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Acute pancreatitis at Al-jala Hospital-Benghazi

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Abstract

Acute pancreatitis

introduction: Acute pancreatitis is a disease of pancreas, an organ and gland in the upper abdominal area .It is characterized by a sudden onset and from mild to severe inflammation of pancreas and can be life –threatening and led to other serious complications.

Aim: review epidemiological and clinical characteristic of the Acute pancreatitis**.**

Method: Files of all patients diagnosed as acute pancreatitis during two years 1 of Jan. of 2007 to 31 of Decem. 2008 who were admitted to Aljala hospital were included**.**

Results: A total of 50 patients were admitted to Aljala Hospital during a period of two years 2007-2008 with diagnosis of acute pancreatitis , male and female were equal in number, their mean age was 55.6±15 years**,** 88% of patient their age was between 21- 60 years.

History of chronic disease diabetic, hypertension….ect was positive in 56% of patients. Surgical history post chlolecyctectomy was recorded positive in 32% of patients.

Conclusion: We revealed from this study that the sex distribution was similar the majority of patients had typical presentation and only 20% of the patients presented in the first day

Recommendation: Prospective study for longer duration and more no. of patients are strongly needed.

Dedication

In this dedication I would give thanks to every single person that helped me for doing this project . I would thank my father (may Allah bless him), my mother and all of my family .
I would give special thanks to the doctor Saeid Omer Al-Soaeiti who helped me a lot and I am given special thanks to doctor Tunis Almedan whom helped me in result and made its tables and figures….

So I hope that this project will be in the appropriate level that you will like it.

List of table

List of figure

Contents **page**

List of symbols

1. **ADIS adult immune deficiency syndrome**
2. **ARDS adult respiratory distresses syndrome**
3. **APACHE acute physiology and chronic health evaluation**
4. **CBC complete blood count**
5. **CTscan computer tomography scan**
6. **CFTR cystic fibrosis trans membrane conductance and regulator gene**
7. **CCK secretin cholycystokinin**
8. **CMV cytomegalo virus**
9. **ERCP endoscopic retrograde cholingio pancreatic gram**
10. **GRP gastrine releasing peptide**
11. **IPMN intra papillary mucinous neoplasm**
12. **ICU intensive care unit**
13. **IL6AND IL8 interleukins 6and 8**
14. **IV intra venous**
15. **PRSS1 protease serine1**
16. **SIRS systemic inflammatory response syndrome**
17. **TNF ALPH tumor necrotizing alpha**
18. **VIP vasoactive intestinal peptide**
19. **VZV varicella zoster virus**
20. **WBC white blood cell**

Chapter 1

Introduction

Normal pancreatic function

The pancreas is a gland located in the upper posterior abdomen. It is responsible for insulin production (endocrine pancreas) and the manufacture and secretion of digestive enzymes (exocrine pancreas) leading to carbohydrate, fat, and protein metabolism. Approximately 98% of the gross weight of the pancreas supports the exocrine function, and the remaining 2% is involved with endocrine function[1].

In normal pancreatic function, up to 15 different types of digestive enzymes are manufactured in the rough endoplasmic reticulum, targeted in the Golgi apparatus and packaged into zymogens as proenzymes. When a meal is ingested, the vagal nerves, vasoactive intestinal polypeptide (VIP), gastrin-releasing peptide (GRP), secretin, cholecystokinin (CCK), and encephalins stimulate release of these proenzymes into the pancreatic duct.

The proenzymes travel to the brush border of the duodenum, where trypsinogen, the proenzyme for trypsin, is activated via hydrolysis of an N-terminal hexapeptide fragment by the brush border enzyme enterokinase. Trypsin then facilitates the conversion of the other proenzymes to their active forms.[1]

A feedback mechanism exists to limit pancreatic enzyme activation after appropriate metabolism has occurred. It is hypothesized that elevated levels of trypsin, having become unbound from digesting

food, lead to decreased CCK and secretin levels, thus limiting further pancreatic secretion.

Because premature activation of pancreatic enzymes within the pancreas leads to organ injury and pancreatitis, several mechanisms exist to limit this occurrence. First, proteins are translated into the inactive proenzymes. Later, posttranslational modification of the Golgi cells allows their segregation into the unique subcellular zymogen compartments.

The proenzymes are packaged in a paracrystalline arrangement with protease inhibitors.

Zymogen granules have an acidic pH and a low calcium concentration, which are factors that guard against premature activation until after secretion has occurred and extracellular factors have triggered the activation cascade.

Under various conditions, disruption of these protective mechanisms may occur, resulting in intracellular enzyme activation and pancreatic autodigestion leading to acute pancreatitis.

Acute pancreatitis

Is an inflammatory conditions of the pancrease characterized clinically by abdominal pain and elevated level of pancreatic enzymems in the blood

Pathogenesis of acute pancreatitis

Acute pancreatitis may occur when the factors involved in maintaining cellular homeostasis are out of balance. The initiating event may be anything that injures the acinar cell and impairs the secretion of zymogen granules; examples include alcohol abuse, gallstones, and certain drugs.

At present, it is unclear exactly what pathophysiological event triggers the onset of acute pancreatitis. It is believed, however, that both extracellular factors (e.g., neural and vascular response) and intracellular factors (e.g., intracellular digestive enzyme activation, increased calcium signaling, and heat shock protein activation) play a role. In addition, acute pancreatitis can develop when ductal cell injury leads to delayed or absent enzymatic secretion, as with the *CFTR* gene mutation.

Once a cellular injury pattern has been initiated, cellular membrane trafficking becomes chaotic, with the following deleterious effects: Lysosomal and zymogen granule compartments fuse, enabling activation of trypsinogen to trypsin Intracellular trypsin triggers the entire zymogen activation cascade Secretory vesicles are extruded across the basolateral membrane into the interstitium, where molecular fragments act as chemo attractants for inflammatory cells Activated neutrophils then exacerbate the problem by releasing superoxide (the

respiratory burst) or proteolytic enzymes (cathepsins B, D, and G; collagenase; and elastase).

Finally, macrophages release cytokines that further mediate local (and, in severe cases, systemic) inflammatory responses. The early mediators defined to date are tumor necrosis factor-alpha (TNF-α), interleukin (IL)-6, and IL-8.

These mediators of inflammation cause an increased pancreatic vascular permeability, leading to hemorrhage, edema, and eventually pancreatic necrosis.

As the mediators are excreted into the circulation, systemic complications can arise, such as bacteremia due to gut flora translocation, acute respiratory distress syndrome (ARDS), pleural effusions ,gastrointestinal (GI) hemorrhage, and renal failure.

The systemic inflammatory response syndrome (SIRS) can also develop, leading to the development of systemic shock.

Eventually, the mediators of inflammation can become so overwhelming to the body that hemodynamic instability and death ensue.

In acute pancreatitis, parenchymal edema and peripancreatic fat necrosis occur first; this is known as acute edematous pancreatitis. When necrosis involves the parenchyma, accompanied by hemorrhage and dysfunction of the gland, the inflammation evolves into hemorrhagic or necrotizing pancreatitis.

Pseudocyst and pancreatic abscesses can result from necrotizing pancreatitis because enzymes can be walled off by granulation tissue (pseudocyst formation) or via bacterial seeding of pancreatic or peripancreatic tissue (pancreatic abscess formation).
Li et al compared 2 set of patients with severe acute pancreatitis—one with acute renal failure and the other without it—and determined that a history of renal disease, hypoxemia, and abdominal compartment syndrome are significant risk factors for acute renal failure in patients with severe acute pancreatitis. In addition, patients with acute renal failure were found to have a significantly greater average length of stay in the hospital and in the intensive care unit (ICU), as well as higher rates of pancreatic infection and mortality.[4]

Etiology;

Long-standing alcohol consumption and biliary stone disease cause most of cases of acute pancreatitis, but numerous other etiologies are known.

Biliary tract disease

One of the most common causes of acute pancreatitis in most developed countries representing around 60 % of all cases [2,3] is gallstones passing into the bile duct and temporarily lodging at the

sphincter of Oddi. The risk of a stone causing pancreatitis is inversely proportional to its size.

It is thought that acinar cell injury occurs secondary to increasing pancreatic duct pressures caused by obstructive biliary stones at the ampulla of Vater, although this has not been definitively proven in humans. Occult microlithiasis is probably responsible for most cases of idiopathic acute pancreatitis. as figure 1

Alcohol

Alcohol abuse is a major cause of acute pancreatitis (accounting for at least 35% of cases).[5] On the cellular level, ethanol leads to intracellular accumulation of digestive enzymes and their premature activation and release. On the ductal level, it increases the permeability of ductules, allowing enzymes to reach the parenchyma and cause pancreatic damage.

