becomes fixed into its normal position, with the small bowel centrally located in the abdomen and the colon (large intestine) draping around the top and sides of the small intestine. When rotation is incomplete and intestinal fixation does not occur, this creates a defect known as intestinal malrotation. [64 , 65]

6)Annular pancreas

Annular pancreas,as first described by Tiedemann in 1818, is a rare congenital abnormality that accounts for 1% of all intestinal obstructions in the pediatric population.[50]

Annular pancreas more commonly affects males. In up to 50% of the cases, it is associated with other congenital anomalies, including Down syndrome (30%),tracheoesophageal fistula, esophageal atresia, imperforate anus, and Hirschsprung's disease.[51 , 52]

Both of the 2 main theories as to how annular pancreas develops involve the left and right ventral buds that normally form the head and neck of the pancreas. Malrotation of the ventral bud that results in pancreatic tissue surrounding the duodenum and failure of the left ventral bud to atrophy have both been proposed.[53]

Annuli most commonly affect the descending duodenum and may be partially or completely circumferential. Complete duodenal obstruction typically occurs below the level of the ampulla of Vater and presents as bilious vomiting that worsens with subsequent feeding. A partially circumferential presentation can occur later in life or can be asymptomatic.[51]

The most common abdominal radiographic finding is the double-bubble sign, which is composed of gaseous dilatation of the stomach and proximal duodenum, with a paucity of bowel gas distally. The double-bubble sign may also be observed on prenatal ultrasound as 2 adjacent fluid-filled structures in the fetal upper abdomen and may be accompanied by polyhydramnios. Differential diagnosis of this sign, detected either prenatally or postnatally, includes duodenal atresia, annular pancreas, duodenal stenosis, duodenal webs, and midgut volvulus . [54]

For neonates with the classic appearance of a double bubble, further radiologic investigation is unnecessary, since all congenital causes of duodenal obstruction require surgery. Surgical repair of annular pancreas with duodenojejunostomy or

duodenoduodenostomy can relieve the obstruction and is commonly successful without complication.[54]

The double-bubble sign is commonly associated with duodenal atresia, but one must keep in mind that it actually represents duodenal obstruction and that other causes of obstruction should be considered. Annular pancreas is an uncommon cause of intestinal obstruction, and patients with this condition usually present in the first year of life. Radiographically, the double-bubble phenomenonis seen in most cases. As with duodenal atresia, annular pancreas is often associated with other congenital anomalies that necessitate thorough examination of these patients. Surgical correction of annular pancreas has a good prognosis. [52 , 54]

7) Gastric polyps

Gastric polypscan present in isolation or as a manifestation of a polyposis syndrome. Pedunculated or antral polyps can present with gastric outlet obstruction. They often bleed, leading to hematemesis, melena or anemia. A prolapsing gastric polyp can also lead to hypergastrinemia with abnormally increased gastric acid production caused by isolation of gastric mucosa from its normal acidic environment . [55]

Gastric polyps can be hyperplastic or adenomatous, with a 1.5–3% chance of malignant transformation in hyperplastic polyps, and a higher risk in adenomatous polyps . [56]

Depending on the etiology, hyperplastic gastric polyps sometimes regress with medical management alone. Polyps are more common in adults, where they are often found and treated by endoscopy . [57 , 58]

In infants, symptoms can mimic IHPS. The diagnosis can be made by US or UGI, which demonstrate an intraluminal mass that might be polypoid or sessile. [58]

8) Neoplasm

Gastric and epigastric neoplasms are rare in children but should always be considered as an etiology of gastric outlet obstruction in the older child. Lymphoma, bothprimary gastric and mesenteric, can cause symptoms ofoutlet obstruction. Mass effect from large or inopportunelypositioned epigastric tumors including hepatoblastoma,hepatocellular

carcinoma, pancreaticoblastomas, and soft-tissue sarcomas of the mesentery or abdominal viscera can also result in gastric outlet obstruction. [59]

Primary gastric mesenchymal tumors such as gastrointestinalstromal tumor (GIST) [60 , 61] and gastric teratomas[62 , 63] can also lead to obstruction.

Study 1 : Gastric outlet obstruction in children was done in *Taiwan*

Gastric Outlet Obstruction in Pediatric Patients Ju-Bei Yen, MD; Man-Shan Kong1 , MD Department of Pediatrics, Chang Gung Memorial Hospital, Chiayi; 1 Department of Pediatrics, Chang Gung Children's Hospital, Taipei. Taiwan

This study reports the etiologies, management and outcome of children with gastric outlet obstruction (GOO) in a children's hospital. The medical records of 11 children with GOO not associated with idiopathic hypertrophic pyloric stenosis (IHPS) were reviewed. They were categorized into one group of anatomic abnormality (AA group) and one group of peptic ulcer disease (PD group). One case underwent episodes of GOO caused by anatomic abnormality and peptic ulcer disease, respectively. Six cases belonged to the AA group. Mean age was 58 months with a male to female ratio of two to four. Underlying etiologies were prepyloric mass (2), web (2) and gastric volvulus (2). Four patients underwent surgery. One patient was lost to follow-up. GOO did not recur in the follow-up period (mean duration 24 months) in the remaining cases. One case in the AA group and the remaining five patients composed the PD Group. Mean age was 49 months and all were male. Underlying causes were gastric ulcers (4) and chronic duodenal ulcers (2). Two of the five patients had Helicobacter pylori infection found by rapid urease test. Four patients recovered after medical management and another two, with normal serum gastrin levels, underwent surgery because of poor response to medical treatment. One case was lost to follow-up. No recurrence of GOO was noted in the follow-up period (mean duration 27 months) in the remaining cases. Peptic ulcer disease was as important as anatomic abnormalities as the etiology for GOO not associated with IHPS, and medical management could release GOO caused by it. Compared to adult patients, H. pylori infection played a less important etiologic role

in pediatric patients with GOO. Anatomic lesions in the prepyloric or antral area of the stomach could cause GOO. Symptoms of gastric atresia, stenosis or web often develop in infancy. However, in this series, one case with web was not diagnosed until the patient was school age, and her assumed condition was not congenital in nature. In fact, some studies report that gastric webs can be acquired. In addition, epidermolysisbullosa or esophageal atresia may be accompanied by gastric atresia or webs but such a condition was not noted in two patients. Prepyloric tumors were found in two cases (hyperplastic polyps and ectopic pancreas). Gastric tumors are uncommon in children and hyperplastic polyps are reported to be the most commonly identified gastric polyps in children. Another study showed that an ectopic pancreas, not common in children, may occur in 1~2% of autopsies and the most likely symptom is epigastric pain. Furthermore, gastrointestinal bleeding, obstruction or malignant transformation could develop in long-term follow-up, and surgical removal was suggested under such circumstances. Gastric volvulus is abnormal rotation of one part of the stomach around another. Some congenital defects, such as diaphragmatic defects, a splenic syndrome or wandering spleen may be noted with gastric volvulus. In two cases with gastric volvulus, one had diaphragmatic hernia and the other suffered from two episodes of acute gastric volvulus with spontaneous resolution. GOO is a common complication of peptic ulcer diseases in adults but it rarely occurs in childhood. In peptic ulcer diseases, GOO is usually caused by a combination of edema, spasm, fibrotic stenosis and gastric atony. Only one had GOO and no special risk factor was identified. The age range of those patients, who underwent surgical treatment, was from five to 43 months. In six patients with GOO induced by peptic ulcer diseases, four were less than three years old and all were male. It appears that younger male children with peptic ulcer diseases have more GOO than others. GOO caused by peptic ulcer diseases can be resolved by medical treatment, vagotomy, pyloroplasty .However, the duration of medical treatment before surgical intervention was not determined. Before using omeprazole with/without antibiotics, five out of seven patients reported by Huang et al. underwent surgery after treatment with cimetidine lasting 12 to 46 days. Weiland et al. suggested that failure to respond to medical treatment within five days was an indication for surgical treatment. In patients, four out of six patients gained spontaneous resolution of GOO after medical

management (including antacids, cimetidine or omeprazole) lasting three to 31 days. The data suggest that a longer duration of medical management may be needed. Apart from this, reversal of GOO after eradication of H. pylori infection has been reported. The two cases with H. pylori infection that we observed in this series were not treated with antibiotic therapy: one had clinical improvement with medical management and the other underwent surgical intervention eventually. According to data, H. pylori infection in children with GOO plays a less important role than it does in adults. According to study, except for IHPS, peptic ulcer diseases were as important as anatomic abnormalities in the etiologies of GOO in pediatric patients. Female predominance in the AA group and male predominance in the PD group was observed. The time interval between onset of symptoms and diagnoses was shorter in the AA group. Surgical intervention was often needed for treatment of GOO in the AA group.

Study 2 : Infantile hypertrophic pyloric stenosis was done in *Tanzania*

Infantile hypertrophic pyloric stenosis at a tertiary care hospital in Tanzania: a surgical experience with 102 patients over a 5-year period

Phillipo L. Chalya, Mange Manyama, Neema M. Kayange, Joseph B. Mabula, and Alicia Massenga

In this study the males were more affected than females with a male to female ratio of 4.7:1 and showed higher incidence of IHPS in the first born infants and male sex . In about 6–33 % of infants with IHPS, associated anomalies have been described in the central nervous system (CNS), gastrointestinal tract (GIT), and urinary tract and associated anomalies were reported in 6.9 % of cases.

The mean duration of illness was 4 weeks . This could be due to lack of awareness of families to consider the vomiting as minor symptom and ignore it, or misdiagnosis of the disease. This delayed presentation is a common phenomenon in developing

countries and can lead to delay in the diagnosis. Delay in diagnosis can result in significant electrolyte imbalance, weight loss, and failure to thrive . The definitive diagnosis of IHPS is usually confirmed by abdominal ultrasound, on which IHPS is characterized by increased pyloric muscle thickness length and diameter. However, in experienced hand, a careful clinical examination provides a definitive diagnosis for most infants with HPS and abdominal ultrasound is reserved for atypical cases . The clinical diagnosis is easily made if the presenting clinical features are typical, with projectile vomiting, visible peristalsis, and a palpable pyloric tumor. In the present study, the diagnosis of IHPS was made clinically in more than 80 % of patients and abdominal ultrasound was employed in only 15.7 % of patients as it was not always readily available in their center.

In patients with IHPS, serum electrolytes should be measured immediately when the patient arrives in hospital. If vomiting has been ongoing for several days, serum electrolytes are frequently deranged. The nature of derangement is a spectrum, ranging from mild to severe hyponatraemia, hypochloraemia, hypokalaemia, and metabolic alkalosis . In the current study, serum electrolytes results revealed hypokalaemia (66.7 %), hyponatraemia (40.7 %) and hypochloraemia (33.3 %) which is an expected occurrence in untreated cases . This electrolyte derangement in this series can be explained by the fact that the majority of patients presented late to the hospital when electrolyte imbalance had set in. Prolonged delay in diagnosis can lead to dehydration, poor weight gain, malnutrition, metabolic alterations, and lethargy. When these derangements occur, they should be corrected before surgical treatment. There was also prolonged preoperative hospital stay (8 days), which could be due to time needed to correct the fluid and electrolyte abnormalities, late admissions, unavailability of beds and busy surgical service.

Surgical intervention was the main stay of treatment performed in all of the patients. Open Ramstedt’s pyloromyotomy remains the standard procedure of choice for hypertrophic pyloric stenosis because it is easily performed and is associated with minimal complications. In this study, only open Ramstedt’s pyloromyotomy was done in all patients as there was no facility for laparoscopic pyloromyotomy at their center.

Mucosal perforation is a rare intraoperative complication of Ramstedt's pyloromyotomy and usually results from extending the myotomy beyond the pyloric–duodenal junction and is indicated by the appearance of bilious fluid. When this occurs, repair is done by using interrupted fine monofilament long-term absorbable sutures placed transversely and covered with omentum . In this series, intraoperative mucosal perforations were reported in 5.9 % of cases. This observation calls for meticulous care to be taken when performing Ramstedt's pyloromyotomy to prevent mucosal perforation, especially at the lower end of the incision (pyloric–duodenal junction).

In the current study, the overall complication rate was 11.8 %.Surprisingly, the rate of surgical site infections in this study was significantly high.This finding calls for a need to identify factors responsible for this sad experience. In this study, all postoperative complications were treated conservatively except wound dehiscence in four patients which required surgical correction.

The median overall length of hospital stay in this study was 12 days. This could be due to prolonged preoperative hospital stay (8 days). The post-operative hospital stay was 5 days. However, due to the poor socio-economic conditions in Tanzania, the duration of inpatient stay for patients may be longer than expected and this might have contributed to prolonged length of hospital stay in some of patients.

The overall mortality rate in this study was reported to be 4.9 % . The high mortality rate in this study was attributed to age < 2 weeks, delayed presentation, severe dehydration on admission, hypokalaemia on admission and surgical site infection. Addressing these factors responsible for high mortality in patients is mandatory to be able to reduce mortality associated with this disease. The fact that all deaths in this study occurred on the same postoperative day after a smooth operation raises a big question on postoperative care both in terms of human power and facilities to care for postoperative infants who needs strict fluid balance, ambient environment to prevent hypothermia and other supportive cares. Such infants are transferred to the general pediatric surgical ward on the immediate postoperative period.

The potential limitation of this study is the fact that information about some patients was incomplete in view of the retrospective nature of the study. This might have introduced some bias in findings. Poor documentation of data leading to exclusion of many patients was also a major limitation in this study. However, despite these limitations; findings from this study provide local data that can be utilized to improve the care of patients with IHPS in local setting.

Study 3 : Duodenal atresia was done in *Taiwan*

Distinct Clinical Characteristics of Patients With Congenital Duodenal Obstruction in a Medical Center in Taiwan

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A total of 30 patients withCongenital Duodenal Obstruction CDO, 16 males and 14 females, were included in this study. All patients were diagnosed and treated within 7 days of birth except for one patient who obtained an accurate diagnosis as late as 7 months of age. The mean gestational age was 37.6 weeks, ranging from 31 to 40 weeks. The mean body weight at birth was 2.5 kg (1.3−3.6 kg). Maternal polyhydramnios was present in 16 of the 30 cases (53%). In 15 patients the diagnosis of duodenal obstruction was made prenatally, based on a double-bubble image noted on ultrasound. The remaining 15 patients were evaluated for CDO because of symptoms including vomiting (n= 13), upper abdominal distention (n= 3), respiratory distress (n= 4), upper gastrointestinal bleeding (n= 2), and abdominal mass (n= 1). Operative findings disclosed 16 cases of atresia, 10 cases of mucosal web, and 4 cases of stenosis. Annular pancreas was found in seven patients (3 with atresia, 2 with stenosis, and 2 with mucosal web). An anterior portal vein and a second duodenal web were found in one patient. According to the obstruction site in relation to the ampulla of Vater, 11 of the 30 cases (37%) were classified in the preampullary group and 19 (63%) were in the postampullary group. Seventeen of the 30 patients (56.7%) had at least one additional

anomaly. Six patients had chromosomal anomalies, including four (13%) with trisomy 21, one (3.3%) with a 2P12 deletion, and one with chromosome 46XY metaphase. In the preampullary group, 3 of the 11 patients (27.3%) had associated anomalies. Two patients had chromosomal anomalies, and the other patient had an anterior portal vein combined with malrotation and complex heart disease. In the postampullary group, 14 of the 19 patients (74%) had at least one associated anomaly, including chromosomal anomalies (n= 4), congenital heart disease (n= 8), esophageal atresia with tracheoesophageal fistula (n= 2), malrotation (n= 4), jejunoileal atresia (n= 1), imperforate anus (n= 3), cleft lip/palate (n= 2), hemivertebrae (n= 1), central nervous system anomalies and a double ureter (n= 1). There were significantly fewer associated congenital anomalies in the preampullary group than in the postampullary group (27% vs. 74%, p= 0.012). Two patients died, because of complex heart disease in one patient and hepatic failure in the other patient. The mortality rate was 6.7%.. Inthis series there were only 4 out of the 30 CDO patients (13%) who had concurrent Down syndrome. The location of the obstruction site for patients with CDO has been found to be predominantly distal to the ampulla of Vater. Only 15% of patients with CDO had their lesion sites proximal to the ampulla. They found, for the first time, that CDO patients with preampullary lesions had significantly fewer associated anomalies than patients with postampullary obstructions. However, the abortion rate is usually high in Chinese once anomalies are detected prenatally. The fetus may be aborted when they have anomalies. This trend could possibly have affected the occurrence of CDO-associated anomalies in the population. Early in the fourth week of embryonic growth, the duodenum begins to develop from the caudal part of the foregut and the cranial part of the midgut. The junction of the two parts of the duodenum is just distal to the origin of the bile duct. The etiology of duodenal obstruction is probably related to a failure of recanalization of the duodenal lumen. During the process of vacuolation and recanalization of the duodenum, the hepato-pancreatic duct develops two lumens and opens into the duodenum with two orifices. These two major canals create a narrow segment of the duodenum, and this narrow zone is most prone to faulty recanalization and atresia formation. Therefore, the vast majority of cases with congenital duodenal atresia occur distal to the ampulla of Vater. Patients with Down syndrome have defects

in cell migration that lead to various canalization-related diseases, such as CDO, anal atresia, and congenital heart diseases.

Study 4 : Gastric volvulus in children was done in *Switzerland*

Gastric volvulus in children.

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The aim of the study was to review the records of all children who presented with gastric volvulus in the past 10 years.

The study group consisted of 21 children with an age range from 0.2 months to 4.3 years who were operated for gastric volvulus from 1992 to 2003.

Initial symptoms included acute abdominal pain after meals, vomiting, and in 8 cases, acute apnea associated with pallor, cyanosis, and hypotonia. After the first episode, barium studies revealed an organoaxial gastric volvulus in all cases. The surgical procedure was an anterior gastropexy with reinforcement of the esophagogastric angle performed by laparoscopy in 13 cases and by laparotomy in 8 (1 converted laparoscopy). An associated antireflux fundoplication was done in 3 patients. All children received postoperative antireflux medication for at least 1 month. The follow-up ranged from 4 months to 4.8 years. Two children in the laparotomy group required reoperation (Toupet fundoplication) for persistent gastroesophageal reflux disease. All children are currently symptom-free and without treatment.

Gastric volvulus is a clinical and radiological reality, which can be treated by a gastropexy. Initial fundoplication is not mandatory. The laparoscopic gastropexy is a good option and allows a repeat laparoscopic procedure if needed.