Ethanol increases the protein content of pancreatic juice and decreases bicarbonate levels and tyrosine inhibitor concentrations. This leads to the formation of protein plugs that block pancreatic outflow.

Figure no . 1

Diagnosis and Management of Acute Pancreatitis by ABILIO MUNOZ, M.D., Austin, Texas, and DAVID A. KATERNDAHL, M.D., M.A., University of Texas Health Science Center, San Antonio, Texas *Am Fam Physician.* 2000 Jul 1;62(1):164-174.

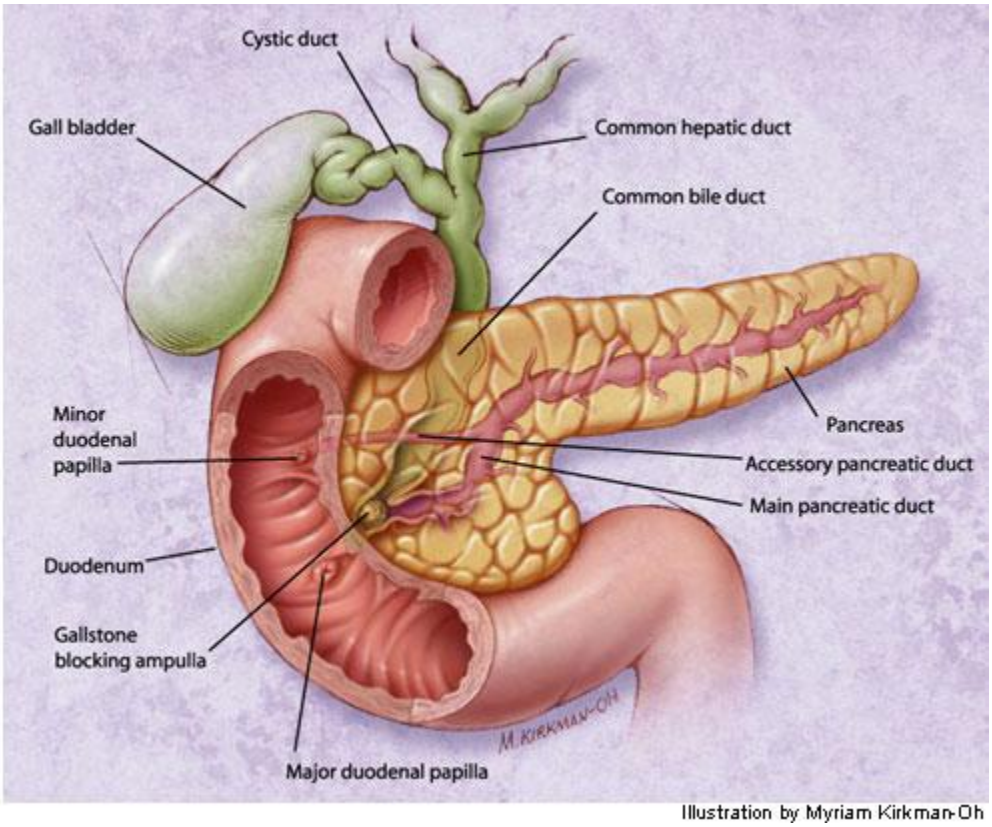


Illustration by Myriam Kirkman-Oh

Most commonly, the disease develops in patients whose alcohol ingestion is habitual over 5-15 years. Alcoholics are usually admitted with an acute exacerbation of chronic pancreatitis.

Occasionally, however, pancreatitis can develop in a patient with

a weekend binging habit, and several case reports have described a sole large alcohol load precipitating a first attack.

Nevertheless, the alcoholic who imbibes routinely remains the rule rather than the exception. Currently, there is no universally accepted explanation for why certain alcoholics are more predisposed to developing acute pancreatitis than other alcoholics who ingest similar quantities are.

Endoscopic retrograde cholangiopancreatography

Pancreatitis occurring after endoscopic retrograde cholangio pancreatco-graphy (ERCP) is probably the third most common type (accounting for approximately 4% of cases). Whereas retrospective surveys indicate that the risk is only 1%, prospective studies have shown the risk to be at least 5%.The risk of post-ERCP acute pancreatitis is increased if the endoscopist is inexperienced, if the patient is thought to have sphincter of Oddi dysfunction, or if manometry is performed on the sphincter of Oddi. No medical measures, with the exception of aggressive preintervention intravenous (IV) hydration, have been durably shown to prevent post-ERCP pancreatitis in randomized studies.

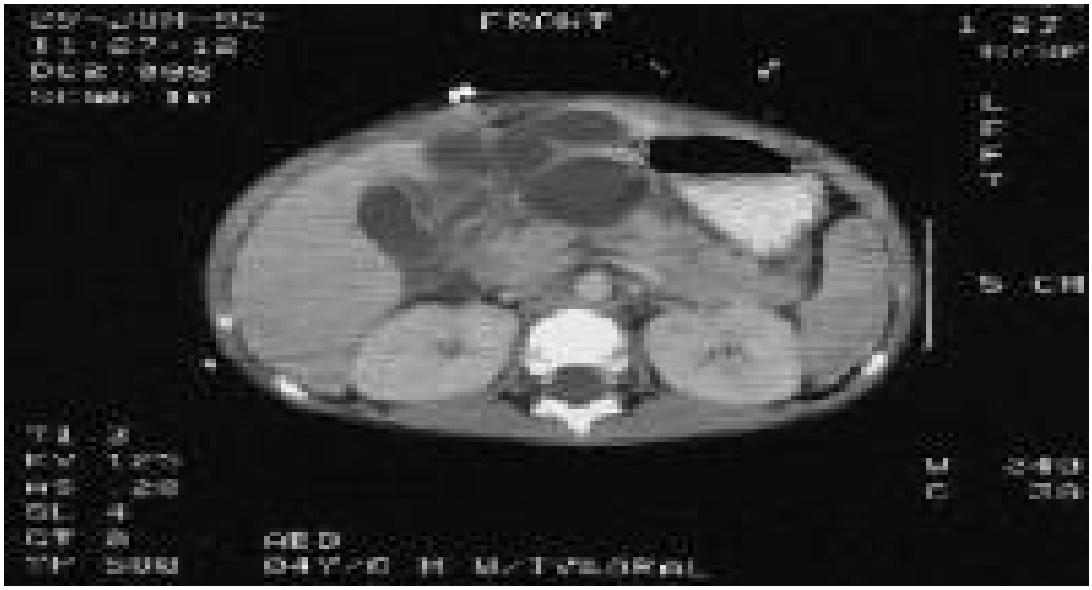
Trauma

Abdominal trauma (approximately 1.5%) causes an elevation of amylase and lipase levels in 17% of cases and clinical pancreatitis in 5% of cases. Pancreatic injury (see the image below) occurs more

often in penetrating injuries (e.g., from knives, bullets) than in blunt abdominal trauma (e.g., from steering wheels, horses, bicycles). Blunt injury to the abdomen or back may crush the gland across the spine, leading to a ductal injury.

Figure no.2

Ref **Pediatric Pancreatitis Workup Author: Andre Hebra, MD; Chief Editor: Carmen Cuffari,** 2005



CT scan of abdomen in a child with traumatic pancreatitis. Fluid collection adjacent to the pancreas will become pseudocyst. and the pancreas is lacerated, nearly cut in half, by force of abdominal trauma. This is the typical location of this injury in relation to vertebral column.

Drugs

Considering the small number of patients who develop pancreatitis compared to the relatively large number who receive potentially toxic drugs, drug-induced pancreatitis is a relatively rare occurrence (accounting for approximately 2% of cases) that is probably related to an unknown predisposition. Fortunately, drug-induced pancreatitis is usually mild.

Drugs definitely associated with acute pancreatitis include the following:

Azathioprin, Sulfonamides, Sulindac ,Tetracycline , Valproic acid, Didanosine ,Methyldopa ,Estrogens ,Furosemide ,6-Mercaptopurine ,Pentamidine ,5-aminosalicylic acid compounds ,Corticosteroids, Octreotide.

Drugs probably associated with acute pancreatitis include the following:

Chlorothiazide and hydrochlorothiazide ,Methandrostenolone (methandienone) ,Metronidazole ,Nitrofurantoin ,Phenformin ,Piroxicam ,Procainamide ,Colaspase ,Chlorthalidone ,Combination cancer chemotherapy drugs (especially asparaginase) ,Cimetidine, Cisplatin ,Cytosine arabinoside ,Diphenoxylate ,Ethacrynic acid.