Study 5 : Congenital antral web was done in ***Korea***

So-Hyun Nam, Soo Hyun Koo,[*] Mi Lim Chung,[*] Yu Jin Jung,[*] and Yun-Jung Lim
Korea

Antral web is a rare cause of congenital gastric outlet obstruction. It is also called mucosal diaphragm of the pyloric antrum or prepyloric diaphragm. Its thickness is usually 2-4 mm and it can be found anywhere from 1 to 7 cm proximal to the pylorus. The etiology of webs remains unknown. The developing mechanism is known to be incomplete recanalization of foregut around gestational age 5-6 weeks. In this period, the epithelial cells rapidly overgrow in the lumen, and vacuoles appear and eventually coalesce to recanalize the gut. The webs may be resulted from an excessive local endodermal proliferation early in gastric development .

Despite of antral web being a congenital disease, the timing of symptom presentation depends on the size of lumen. In general, lumen sizes larger than 1 cm do not lead to obstructive symptoms . So a few cases of antral web discovered in older children or adults. According to the literature, symptomatic presentation within 10 days postbirth is extremely rare . Most patients show intermittent atypical symptoms such as vague abdominal pain, vomiting, or abdominal distension. Some of them show melena or hematemesis .

In neonate period, there are many causes of non-bilious vomiting such as pyloric stenosis, feeding intolerance, milk allergy, metabolic disorders (e.g., hyperammonemia, metabolic acidosis), increased intracranial pressure, sepsis, necrotizing enterocolitis, or adrenal insufficiency . Necrotizing enterocolitis is often manifested through increased gastric residue with/without bloody stool after milk feeding. However, non-bilious vomiting even nil per os without abdominal distension gave an impression of congenital gastric outlet obstruction. Pyloric stenosis was excluded, because it is usually apparent after 2 or 3 weeks after birth.

For the diagnosis, USG and UGI series are useful. The conditions causing gastric obstruction on UGI series include prepyloricantral web, pyloric stenosis, pylorospasm, redundant or hypertrophied mucosal folds, perigastric adhesions, or heterotopic

pancreatic tissue . For antral web, UGI series reveals persistent, sharp band-like linear defect in antrum and the "double bulb sign". One is normal duodenal bulb and the other one is proximal antral chamber between web and pylorus . Up to UGI series findings, multiple hypertrophied redundant mucosal folds show usually multiple, more irregular than antral webs and do not encircle the stomach circumferentially .Perigastric adhesions are usually anterior and deform only one wall and ectopic pancreatic tissue has central niche . USG after filling the stomach with milk or saline shows an echogenic diaphragm-like structure in the antrum with gastric dilatation, and delay in gastric emptying with normal pylorus . From endoscopy, can find the small opening mimicking the pylorus, but it doesn't have a normal mucosal fold, normal peristaltic movement, and cannot close itself .

However, accurate diagnosis is difficult and often delayed even if the patient undergoes endoscopy or UGI series, because antral web is very rare and does not give an impression to the clinician who doesn't have previous knowledge about it. Patients often have been treated for ulcer or pyloric spasm . Asymptomatic antral web become worse from peptic ulcer with edema of antrum. In some cases, the wrong interpretation of UGI series and the partial improvement with H2-blocker may make believing that the obstruction was the result of chronic peptic ulcer .

The symptomatic antral web needs surgical correction . From anatomical lesion, antroplasty or web excision with or without pyloroplasty are possible surgical options .The lumen of antrum became very narrow after web excision even though considering for edematous change of antrum. For widening the lumen, pyloroplasty was done. The prognosis is very good after surgical correction . The endoscopic treatment is another option of antral web depending on the feasibility of endoscopic intervention.

Endoscopic transection is possible if the mucosal structure is uniform without major vessels or muscular or serosal layers and the membrane is tense and consistent with perpendicular insertion . Howeverendoscopy is impossible to perform in premature baby whose body weight is less than 2 kg.

Persistent non-bilious vomiting in neonate should be evaluated by USG after excluding possible another causes. Because, antral web is very rare but can cause gastric outlet obstruction from neonatal period. UGI series assuming antral web is important for accurate diagnosis. It can lead early surgical correction with favorable outcome.

Study 6 : Duodenal webwas done in*India*

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Antenatal diagnosis of congenital duodenal obstruction on ultrasonography is made by the presence of double-bubble appearance and polyhydramnios. Waever et al have reported that prenatal ultrasonography picked up an abnormality in 90% of cases (n=40) with duodenal atresia . In this series, only 2 patients had an antenatal diagnosis of intrinsic duodenal obstruction. Although this congenital anomaly, even if picked up antenatally does not warrant medical termination of pregnancy, early induction of labor, Caesarian section or fetal intervention, but an antenatal detection may suggest delivery in a tertiary center where surgical correction could be done postnatally without any delay.

As seen in three of patients, those with fenestrated duodenal membranes may present as late as infancy or childhood or occasionally even in adulthood . The fenestrated membrane may be choked with the food residue / foreign body. Another cause of delayed presentation is the gradual onset of atony and ineffective peristalsis in the dilated proximal segment of duodenum along with the development of patulous pylorus . An upper GI contrast study is needed in older children to diagnose a partial duodenal obstruction as was seen in two of our patients.

Although some studies have reported non-bilious vomiting as the most common presenting feature, majority of our patients had bilious vomiting indicating that the site of obstruction was post-ampullary. A plain X-ray abdomen with a characteristic ‘double-bubble’ sign was diagnostic in most neonates. The most common site of location is between the first and second parts (85%) . In a series of 10 patients, Rowe et al have described the location of a windsock anomaly to be preampullary in 40% of cases .

The delay in diagnosis was mainly due to fenestrated membranes, or missed diagnosis in children with associated lower G.I. obstruction requiring surgical intervention. About half of patients were brought beyond 1 month of age. In this series, 2 patients were operated elsewhere for malrotation during which the duodenal web was missed. There are several reports in the early 20th century where intrinsic duodenal obstruction has been missed during the Ladd’s procedure . Inability to pass a stiff catheter into the duodenum should raise the suspicion of a duodenal web and demands a careful inspection. Rowe et al described 10 patients with windsock anomaly of the duodenal. Only 25% of patients with duodenal web and associated low intestinal obstruction (n=4) had classical findings on X-Ray preoperatively. All were diagnosed on upper GI contrast series done for persistent vomiting after correction of low intestinal obstructions . In this series there were 2 patients with imperforate anus and 1 patient with esophageal atresia in which the diagnosis of duodenal web was missed at the time of initial surgery.

One case of double duodenal web in this series emphasizes the need for mandatory checking of distal patency of gut at the time of surgery for bowel atresia. Only 32 cases of double duodenal webs have been reported in literature till date to the best of our knowledge out of which 2 were reported in adults . Reid in his study of 140 patients of intrinsic duodenal obstructions, found only four double duodenal intrinsic obstructions, of which two were due to webs . Stinger et al have reported four patients with double duodenal obstructions of which two were due to webs .

The duodenotomy should be made in the lateral wall in the distal segment near the membrane. The membrane should be excised with electrocautery leaving the medial part in order to avoid injury to the Papilla of Vater. A proximal 'mega-duodenum' (duodenal diameter of 5 cm or more) may require imbrications or a tapering duodenoplasty procedure to avoid prolonged duodenal ileus .

The use of TAT for enteral feeding is controversial with no advantage being noted by some authors . However recent reports have emerged enumerating the benefits of TAT feeding . This was used in 8 of this patients all of whom had early institution of oral feeding. Prolonged duodenal ileus may sometimes persist for sometime post operatively; the use of TPN or TAT feeding may benefit this group of patients. The use of prokinetic agents is of doubtful value in these patients. Persisting signs of duodenal obstruction for more than three weeks post operatively should raise doubts of residual or incomplete excision of the duodenal membrane. This has to be confirmed radiologically before going in for a re-exploration.

With advancement in pediatric intensive care and anesthesia the survival rates for duodenal obstructions have improved to 90- 95% in the developed world; the major causes of mortality being associated life threatening congenital anomalies. However, there is no available data from the developing countries where a combination of life threatening congenital anomalies, pre-maturity, low birth weight and sepsis are considerable contributing factors to mortality. Crowded nurseries, nosocomial infections, cross infections and poor infra-structures were added

There were 4 deaths in the postoperative period giving a mortality rate of 22%. The causes of death were sepsis with refractory shock (n=2), aspiration (n=1), and necrotizing enterocolitis necessitating re- exploration (n=1). Three out four deaths were seen in preterm babies. Only one of the 4 preterm babies in this series survived. The mean hospital stay for the survivors was 18 days (range 11-25 days). The mean time taken to achieve oral feeds ad lib in these 14 patients was 10 days (range 7 to 13 days). Two patients survived fulminant Klebsiella sepsis. Total parenteral nutrition (TPN) was not used in any of the patients in this series. The 8 patients in whom TAT was used for

nutritional support had an earlier establishment of normal oral feeding pattern. For lack of numbers, any test of statistical significance could not be performed.

Study 7 : Intestinal malrotationwas done in *China*

Clinicalanalysis of 70 Cases of Neonatal Intestinal Malrotation Chongqing Medical Universityby ChenMingXiang

Retrospective study of the clinical data of70cases of neonatal congenital intestinal malrotation. The70cases were underwent the Ladd surgery, there were50cases(71.4%) correctly diagnosed as congenital intestinal malrotation before surgery and other20cases were found in laparotomy. There were55cases of Ladd band oppression,51cases of midgut volvulus,36cases of upper jejunum membranous adhesions,7cases of intestinal necrosis, and1case of cecum oppression. Fourteen cases in all were accompanied with other digestive tract malformation(20%), with3cases of duodenal atresia,2cases of annular pancreas,2cases of jejunal atresia,2cases of ectopic pancreas,1case of congenital short bowel syndrome,1case of Meckel's diverticulum,1case of acromphalus,1case of duodenal ectopic thyroid and1case of paraduodenal hernia.7cases have outside the alimentary tract malformation(20%),with five cases of atrial septal defect,1case of ventricular septal defect and hydronephrosis.

65 cases were cured after operation, and the curative rate reached 92.9%,1case did not obviously improve after operation in short term, and4cases given up treatment.

Most of congenital malrotation onset1-3days after birth,In clinical, neonatal congenital intestinal malrotation is incomplete obstruction of the duodenum, it may attack recurrently,the incidence of volvulus and bowel necrosis is high. and most of them can be diagnosed through imageology. If there were not complicated by generally gangrene of intestine or other serious congenital malformation, traditional Ladd operation method could get favorable prognosis.

Study8 : Annular pancreas was done in*USA*

Annular pancreas in children by Jimenez JC, Emil S, Podnos Y, Nguyen N InstitutionDivision of Pediatric Surgery, Irvine Medical Centre, Orange, California, USA

A 10-year review of clinical, radiological, and prognostic findings in patients with annular pancreas. Retrospective review of all cases of annular pancreas between 1993 and 2002 The incidence of this congenital anomaly is reported as 1–3 in 20,000 and more common in males. Detection of the condition is variable, as it may be asymptomatic and therefore detected only incidentally or at postmortem. However, the vast majority of cases are diagnosed either prenatally or in the first few days of life. If the condition is not diagnosed prenatally or does not present with complications in early life, it may be undetected until adulthood. The detection of an annular pancreas may occur at any time during adulthood and may be discovered either incidentally or after presentation due to a complication such as pancreatitis. There is a strong association between annular pancreas and other congenital abnormalities—up to 71% of cases have coexisting congenital anomalies. The most common association is with Down syndrome. However, there may be a wide range of associated cardiac and gastrointestinal anomalies (including Hirschsprung's disease and imperforate anus), as well as tracheo-oesophageal fistula and oesophageal atresia.All patients required surgical intervention, and all patients survived to be discharged from hospital (mean stay, 24 days).

CLINICAL ASPECTS OF GASTRIC OUT LET OBSTRUCTION

Vomiting is the cardinal symptom of gastric outlet obstruction. Vomiting usually is described as nonbilious, and it characteristically contains undigested food particles. In

the early stages of obstruction, vomiting may be intermittent and usually occurs within 1 hour of a meal.

Patients with gastric outlet obstruction resulting from incomplete obstruction typically present with symptoms of gastric retention, including early satiety, bloating or epigastric fullness, and weight loss. They are frequently malnourished and dehydrated and have a metabolic insufficiency.

Physical examination often demonstrates the presence of chronic dehydration and malnutrition. A dilated stomach may be appreciated as a tympanitic mass in the epigastric area and/or left upper quadrant.

Dehydration and electrolyte abnormalities can be demonstrated by routine laboratory examinations. Increases in BUN and creatinine are late features of dehydration. Prolonged vomiting causes loss of hydrochloric (HCl) acid and produces an increase of bicarbonate in the plasma to compensate for the lost chloride and sodium. The result is a hypokalemic hypochloremic metabolic alkalosis. Alkalosis shifts the intracellular potassium to the extracellular compartment, and the serum positive potassium is increased factitiously. With continued vomiting, the renal excretion of potassium increases in order to preserve sodium. The adrenocortical response to hypovolemia intensifies the exchange of potassium for sodium at the distal tubule, with subsequent aggravation of the hypokalemia.

The stomach is located mainly in the left upper quadrant beneath the diaphragm and is attached superiorly to the esophagus and distally to the duodenum. The stomach is divided into 4 portions, the cardia, the body, the antrum, and the pylorus. Inflammation, scarring, or infiltration of the antrum and pylorus are associated with the development of gastric outlet obstruction.

The duodenum begins immediately beyond the pylorus and mostly is a retroperitoneal structure, wrapping around the head of the pancreas. The duodenum classically is divided into 4 portions. It is intimately related to the gallbladder, liver, and pancreas;

therefore, a malignant process of any adjacent structure may cause outlet obstruction due to extrinsic compression.

Most patients benefit from an initial period of gastric decompression, hydration, and correction of electrolyte imbalances. In patients who are severely malnourished, postponing surgical intervention until the nutritional status has been optimized may be wise. In selective cases, some patients may benefit from total parenteral nutrition (TPN) or distal tube feeding (eg, placed via a percutaneous jejunostomy).

Overall, every patient with gastric outlet obstruction deserves evaluation by a surgeon. Even if the patient has unresectable disease, palliative surgical measures may improve the quality of life.

Laboratory Studies

- Obtain a CBC. Check the hemoglobin and hematocrit to rule out the possibility of anemia.
- Obtain an electrolyte panel. As noted previously, identifying and correcting electrolyte abnormalities that tend to occur is essential.
- Liver function tests may be helpful, particularly when a malignant etiology is suspected.
- A test for *H pylori* is helpful when the diagnosis of PUD is suspected.

Imaging Studies

- Plain abdominal radiographs, contrast upper GI studies (Gastrografin or barium), and Ultrasonography. Plain radiograph of the abdomen. Enlarged stomach with calcified content. Contrast study demonstrating an enlarged stomach. The point of obstruction is visualized at the pyloric-duodenal junction (string sign).
- Plain radiographs, including the obstruction series (ie, supine abdomen, upright abdomen, chest posteroanterior), can demonstrate the presence of gastric dilatation and may be helpful in distinguishing the differential diagnosis.

Medical Therapy

Initial management of gastric outlet obstruction (GOO) should be the same regardless of the primary cause. After a diagnosis is made, admit patients for hydration and correction of electrolyte abnormalities. Remembering that the metabolic alkalosis of GOO responds to the administration of chloride is important; therefore, sodium chloride solution should be the initial IV fluid of choice. Potassium deficits are corrected after repletion of volume status and after replacement of chloride. Place a NGT to decompress the stomach. Occasionally, a large tube is required because the undigested food blocks tubes with small diameters.

SurgicalTherapy

Surgery is indicated in cases of gastric outlet obstruction in which there is significant obstruction and in cases where medical therapy has failed.Endoscopic balloon therapy may be attempted as an alternative to surgery, with balloon dilation reporting success rates of 76% after repeat dilations. The operation usually performed is an Pyloromyotomy, Pyloroplasty,Duodenoduodenostomy , Duodenoplasty, Release of Band .

RESULTS

During this 5-year period that encompassed in pediatrics surgery department *(between July 2008 and June 2013),* there were 70 cases of GOO, of which 30 patient

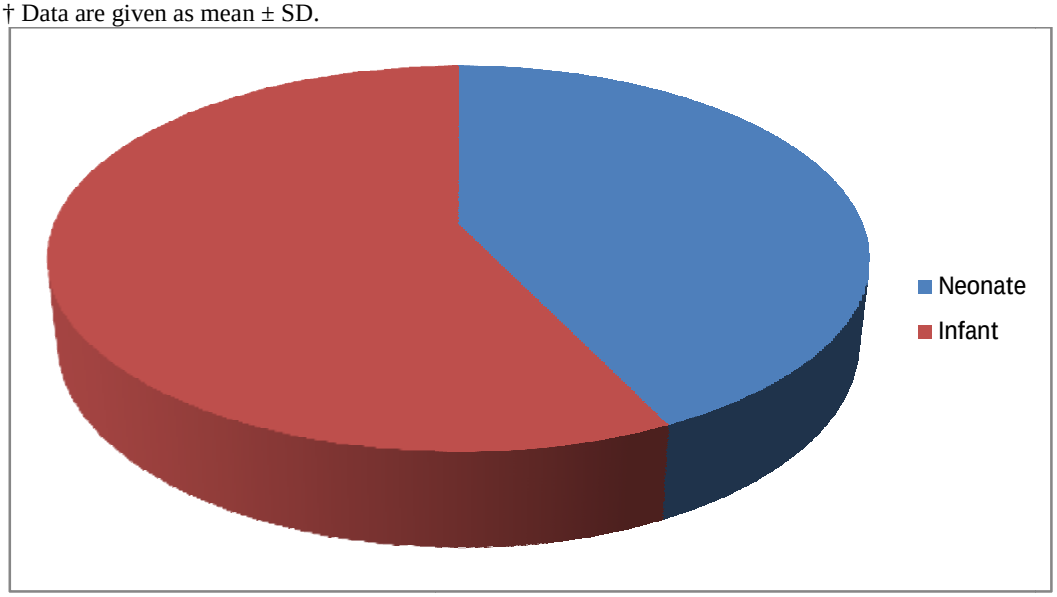
were neonate (age from 1 day up to 28 days n= 30) and 40 patient were infants (age from 29 days up to 1 year n =40). [67]

Tables 1 , 2 , 3 and4 provides relative information regarding the demographic characteristics (age , gender , nationality and residence) of neonate versus infants groups.