In addition, there are many drugs that have been reported to cause acute pancreatitis in isolated or sporadic cases.

Less common causes

The following causes each account for less than 1% of cases of pancreatitis.

Infection :

Several infectious diseases may cause pancreatitis, especially in children. These cases of acute pancreatitis tend to be milder than cases of acute biliary or alcohol-induced pancreatitis.

Viral causes include mumps virus, coxsackievirus, cytomegalovirus (CMV), hepatitis virus, Epstein-Barr virus (EBV), echovirus, varicella-zoster virus (VZV), measles virus, and rubella virus.

Bacterial causes include *Mycoplasma pneumoniae, Salmonella, Campylobacter,* and *Mycobacterium tuberculosis.* Worldwide, *Ascaris* is a recognized cause of pancreatitis resulting from the migration of worms in and out of the duodenal papillae.

Pancreatitis has been associated with AIDS; however, this may be the result of opportunistic infections, neoplasms, lipodystrophy, or drug therapies.

Hereditary pancreatitis :

Hereditary pancreatitis is an autosomal dominant gain-of-function disorder related to mutations of the cationic trypsinogen gene (*PRSS1*), which has an 80% penetrance. Mutations in this gene cause premature activation of trypsinogen to trypsin.

In addition, the *CFTR* mutation plays a role in predisposing patients to acute pancreatitis by causing abnormalities of ductal secretion. At present, however, the phenotypic variability of patients with the *CFTR* mutation is not well understood. Certainly, patients homozygous for the *CFTR* mutation are at risk for pancreatic disease, but it is not yet clear which of the more than 800 mutations carries the most significant risk. In addition, the role of CFTR heterozygotes in pancreatic disease is unknown.

Mutations in the SPINK1 protein, which blocks the active binding site of trypsin, rendering it inactive, also probably play a role in causing a predisposition toward acute pancreatitis.

This probably explains the predisposition, rather than the cause, of acute pancreatitis in these patients. If enough mutant enzymes become activated intracellularly, they can overwhelm the first line of defense (i.e., pancreatic Secretory trypsin inhibitor) and resist backup defenses (i.e., proteolytic degradation by mesotrypsin, enzyme Y, and trypsin itself). Activated mutant cationic trypsin can then trigger the entire zymogen activation cascade.

***Hypercalcaemia* :**

Hypercalcaemia from any cause can lead to acute pancreatitis. Causes include hyperparathyroidism, excessive doses of vitamin D, familial hypocalciuric hypocalcaemia, and total parenteral nutrition (TPN). Routine use of automated serum chemistries has allowed

earlier detection and reduced the frequency of hypocalcaemia manifesting as pancreatitis.

Developmental abnormalities of pancreas :

The pancreas develops from 2 buds stemming from the alimentary tract of the developing embryo. There are 2 developmental abnormalities commonly associated with pancreatitis: pancreas divisum and annular pancreas.

Pancreas divisum

Is a failure of the dorsal and ventral pancreatic ducts

To fuse during embryogenesis. Probably a variant of normal anatomy, it occurs in approximately 5% of the population in most cases, it may actually protect against gallstone pancreatitis. It appears that the presence of stenotic minor papillae and an atretic duct of Santorini are additional risk factors that together contribute to the development of acute pancreatitis through an obstructive mechanism (although this is controversial) as figure 3

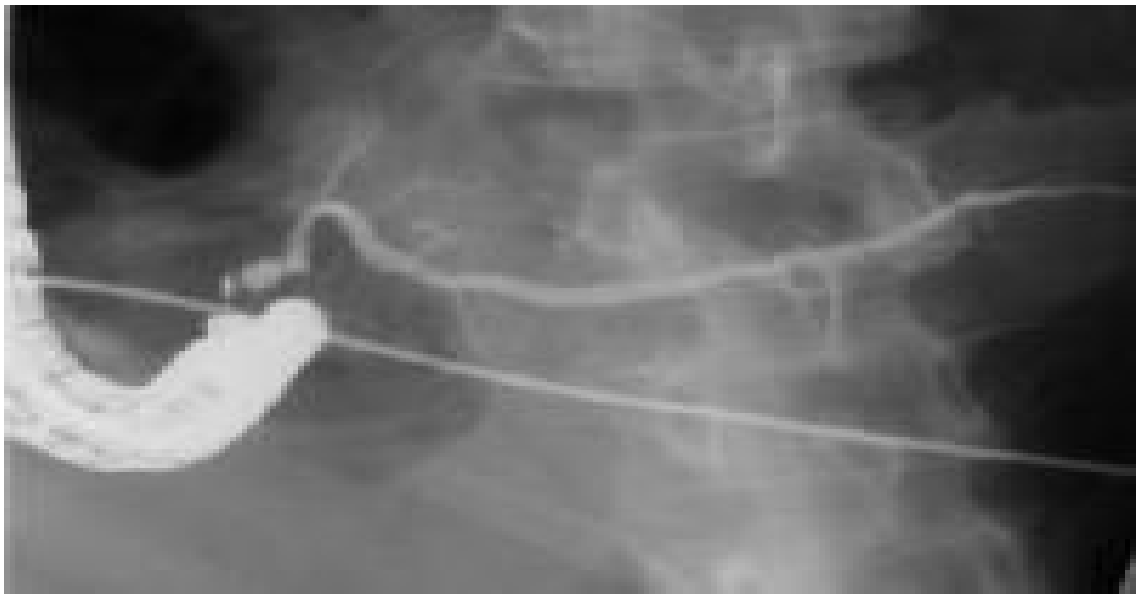
Annular pancreas

Is an uncommon congenital anomaly in which a band of pancreatic tissue surrounds the second part of the duodenum. Usually, it does not cause symptoms until later in life. This condition is a rare cause of acute pancreatitis, probably through an obstructive mechanism. Sphincter of Oddi dysfunction can lead to acute pancreatitis by causing increased pancreatic ductal pressures. However, the role of

pancreatitis induced by such dysfunction in patients without elevated sphincter pressures on manometry remains controversial.

Figure no. 3

References Acute Pancreatitis Workup Author: Timothy B Gardner, MD; Chief Editor: BS Anand, 2005



Pancreas divisum associated with minor papilla stenosis causing recurrent pancreatitis. Because pancreas divisum is relatively common in general population, it is best regarded as variant of normal anatomy and not necessarily as cause of pancreatitis. In this case, note bulbous contour of duct adjacent to cannula. This appearance has been termed Santorinicele. Dorsal duct outflow obstruction is a probable cause of pancreatitis when Santorinicele is present and associated with a minor papilla that accommodates only

Hypertriglyceridemia :

Clinically significant pancreatitis usually does not occur until a person's serum triglyceride level reaches 1000 mg/dL. It is associated with type I and type V hyperlipidemia. Although this view is somewhat controversial, most authorities believe that the association is caused by the underlying derangement in lipid metabolism rather than by pancreatitis causing hyperlipidemia.

This type of pancreatitis tends to be more severe than alcohol- or gallstone-induced disease.

Tumors :

Obstruction of the pancreatic ductal system by a pancreatic ductal carcinoma, ampullary carcinoma, islet cell tumor, solid pseudo tumor of the pancreas, sarcoma, lymphoma, cholangiocarcinoma, or metastatic tumor can cause acute pancreatitis. The chance of pancreatitis occurring when a tumor is present is approximately 14%. Pancreatic cystic neoplasm, such as intraductal papillary-mucinous neoplasm (IPMN), mucinous cystadenoma, or serous cystadenoma, can also cause pancreatitis.

Toxins

Exposure to organophosphate insecticide can cause acute pancreatitis. Scorpion and snake bites may also be causative; in Trinidad, the sting of the scorpion *Tityus trinitatis* is the most common

cause of acute pancreatitis. Hyper stimulation of pancreas exocrine secretion appears to be the mechanism of action in both instances.

Surgical procedures :

Acute pancreatitis may occur in the postoperative period of various surgical procedures (e.g., abdominal or cardiopulmonary bypass surgery, which may insult the gland by causing ischemia). Postoperative acute pancreatitis is often a difficult diagnosis to confirm, and it has a higher complication rate than pancreatitis associated with other etiologies. The mechanism is unclear.

Vascular abnormalities :

Vascular factors, such as ischemia or vasculitis, can play a role in causing acute pancreatitis. Vasculitis can predispose patients to pancreatic ischemia, especially in those with polyarteritis nodosa and systemic lupus erythematosus.

Autoimmune pancreatitis:

Autoimmune pancreatitis, a relatively newly described entity, is an extremely rare cause of acute pancreatitis (prevalence, 0.82 per 100,000 individuals). When it does cause acute pancreatitis, it is usually in young people (approximately 40 years) who also suffer from inflammatory bowel disease. The pathogenesis is unclear.

Epidemiology

The reported annual incidence of acute pancreatitis has ranged from 4.9 to 35 per 100,000 population [6] . Acute pancreatitis is the leading gastrointestinal cause of hospitalization in the United States [7]. The incidence of acute pancreatitis is increasing in many European and Scandinavian countries due to increased alcohol consumption and better diagnostic capability [7]. In a retrospective study from the Netherlands, the observed incidence of acute pancreatitis increased by 28 percent between 1985 and 1995.

Advances in diagnostic and therapeutic interventions have led to a decrease in mortality from acute pancreatitis, especially in those with severe, often necrotizing pancreatitis. Mortality in acute pancreatitis is usually due to systemic inflammatory response syndrome and organ failure in the first two-week period, while after two weeks it is usually due to sepsis and its complications [8,9]

Age-related demographics

The median age at onset depends on the etiology.[13]
Hospitalization rates increase with age. For people aged 35-75 years, the rate doubles for males and quadruples for females.

Sex-related demographics

Generally, acute pancreatitis affects males more often than females. In males, the etiology is more often related to alcohol; in females, it is more often related to biliary tract disease.

Idiopathic pancreatitis has no clear predilection for either sex.

Race-related demographics

The hospitalization rates of patients with acute pancreatitis per 100,000 population are 3 times higher for blacks than whites. These racial differences are more pronounced for males than females. The risk for African Americans aged 35-64 years is 10 times higher than for any other group. African Americans are at higher risk than whites in that same age group.

The annual incidence of acute pancreatitis in Native Americans is 4 per 100,000 population; in whites, 5.7 per 100,000 population; and in blacks, 20.7 per 100,000 population.[14]

The following are median ages of onset for various etiologies:

Table no. 1

cause	Age related
Alcohol	39 years
Biliary tract–related	69 years
Trauma	66 years
Drug	42 years
ERCP	58 years
AIDS	31 years
Vasculitis	36 years

Laboratory diagnosis

Amylase:

Serum amylase levels in patients with pancreatitis vary depending on the severity of the disease. On average, during uncomplicated cases, the serum amylase level starts increasing from two to 12 hours after the onset of symptoms and peaks at 12 to 72 hours.[15,16] It usually returns to normal within one week.[17,18] Although it lacks sensitivity (75 to 92 percent) and specificity (20 to 60 percent), measurement of the serum amylase level is the most widely used method of diagnosing pancreatitis. The advantages of amylase testing are that it is quickly

performed, easily obtained and inexpensive.[19] However, a variety of non pancreatic conditions cause increased amylase levels.[18]

Lipase:

Lipase levels increase within four to eight hours of the onset of clinical symptoms and peak at about 24 hours. Levels decrease within eight to 14 days. The specificity (50 to 99 percent) and sensitivity (86 to 100 percent) of lipase measurements are better than those of amylase measurement, particularly in detecting alcoholic pancreatitis.[19]The specificity of lipase measurement, as well as amylase measurement, may be improved by raising the threshold to at least three times the upper limit of the normal reference values.[20]

Trypsin/Elastase:

Based on median sensitivities and specificities, an elevated trypsin level has a better likelihood ratio for detecting pancreatitis than the amylase level and is probably the most accurate serum indicator for acute pancreatitis.[21]The elastase level has not proved to be better than trypsin or lipase levels in assisting the diagnosis of acute pancreatitis. However, a serum trypsin assay is not widely available and therefore is not routinely used.

Hepatic Function Studies:

Hepatic transaminase levels may be elevated in patients with pancreatitis caused by alcohol abuse or cholelithiasis with obstruction.

However, these tests are not sufficiently reliable for diagnosing acute biliary pancreatitis or determining its etiology.

Radiologic Studies

Plain Radiographs:

Plain radiographs may support the diagnosis of acute pancreatitis when certain findings are present[11]. Of these findings, a gas-filled duodenum (sentinel loop) secondary to obstruction is the most specific for pancreatitis.[16]However, none of the radiologic abnormalities on plain films can be used for specific diagnostic purposes.

Ultrasonography:

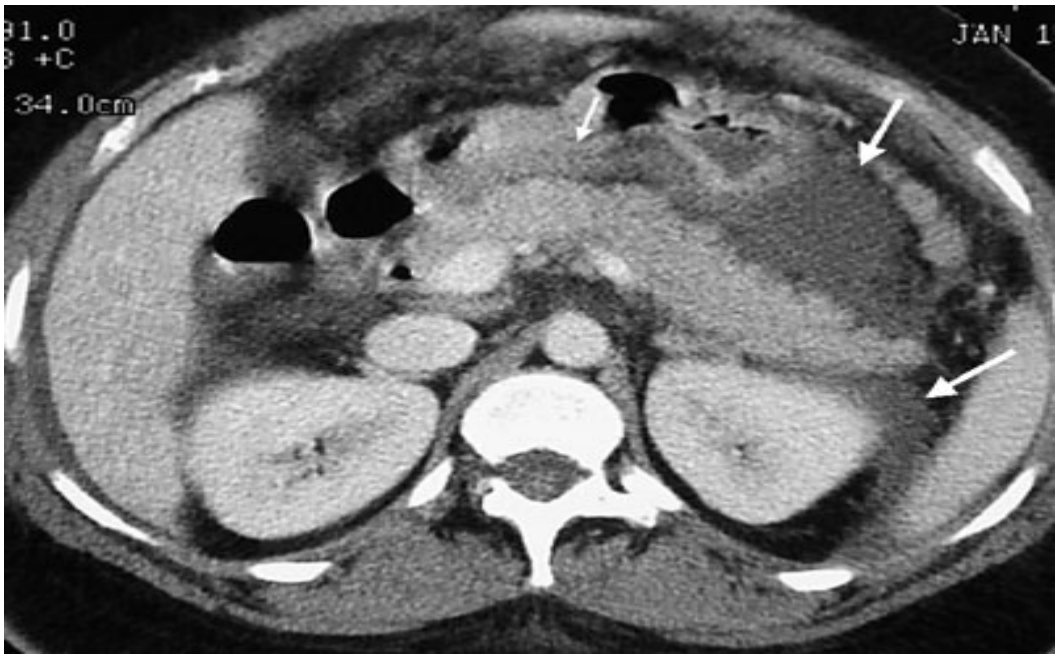
Ultrasonography is an acceptable study for initial evaluation when biliary causes are suspected. Pancreatic ultrasonography has these advantages: it is noninvasive, relatively inexpensive and may be performed at the bedside. The sensitivity of this study in detecting pancreatitis is 62 to 95 %.[22,23] However, in 35 % of cases, the pancreas is obscured secondary to bowel gas.[19]

Computed Tomography (CT):

The contrast-enhanced CT scan provides the best imaging of the pancreas and surrounding structures. A CT study may be useful when other diagnostic studies are inconclusive, when the patient has severe symptoms, when fever is present or in the face of persistent

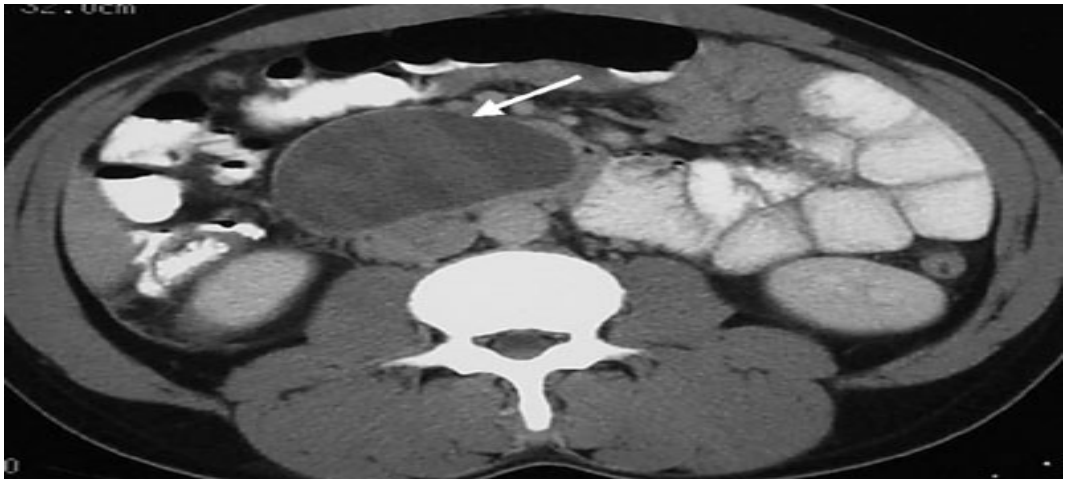
leukocytosis that suggests secondary infection.[19] in addition, CT scanning is especially helpful in assessing complications related to acute pancreatitis or as a follow-up study in patients who are clinically deteriorating. The CT findings in pancreatitis may show inflammation characterized by diffuse or segmental enlargement of the pancreas, with irregular contour and obliteration of peripancreatic fat, necrosis or a pseudo cyst.