Table 1:Distribution of patients according to age

	Neonate	Infant
Age	1day – 28 days	29 days – 1 year
Number	30	40
%	42.8 %	57.2 %M
Mean SD (days) †	8.806 ± 7.05	45.175 ± 12.90

† Data are given as mean ± SD.



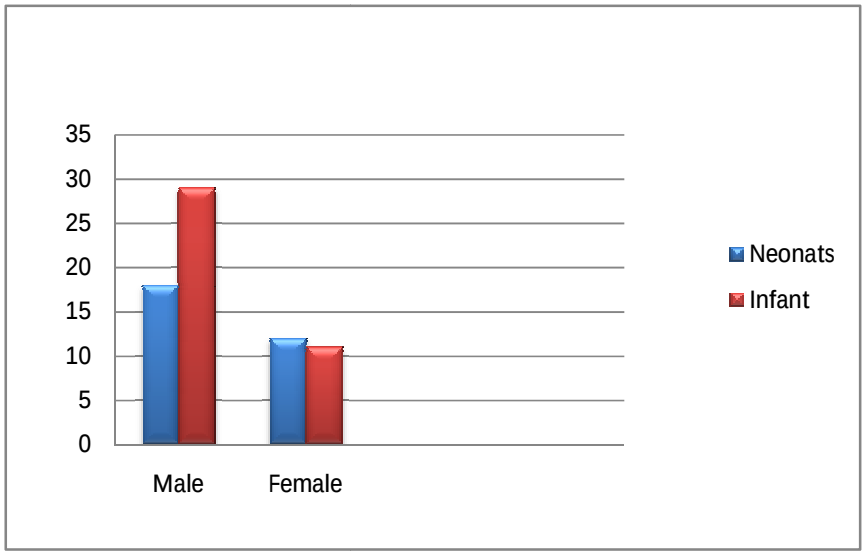
Graph 1:Distribution of patients according to age

Table 2:Distribution of patients according to gender

	Neonate(n= 30)	**infants(n =40)**	***P* value***

Gender (n)			
Male	18	29	0.454
%	60%	72.5%	
Female	12	11	0.454
%	40%	27.5%	

***** Student *t* test and Fisher's exact test results with a probability value of ≤ .05 were considered statistically significant.



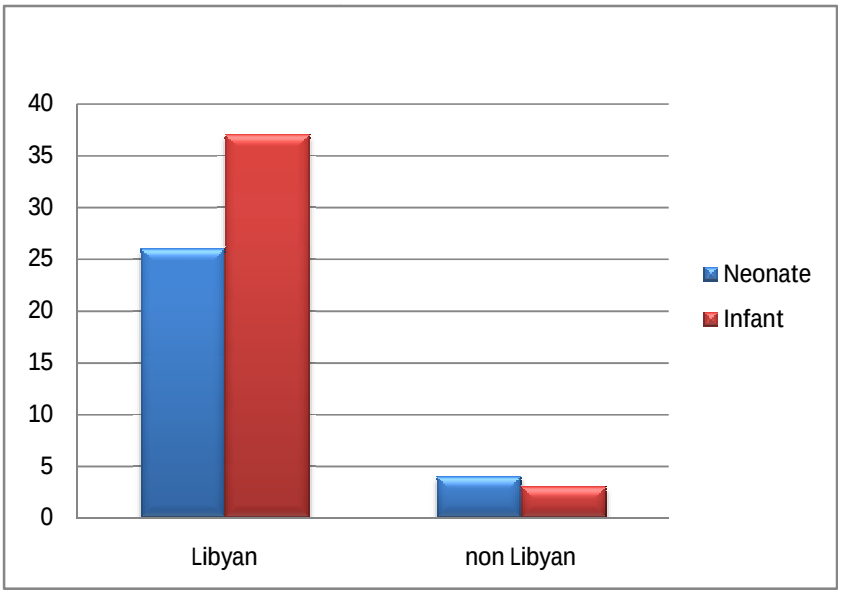
Graph 2:Distribution of patients according to gender

Table 3:Distribution of patients according to nationality

	Neonate(n= 30)	**infants(n =40)**	***P* value***
Nationality (n)			0.451

Libyan	26	37	
%	87%	93%	
			0.451
Non Libyan	4	3	
%	13%	7%	

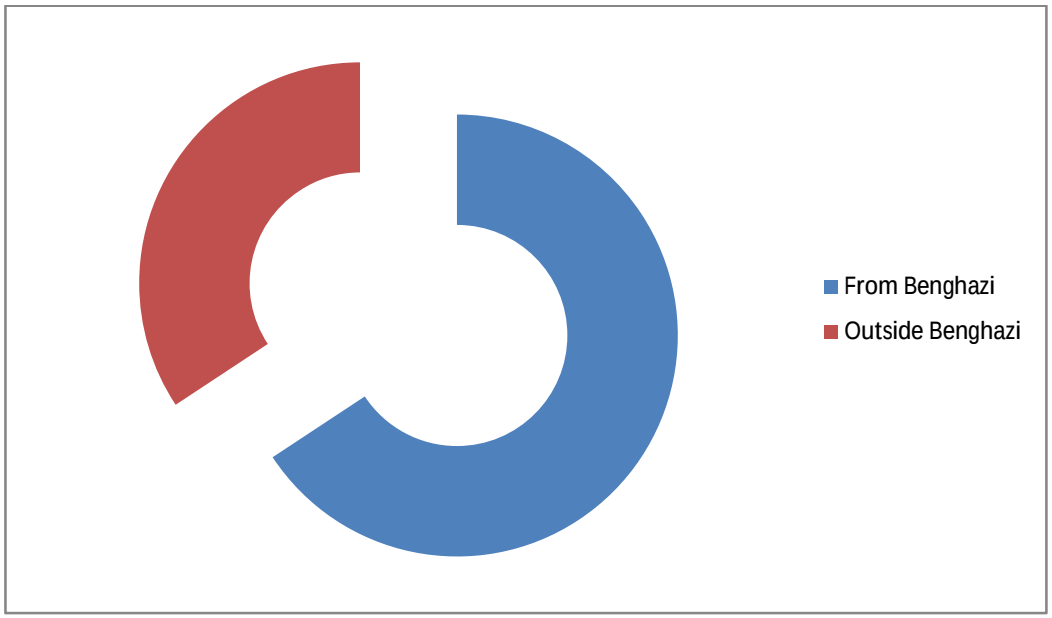
***** Student *t* test and Fisher's exact test results with a probability value of ≤ .05 were considered statistically significant.



Graph 3:Distribution of patients according to nationality

Table 4:Distribution of patients according to residence

Patients	Number	Percentage
From Benghazi	46	65.7 %
Outside Benghazi	24	34.3 %



Graph 4:Resedence of patients

Table 5A provide the early clinical presentation here the vomiting as the cardinal symptoms mainly non bilious and epigastric fullness as the cardinal sign

Table 5A : Early presentations

Presentation	Number	Percentage
Projectile non bilious vomiting	47	67.1 %

Non bilious vomiting	14	20 %
Bilious vomiting	9	12.8 %
Epigastric fullness	64	91.4 %
Epigastric mass (Olive)	25	35.7 %

Table 5B provide the late presentation which more in patients from out side Benghazi

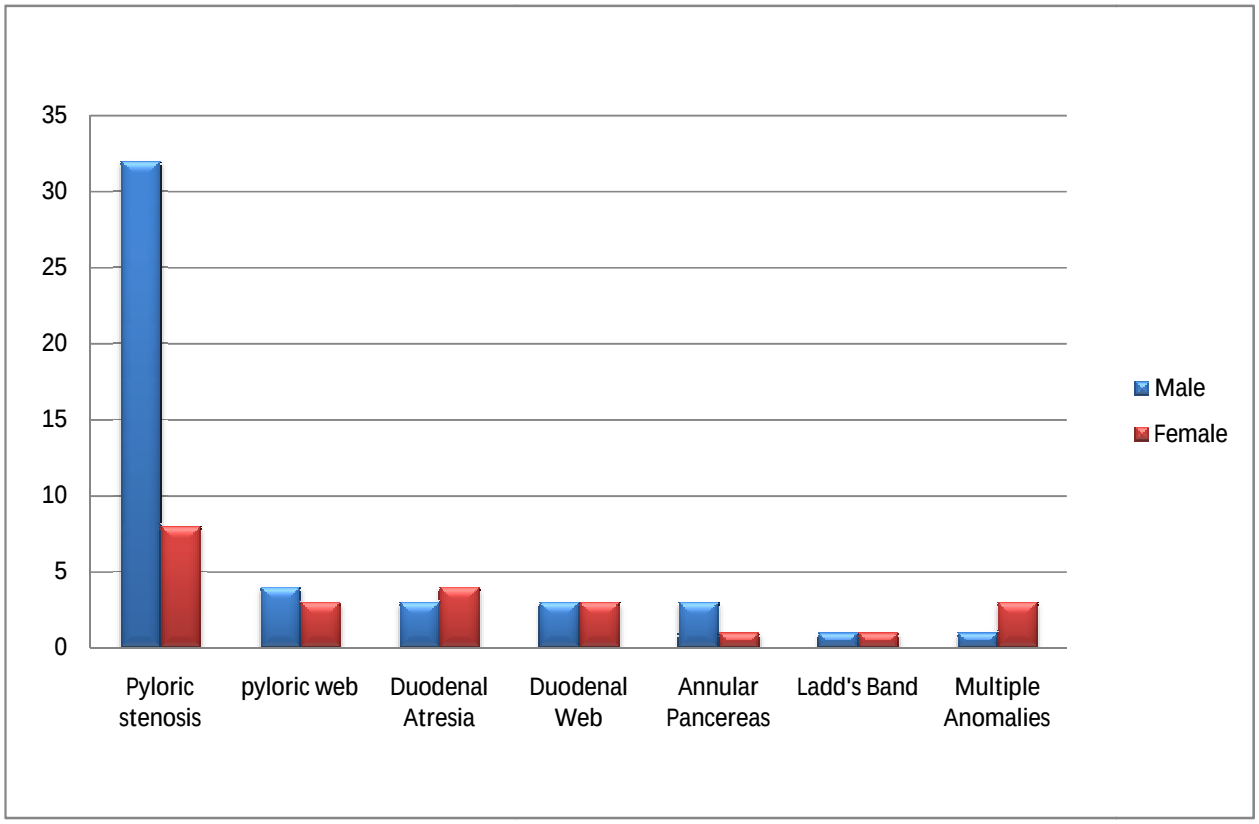
Table 5B : Late presentations

Presentation	From Benghazi N = 46	Outside Benghazi N = 24
Dehydration	1	5
Electrolyte disturbance	0	2
Anemia	1	1