Figure no. 4



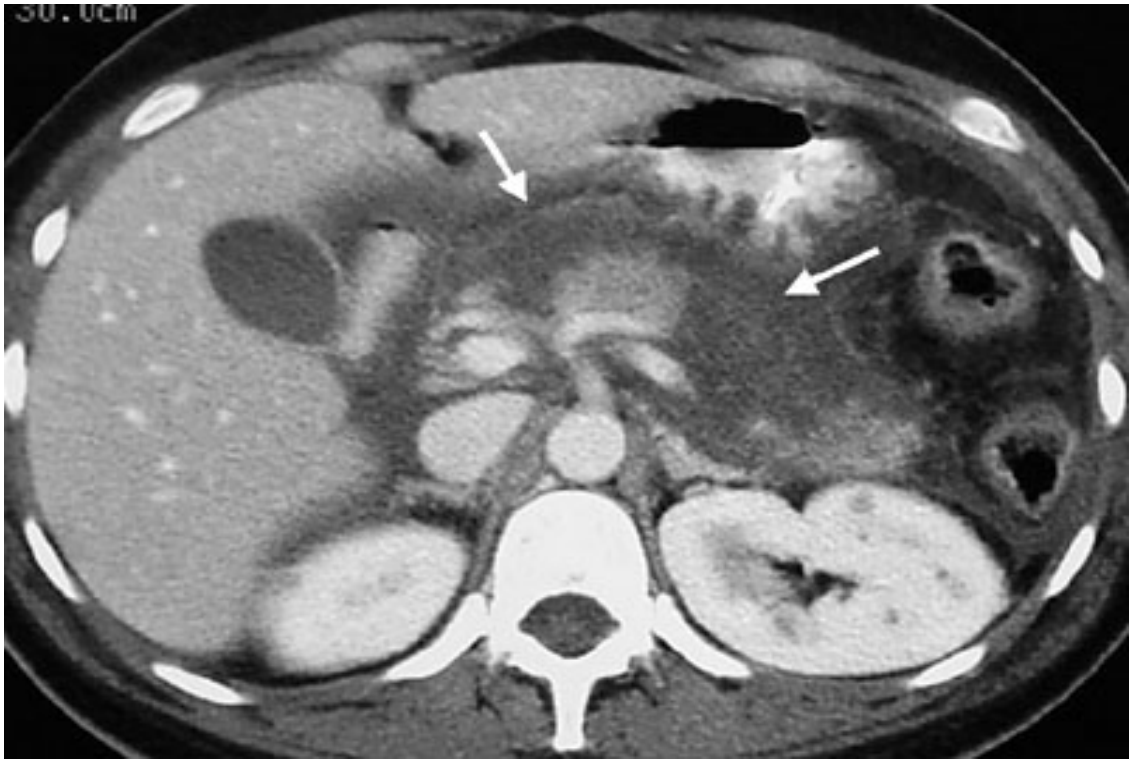
Contrast-enhanced axial computed tomographic section of the upper abdomen showing peripancreatic and retroperitoneal edema (large arrows) and stranding. The pancreas itself (small arrow) appears relatively normal.

figure no. 5



Contrast-enhanced axial computed tomographic section of the upper abdomen showing a well-defined fluid collection in the retro peritoneum (arrow) just below the level of the pancreas

Figure no. 6



Contrast-enhanced axial computed tomographic section of the upper abdomen showing peripancreatic and retroperitoneal edema. Large non-enhancing areas of necrosis are visible in the body and neck of the pancreas (arrows).

Ref. of figure 4,5,6 Balthazar EJ, Freeny PC, van Sonnenberg E. Imaging and intervention in acute pancreatitis. Radiology. 1994;193:297–306.

Endoscopic Retrograde Cholangiopancreatography (ERCP).

ERCP has a limited role in management of acute pancreatitis. It is primarily indicated in patients with severe disease who are suspected of having biliary obstruction this procedure is sometimes done to enable endoscopic sphincterotomy and remove impacted stones.[25] The risks of performing ERCP with sphincterotomy include precipitating an acute episode of pancreatitis, introducing infection and causing hemorrhage and perforation. At least one study has shown that patients with severe biliary pancreatitis show a reduction in morbidity and mortality with early (less than 24 hours) ERCP.[26]

Clinical Outcomes and Predictive Value of Prognostic Scoring Systems for Acute Pancreatitis

table no.2

Prognostic scoring system	Associated outcome	Positive LR	Negative LR
APACHE II score>8 at 24hrs	Need for intensive care unit, infection, pancreatic necrosis, mortality, organ failure and longer hospital stay	1.7 to 4	0.25
Imrie score>3	Mortilty, severty, pancreatic fluid collection	2.4	0.36
Ranson's criteria score>3 at 48 hrs	Major complications, severity, organ failure, pancreatic necrosis, mortality, long hospital stay	2.4 to 2.5	0.47

LR = likelihood ratio; APACHE II = Acute Physiology and Chronic Health Evaluation. [27,29]

CT grade

Table no. 3

A	Normal Pancreas	0 points
B	Edematous Pancreas	1 point
C	B plus mild extra pancreatic changes	2 points
D	Severe Extra Pancreatic Changes Plus One Fluid Collection	3 points
E	Multiple Or Extensive Fluid Collections	4 points

Necrosis score:

Table no.4

Scoring: CT grade + necrosis score

None	(0 points)
< 1/3	(2 points)
>1/3but <1/2	(4 points)
> 1/2	(6 points)

Imrie scoring system

table no .5

criterion met 48 hours after admission
Age > 55 years
White blood cell count > 15,000 per mm3 (15.0 × 109 per L)
Blood glucose > 180 mg per dL (10 mmol per L) in patients without diabetes
Serum lactate dehydrogenase > 600 U per L
Serum AST or ALT > 100 U per L
Serum calcium < 8 mg per Dl
PaO2 < 60 mm Hg
Serum albumin < 3.2 g per dL (32 g per L)
Serum urea > 45 mg per dL (16.0 mmol per L)

Scoring: One point for each criterion met 48 hours after admission

Ranson's criteria

Table no. 6

Scoring: One point for each criterion met

At admission or diagnosis	During initial 48 hours
Age > 55 years	Hematocrit decrease > 10%
White blood cell count > 16,000 per mm3	Blood urea nitrogen increase > 5 mg per dL
Blood glucose > 200 mg per dL	Serum calcium < 8 mg per dL (2 mmol per L
Serum lactate dehydrogenase > 350 U per L	Base deficit > 4 mmol per L (4 mEq per L)
AST > 250 U per L	Fluid sequestration > 6,000 mL
	PaO2 < 60 mm Hg

APACHE II = Acute Physiology and Chronic Health Evaluation; PaO2 = partial arterial oxygen tension; CT = computed tomography; AST = aspartate transaminase; ALT = alanine transaminase 20,23,24,25.

Chapter 2

Aim

Aim of the study:

The aim of this study is to review epidemiological and clinical characteristic of the Acute pancreatitis over a period of two years 1st of Jan. 2007 up to-31 of Dec. 2008 , department of general surgery , Aljala- Hospital ,Benghazi

Chapter 3

Methods
and subject

Subjects and methods

All cases included in this study are from the medical records of the Department of surgery , Aljala Hospital . From 1st of Jan.2007 to 31st dec.2008

Data Collection

All files of patients diagnosed having acute pancreatitis during two years period were included in the study , all information in the files were retrospectively analyzed , age, sex , symptoms of epigatric pain, vomiting ,fever, and part of causes like history of GBD or post surgical chloecystectomy or ERCP, associated chronic illness like diabetes hypertension or hyperlipidemia also collecting

data about investigation that available in our hospital and files: CBC ,WBC, serum and urine amylase, serum calcium ,urea ,blood suger, LDH and alkline phoshpatase and imaging like erect abdominal X ray US abdomen CT scan and way of management like to keep our patient NPO ,NGT ICU admission according to severity of vital sign monitoring and number of days was staying every patient in both ICU and hospital and the complication of diseased patient and final outcome.

Type of the study:

Descriptive retrospective study **.**

Chapter 4

results

Results

All data that mention in methods section analyzed them by using spss for statistical analysis

We found the no. of male and female ration in this study are equal 50% in each as the table no. 7 ,and about 48% of this group study affected age between 40 to 60 yrs and also 40% of age between 20 to 40yrs and only few nos. in extreme age as figure no 8 .

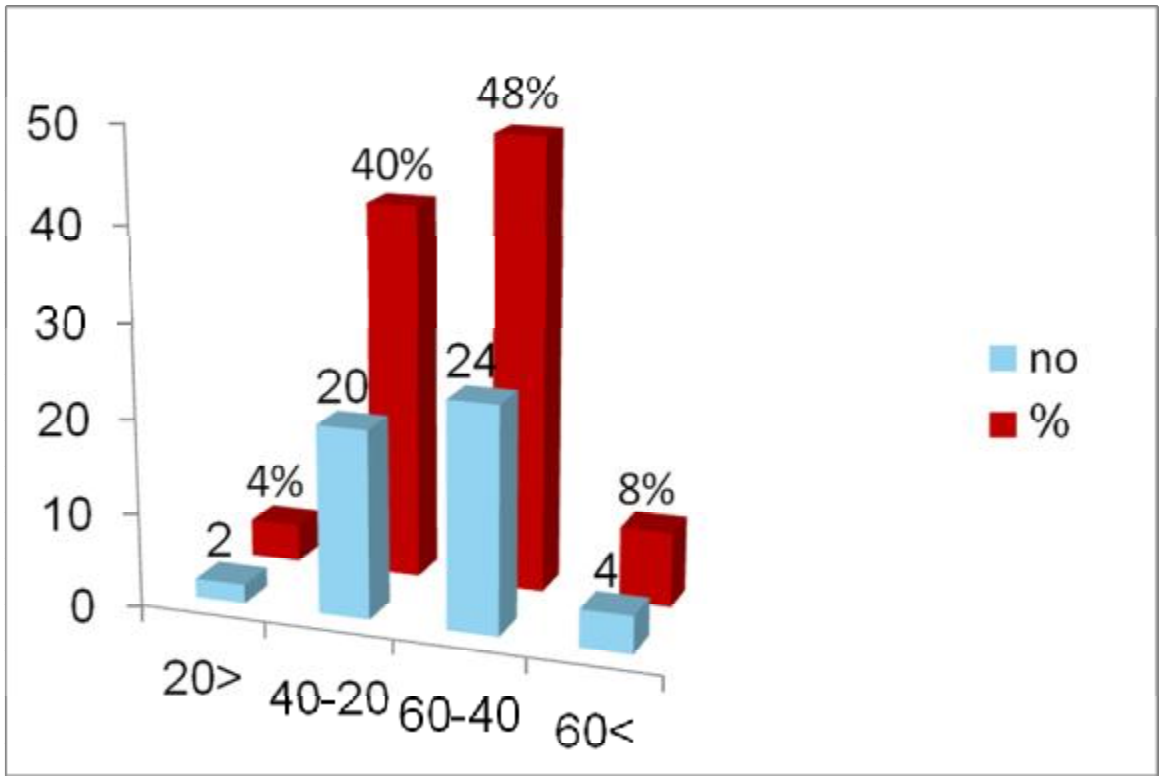
Table no. 7

Distribution of patients according to the sex.

Sex	No.	%
Male	25	50
Female	25	50
Total	50	100

Figure no.7

Distribution of patients according to the age.



And we found in this study the affected group represented by typical history of abdominal pain in epigastrium which is sever radiating back with allover guarding and vomiting with elevated serum and urine amylase which is about 96% as showing figure no. 8

We found only 20% of patients are seeking the medical places in first 24 hrs from symptoms developed and 22% that comes to hospital between 2-7 days from developed the symptoms as figure no. 9

figure no.8

Distribution of patients according to symptoms.

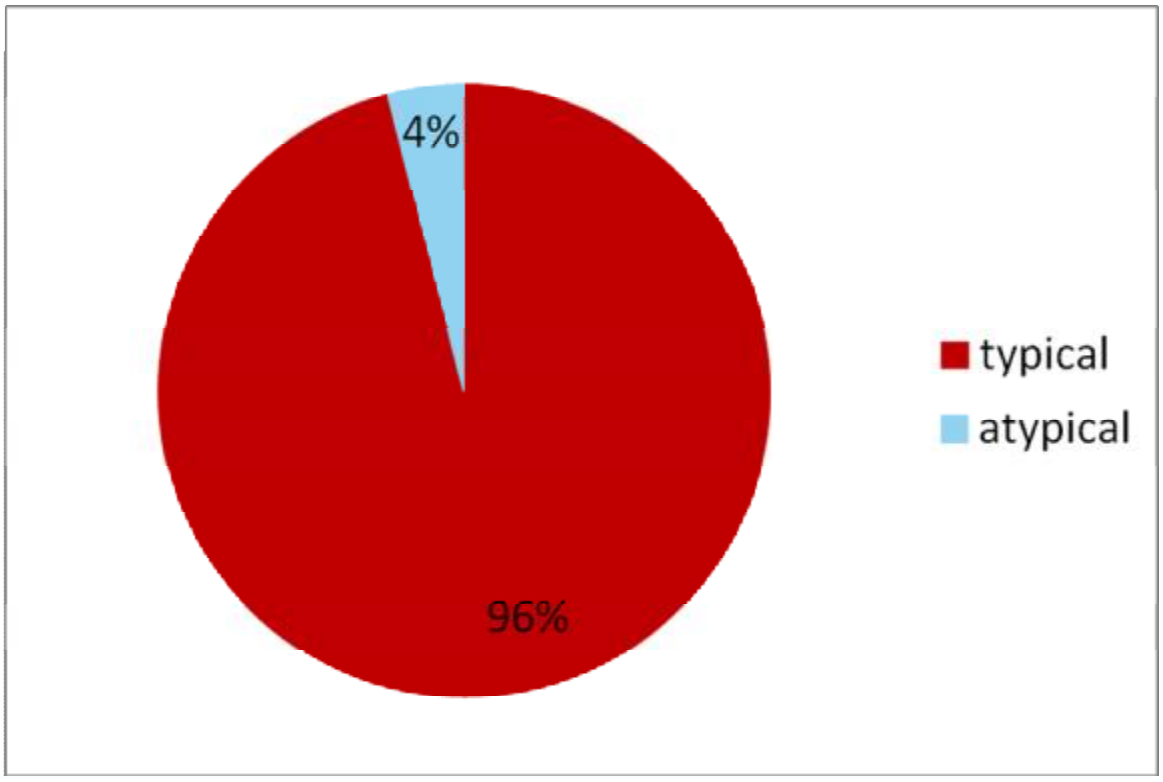


Fig. 8 :Distribution of patients according to symptoms.

Fever which is one of symptoms with tachycardia which indicate the severity of the disease only 34% and 36% represented respectively in this study group as table no. 8 and 9.

Figure no. 9

Distribution of patients according to duration of symptoms.