Table 6provides relative information regarding the causes of GOO and sex ratio. The majority of patients in this study had pyloric stenosis (n=40) ,out of those there were 32 male and 8 female, giving a ratio of 4:1 which corresponding with other studies , where 7 patientshad duodenal atresia . Other causes of GOO has no sex related incidence difference, we notice in our study, the annular pancreas constitutes (n=4) giving a ratio of 3:1.

Table 6: Causes of GOO

Causes Of GOO	Male	Female	Ratio
Pyloric stenosis	32	8	4:1
pyloric web	4	3	1.3:1
Duodenal Atresia	3	4	1:1.3
Duodenal Web	3	3	1:1
Annular Pancreas	3	1	3:1
Ladd's Band	1	1	1:1
Multiple Anomalies	1	3	1:3



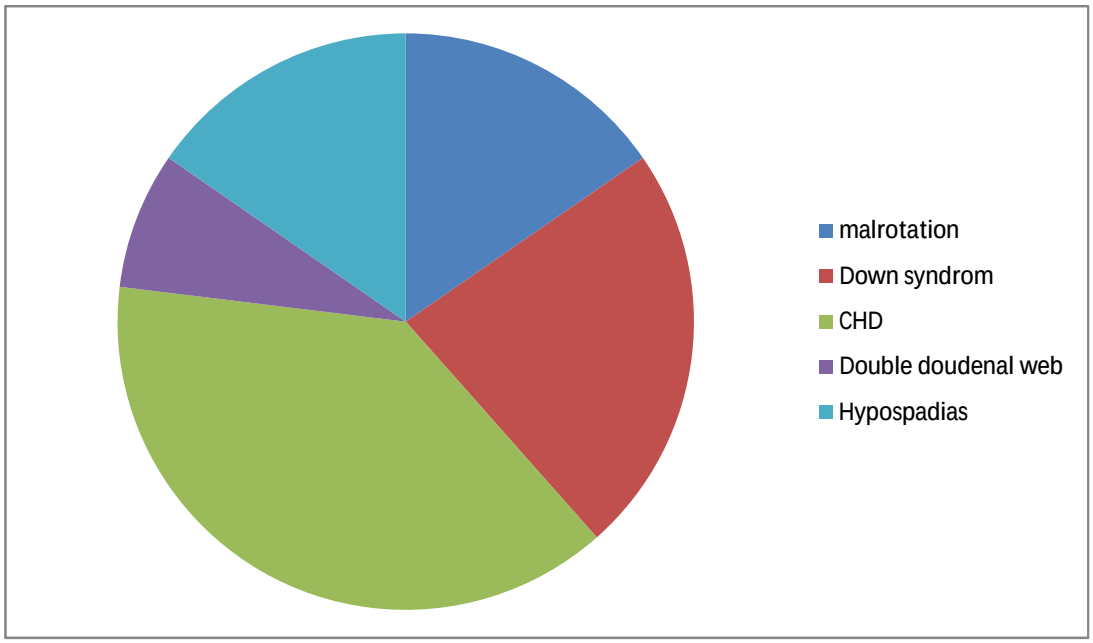
Graph 5 : Causes of GOO

Table 7 shows the associatedanomalies discovered in our series with total number of 13 which gives ratio of 18.5%

Table 7: Associated congenital anomalies

Associated congenital anomalies	Number

Malrotation of gut	2
Down syndrome	3
Congenital heart disease	5
Double duodenal web	1
Hypospadias	2



Graph 6:Associated cong. anomalies

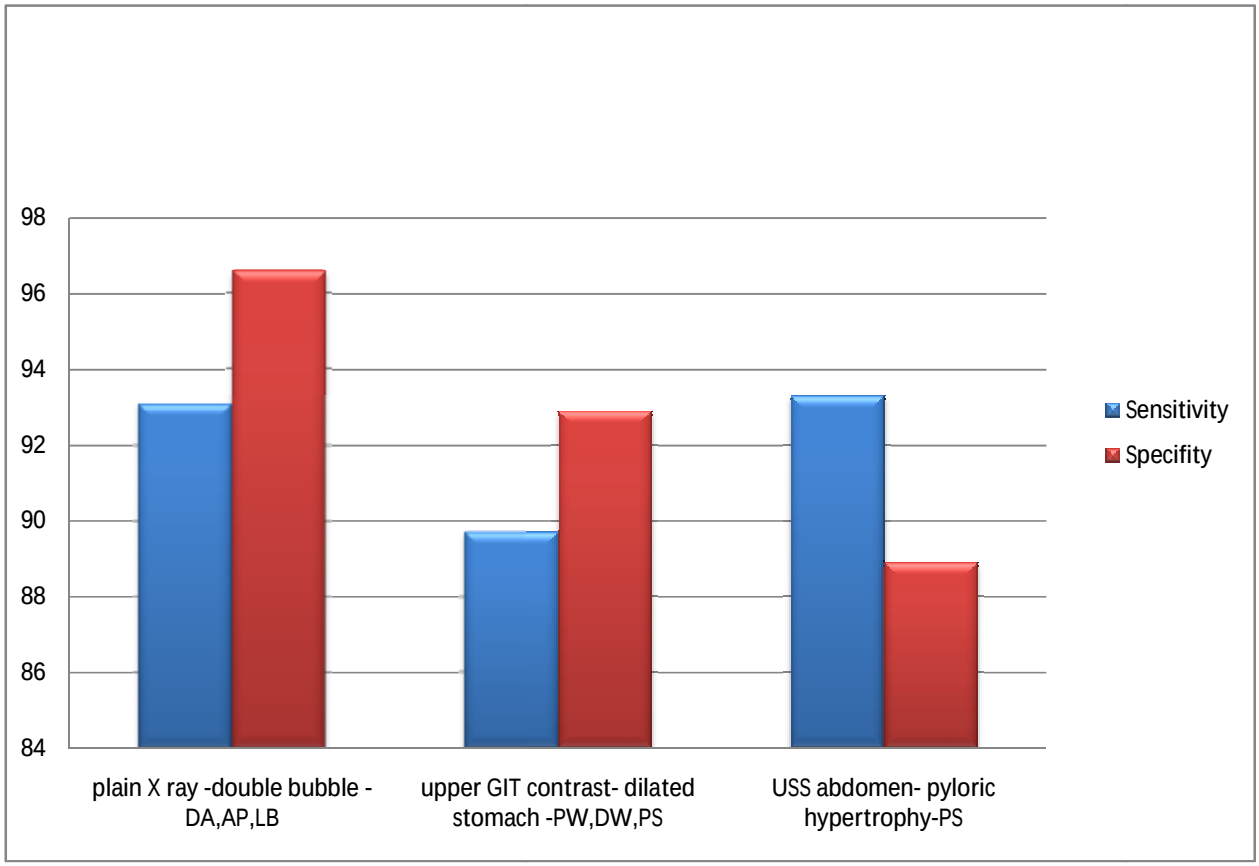
Table 8 A radiological study was performed for all patients in this study, most common X ray findings in diagnosis of duodenal atresia, annular pancreas and Ladd's band is double bubble sign, which is very sensitive in detecting the abnormality, with

sensitivity 93 % and specificity of 96%, where dilated stomach is seen as the prominent sign in duodenal web, pyloric web and pyloric stenosis during the upper GIT contrast ,with high positive predictive value (93%). Ultrasound study was performed for all patients with clinical diagnosis of pyloric stenosis, the pyloric hypertrophy is the hall mark, the sensitivity of this test is high in detecting the underlying disease is 93 % and specificity 89 %.