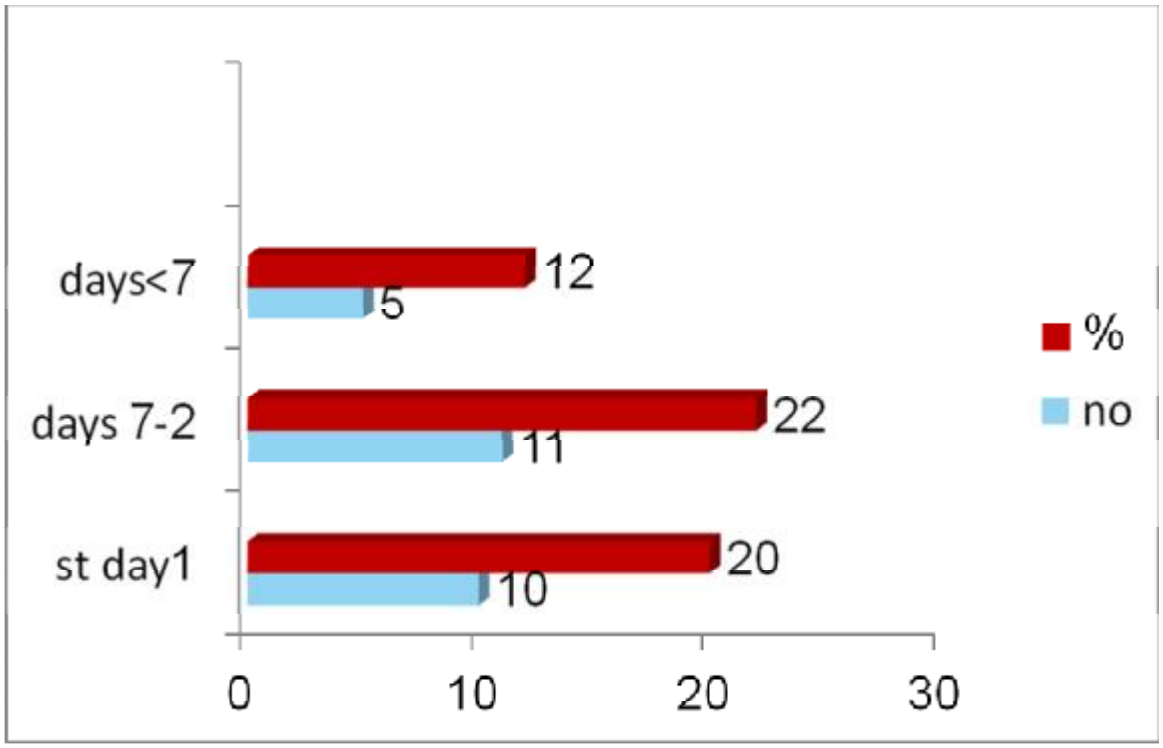


Fig.9 :Distribution of patients according to duration of symptoms.

Table no. 8

Distribution of patients according to presence of fever

Presence of fever	No.	%
yes	17	34
No	33	66
Total	50	100

Table no. 9

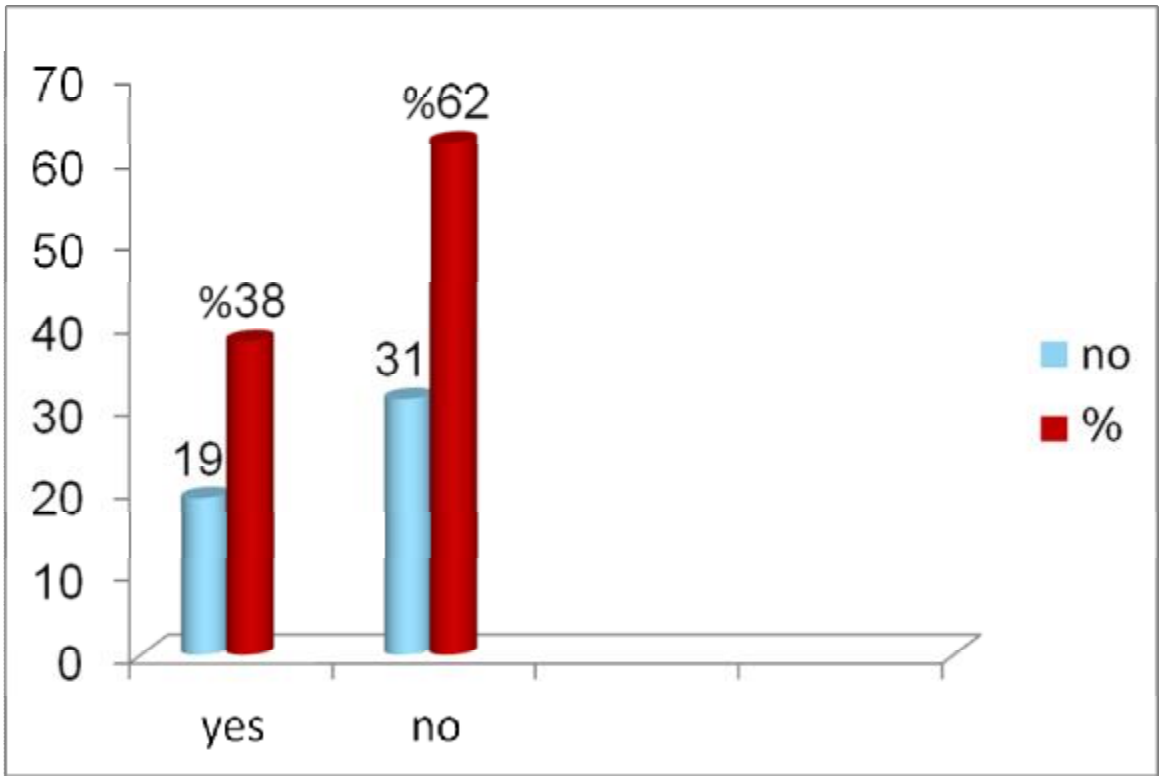
Distribution of patients according to presence of tachycardia.

Presence of tachycardia	No.	%
Yes	18	36
No	32	64
Total	**50**	**100**

Also as apart oh history of chronic diseases like diabetes, hypertension and hyperlipidemia we found 20% ,14%, and 4% respectively and total represented as 38% of chronic illness

figure no. 10

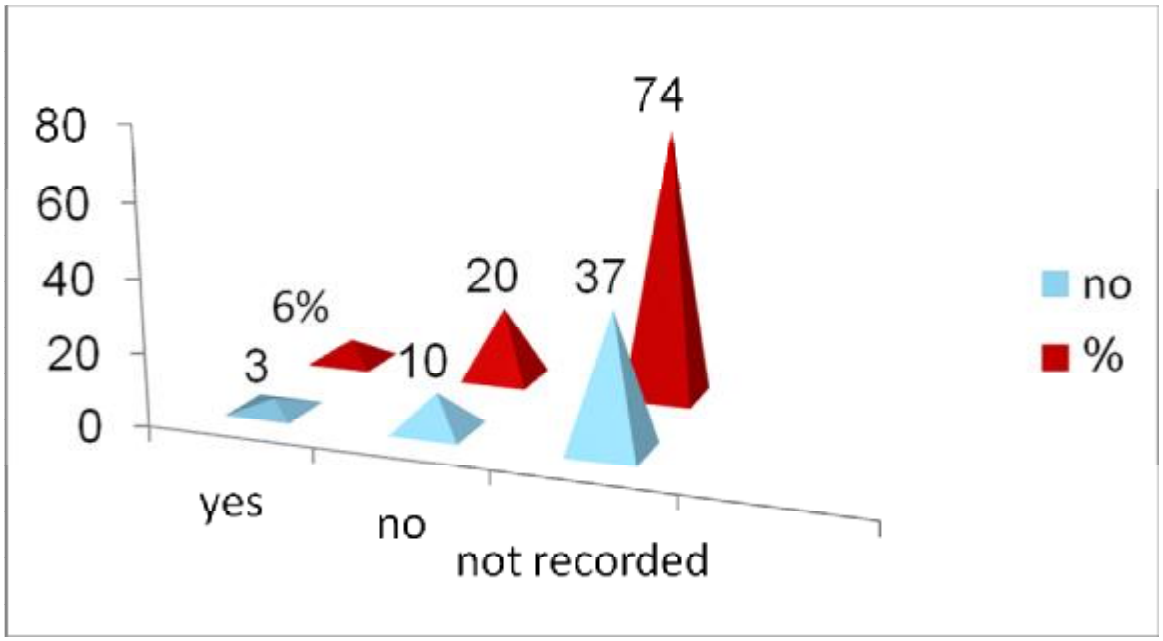
Distribution of patients according to history of chronic disease.