Table 8: Sensitivityand specificity of radiological findings

Image	**Finding**	**Disease**	**Sensitivity Estimate 95% CI**	**Specificity Estimate 95% CI**
Plain X-ray abdomen	Double Bubble	DA,AP,LB	0.931 [0.78 to 0.981]	0.966 [0.828 to 0.994]
Upper GIT Contrast	Dilated stomach	PW,DW,PS	0.897 [0.736 to 0.964]	0.929 [0.774 to 0.98]
USS abdomen	Pyloric hypertrophy	PS	0.933 [0.702 to 0.988]	0.889 [0.672 to 0.969]

DA =Duodenal Atresia, **AP**=Annular Pancreas, **LB**=Ladd's Band, **DW** =Duodenal Web, **PS**=Pyloric Stenosis, **PW**=Pyloric Web



Graph 7: Sensitivity and specificity of radiological findings

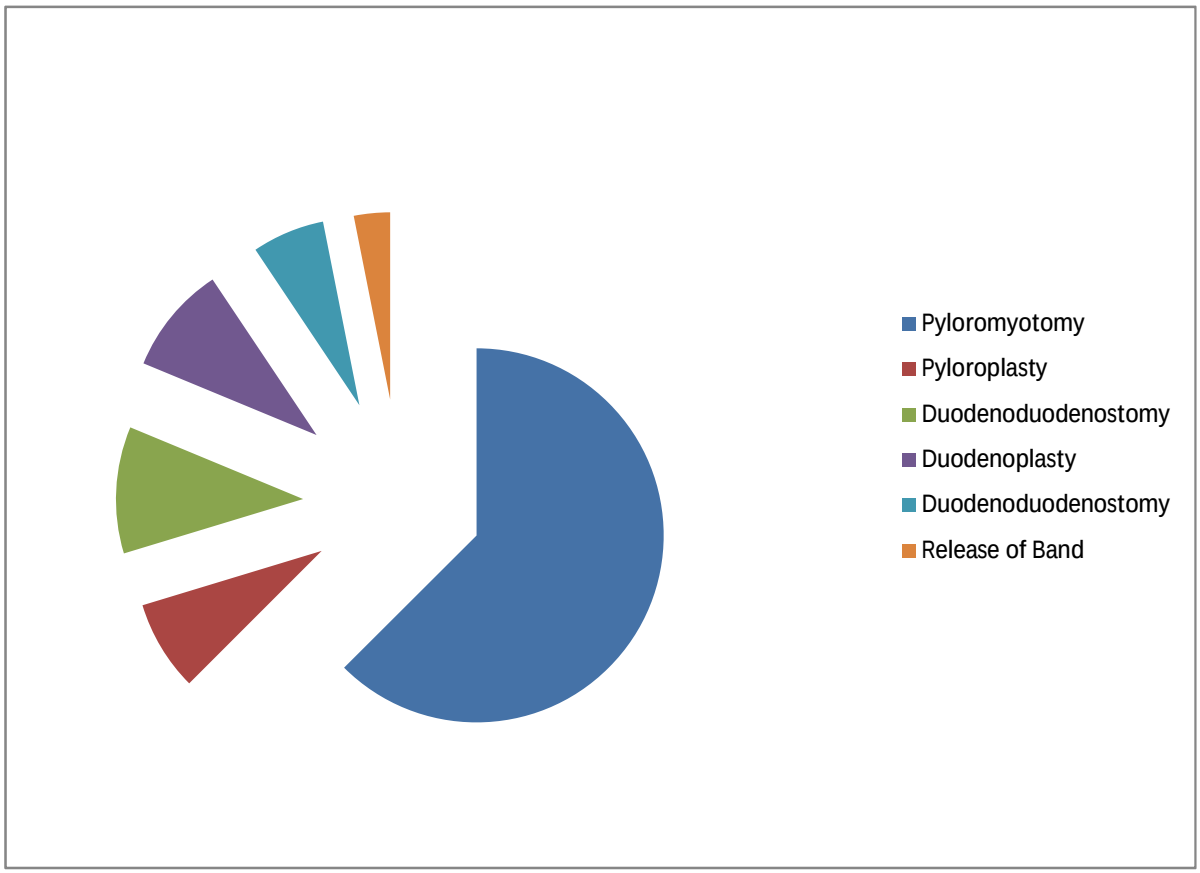
Table 9 The treatment of gastric outlet obstruction depends on the cause, but is usually surgical, In the current study, pyloromyotomy was the most frequent type of surgical procedure performed.

The types of surgical procedures reveal significant difference between the 2 groups in pyloromyotomy, pyloroplasty and duodenoduodenostomy (P <0.05) and no significant difference between the 2groups in duodenoplasty, duodenoduodenostomy(for annular pancreas)and Release of band (P >0.05) this difference is because most of the patients with pyloric stenosis are present relatively later where as the patient with congenital duodenal atresia typically present in the first week of life.

Table 9: Types of surgical procedures

Surgical procedure	Neonate(n= 30)	infants(n =40)	*P* value*
Pyloromyotomy	8	32	**0.001**
Pyloroplasty	5	2	**0.011**
Duodenoduodenostomy	6	1	**0.037**
Duodenoplasty	5	1	**0.077**
Duodenoduodenostomy (for annular pancreas)	3	1	**0.306**
Release of band	0	2	**0.503**

***** Student *t* test and Fisher's exact test results with a probability value of ≤ .05 were considered statistically significant.

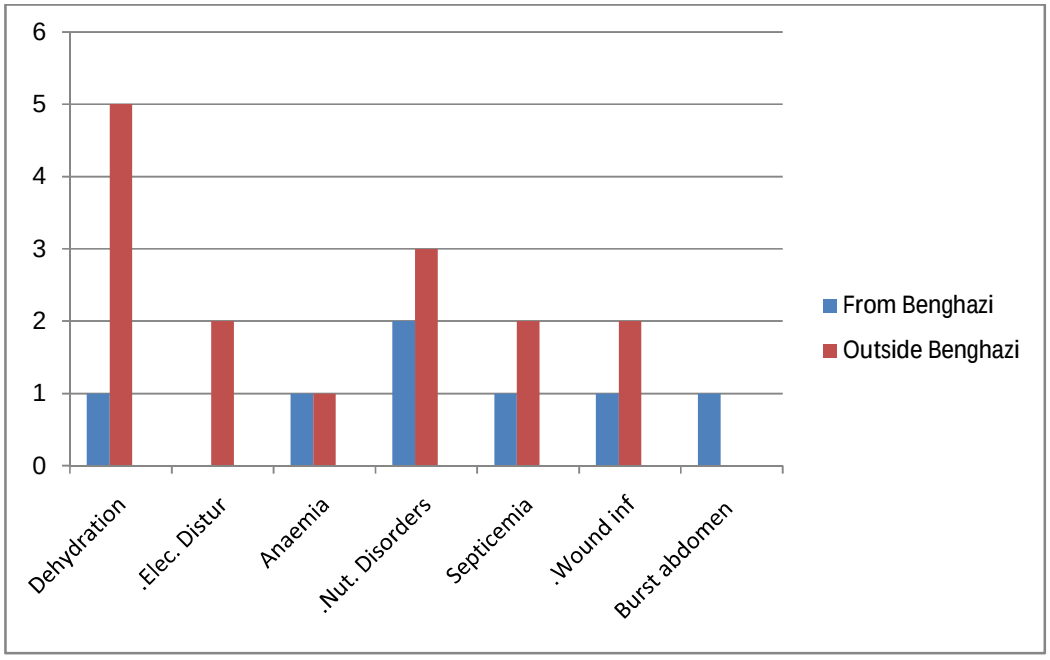


Graph 8: Types of surgical procedures

Table 10 shows postoperative complications which more in patients from outside Benghazi and rural areas

Table 10: postoperative complications

Complication	From Benghazi	Outside Benghazi
Nutritional disorders	2	3
Septicemia	1	2
Wound infection	1	2
Burst abdomen	1	0

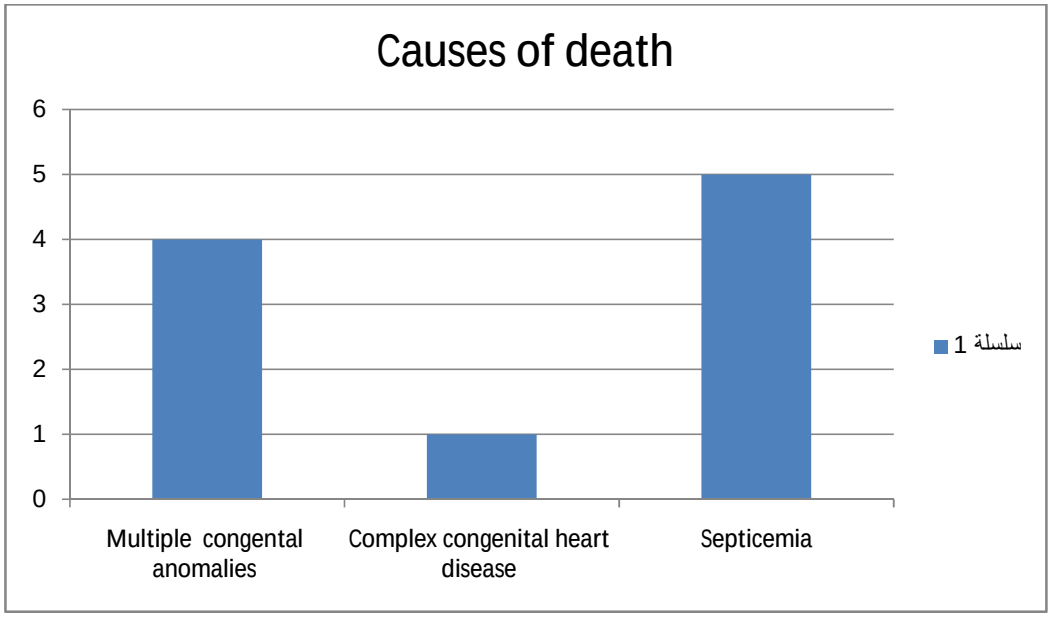


Graph 9: Presence of complication

Table 11lists the causes of death of the patient the total number is 10 giving ratio of 14.28 % . The most common causes were multiple congenital anomalies and septicemia

Table 11: Causes of death

Causes of death	Number
Multiple Anomalies	4
Complex Congenital Heart Disease	1
Septicemia	5



Graph 10: Causes of death

DISCUSSION

This study was conducted to describe our own experiences in the management of this disease . In my review the total number were 70 patients . The patients divided in 2 groups , the first group aged from 1 day up to 28 days called neonates , the second group aged from 29 days up to 1 year called infants [67]. The highest age incidence of the patient at presentation were from Day 15 up to Day 35 . The youngest age was 1 day old and oldest age was 3 months old .

Males were more affected giving ratio of 67.14% while females were 32.86% . 3 patients were Down syndrome 2 had duodenal atresia and 1 had annular pancreas giving ratio of 27.2 % which nearly same as other reviews [18,51] , other associated anomalies were congenital heart disease 5 cases and 2 babies with hypospadias .

Libyan babies were 63 giving ratio of 90 % and non-Libyan were 7 giving ratio of 10 % . 46 babies from Benghazi giving ratio of 65.7 % and 24 babies from outside Benghazi giving ratio of 34.3 %

The clinical presentation of gastric outlet obstruction in our patients was not different from those in other studies [2,7] with projectile nonbillous vomiting being common which found in 47 patients giving ratio around of 67% . History of polyhydramnios was not mentioned in all patients because some mothers were not available during admission.

Epigastric abdominal fullness found in 64 patients (91.4 %) , epigastric mass (olive) palpated in 25 patients who later diagnosed as pyloric stenosis and this gave ratio of 62.5% which was corresponding with other studies [12] .

The majority of patients in this study had pyloric stenosis (n=40 , 57.1 %) ,out of those there were 32 male and 8 female, giving a ratio of 4:1 which corresponding with other studies [7,9,69] , the annular pancreas constitutes 5.7 % (n=4) affecting more

males which as other studies [50,51] giving a ratio of 3:1 . Other causes of GOO have no sex related incidence difference which were pyloric web (n=7, 10%) , duodenal web (n=6 , 8.6%) , Lad's band (n=2 , 2.8%) and multiple anomalies (n=4 , 5.7 %)

In agreement with other studies [1,64], the diagnosis of gastric outlet obstruction was based on clinical presentation and an upper gastrointestinal barium study, and confirmed by intra-operative findings. Other diagnostic investigations included abdominal ultrasound and computerized tomography (CT) scan.

A radiological study was performed for all patients in this study , most common X ray findings in diagnosis of duodenal atresia, annular pancreas and Ladd's band is double bubble sign, which is very sensitive in detecting the abnormality, with sensitivity 93 % and specificity of 96%, where dilated stomach is seen as the prominent sign in duodenal web, pyloric web and pyloric stenosis during the upper GIT contrast ,with high positive predictive value (93%).

Ultrasound study was performed for all patients with provisional diagnosis of pyloric stenosis, the pyloric hypertrophy is the hall mark, the sensitivity of this test is high in detecting the underlying disease is 93 % and specificity 89 %.

In all our patients investigations are obtained. CBC, check the hemoglobin and hematocrit to rule out the possibility of anemia, and an electrolyte panel. As noted previously, identifying and correcting electrolyte abnormalities that tend to occur is essential. Liver function tests may be helpful.

Preoperative resuscitation for dehydration (6 patients ; 1 from Benghazi and 5 from outside Benghazi) and electrolyte disturbance (2 patients ; from outside Benghazi) applied to the patients mostly from outside of Benghazi because of delay in presentation due to long distance , lack of subspecialty and parent ignorance. 2 patient with anemia (1 from Benghazi and 1 from outside of Benghazi) .

The treatment of gastric outlet obstruction depends on the cause, but is usually surgical, In the current study, pyloromyotomy was the most frequent type of surgical procedure performed.

The types of surgical procedures reveal significant difference between the 2 groups in pyloromyotomy, pyloroplasty and duodenoduodenostomy (P <0.05) and no significant difference between the 2 groups in duodenoplasty, duodenoduodenostomy (for annular pancreas)and release of band (P >0.05) this difference is because most of the patients with pyloric stenosis are present relatively later where as the patient with congenital duodenal atresia typically present in the first week of life.

Delay in diagnosis was mainly due to webs because of partial obstruction caused by these webs

Double duodenal web found in 1 patient in this series so this emphasize the need for mandatory checking of distal patency of gut at time of surgery for bowel atresia

Post-operative complications include nutritional disorders in 5 patients , sepsis in 3 patients , wound infection in 3 patients and burst abdomen in 1 patient

Prolonged duodenal ileus may sometimes persist for some time post operatively this will lead to keeping patients prolonged time NPO so the use of TPN and TAT feeding may benefit this group which unfortunately not available in our department and leads to death of 3 patients (30 % of total mortality) due to postoperative nutritional disorders complications .

The overall mortality rate in this study was 14.28 % which nearly the same as other studies [66] and it was significantly associated with presence of other congenital anomalies.

CONCLUSION

- Congenital pyloric stenosis is the commonest cause of gastric outlet obstruction.
- Early presentation to the hospital givesoptimal results in management
- The X ray , ultrasound and the upper GIT contrast study are highly sensitive in detecting the underlying disease.
- Pyloromyotomy is the most common surgical procedure performed with no significant complications.
- The outcome in patients with gastric outlet obstruction is further marred by the high incidence of associated anomalies.
- We infer that a though the facilities for antenatal ultrasonography are amply available even in our city Benghazi the pick-up rates for congenital abnormalities are dismal and this will affect antenatal diagnosis .

RECOMENDATION

- Antenatal diagnosisshould be performed
- Presence of one tertiary pediatric surgery center in east of Libya leads to undue delay in the surgical management of such neonates , thus affecting outcomes adversely.
- Post-operative complications and mortality rate can be reduced by providing our department by nutrition supplementation (TPN) and trans anastomotic tubes (TAT)
- Lack of some medical information require improving the file system
- Awareness of practitioners in rural area by regularworkshops and meetings gives best results in earlydetection of the disease

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الملخص

الاهداف : - الهدف من هذه الدراسة هو مراجعة وتحليل اسباب انسداد مخرج المعدة وعلاماته واعراضه المختلفة ومراجعة الانواع المختلفة للتحاليل وطرق العلاج ونتائجها .

المرضي والطرق : - تم ادراج جميع الحالات التي دخلت لمستشفى الاطفال بنغازي في مدة 5 سنوات خلال الفترة من يوليو 2008 وحتي يونيو 2013 . وقد تم دراسة وتحليل جميع السجلات الطبية من تاريخ مرضي والعلامات والاعراض والفحوصات السريرية ونتائج التحاليل المعملية الخاصة بهم .وقد تم تقسيم المرضي الي مجموعتين مجموعة حديثي الولادة ومجموعة الاطفال حتي عمر سنة واحدة .

النتائج : - خلال الفترة المستهدفة في الدراسة تم اكتشاف 70 حالة كان تشخيصها انسداد مخرج المعدة ووجد متوسط الاعمار 7.05 ±8.80645 يوم لحديثي الولادة 12.90 ±45.175 يوم للمرضي حتي عمر سنة واحدة . المرضي الذين يشكون من قئ غير صفراوي كان عددهم 47 بنسبة 67.14 % . معظم المرضي في هذه الدراسة تم تشخيصهم بالضيق البوابي بعدد 40 حالة بنسبة 57.1 % 32 ذكر و 8 انثي معطيا معدل 4 : 1 كما تم ملاحظة ان عدد المرضي المشخصين بالبنكرياس الحلقي 4 مرضي بنسبة 5.7 % ثلاثة ذكور وانثي واحدة معطيا معدل 3 : 1 اما الاسباب الاخري لانسداد مخرج المعدة لا توجد علاقة بين عدد الذكور والاناث . فحوصات الاشعة السينية (علامة الفقاعة المزدوجة) اظهرت حساسية عالية في تشخيص المرض فقد كانت نسبة الحساسية 93 % والنوعية 96 % وعلامة توسع المعدة لوحظ خلال فحوصات الاشعة الملونة للقناه الهضمية العليا بنسبة 93 % للقيمة التنبؤية الموجبة . تم اجراء فحص الموجات فوق الصوتية لجميع المرضي الذين كان تشخيصهم المبدئي الضيق البوابي حيث وجد ان تضخم الجزء البوابي للمعدة علامة اساسية بنسبة 93 % للحساسية و 89 % للنوعية. وجد ان هناك اختلاف بين نوعية التدخل الجراحي للمجموعتين فقد كان مفاغرة اجزاء الاثني عشر هي الاكثر في مجموعة حديثي الولادة وشق العضلة البوابية هو الاكثر في المجموعة الاخري.

الأستنتاج : - يعتبر الضيق البوابي من اكثر مسببات انسداد مخرج المعدة في الاطفال وعملية شق العضلة البوابية هي الشائعة كتدخل جراحي لمعالجة هذا المرض وبدون مضاعفات تذكر . كما ان الاشعة السينية والفحص بالموجات فوق الصوتية ذات حساسية عالية في اكتشاف الاسباب المختلفة لأنسداد مخرج المعدة .