Also we found only 3 patients who able to said about alcohol Consumption where is about 44% not known about them

Figure no.11

Distribution of patients according to consumption of alcohol



The patient that given the positive surgical history is about 30% between cholecystectomy and spleenectomy while patients have positive gall bladder stones proved by US is about 48%

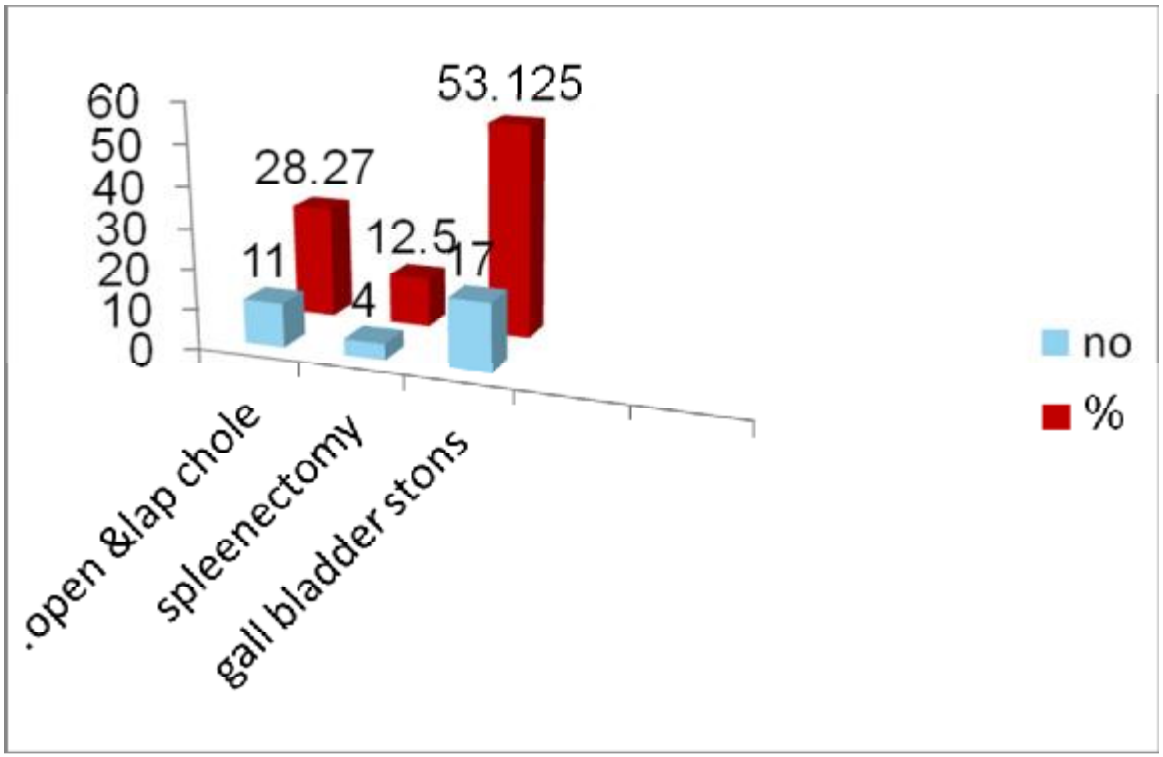
Table no. 10

Distribution of patients according to surgical history.

Surgical history	No.	%
yes	15	30
No	35	70
Total	50	100

Figure no. 12

Distribution of patients according to type of operation .



By examination we found only 3 patients have low blood pressure and about 94% have typical finding of acute pancreatitis as epigatic tenderness abdominal guarding and distention

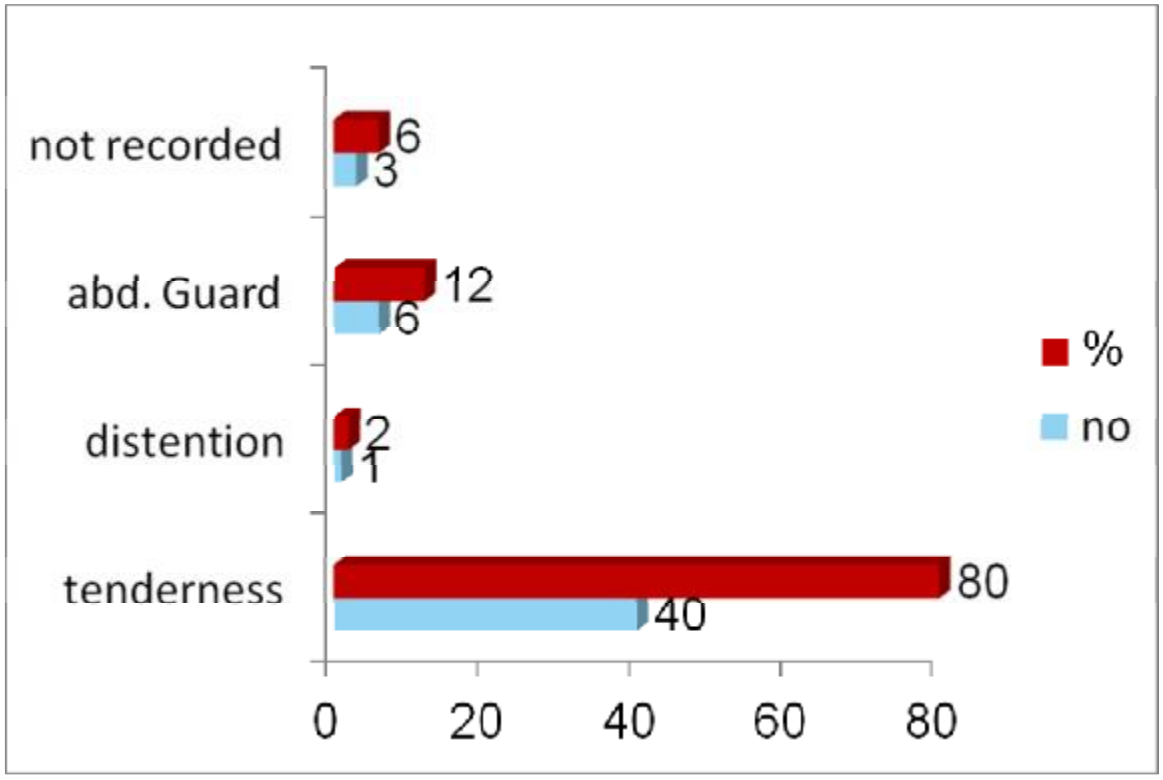
Table no. 11

Blood pressure status in patients.

Blood pressure status	No.	%
Normal	47	94
Low	3	6
Total	50	100

Figure no. 13

Finding in abdominal examination



We start with investigation which are taken places as base line investigation , supportive diagnostic like serum and urine amylase ,part of follow up patient to improve with clinical data like white cell count ,US finding, also part of prognosis and total fait ,

Starting with base line investigation.

Figure no. 14

Distribution of patients according to hemoglobin level in male.

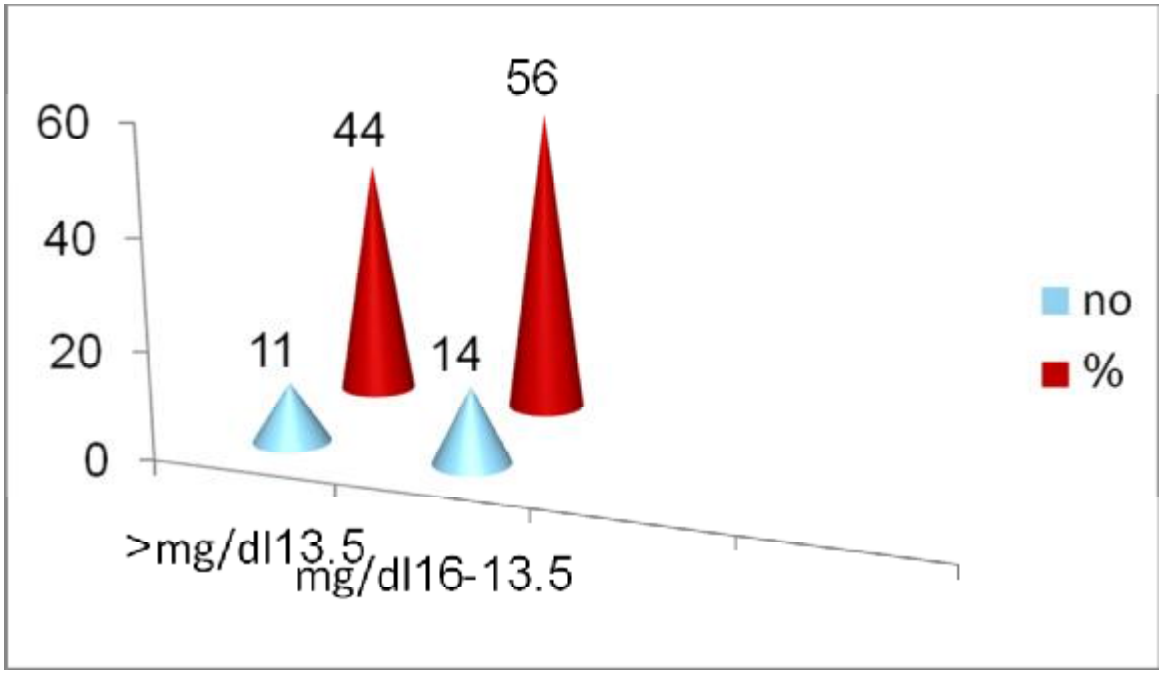
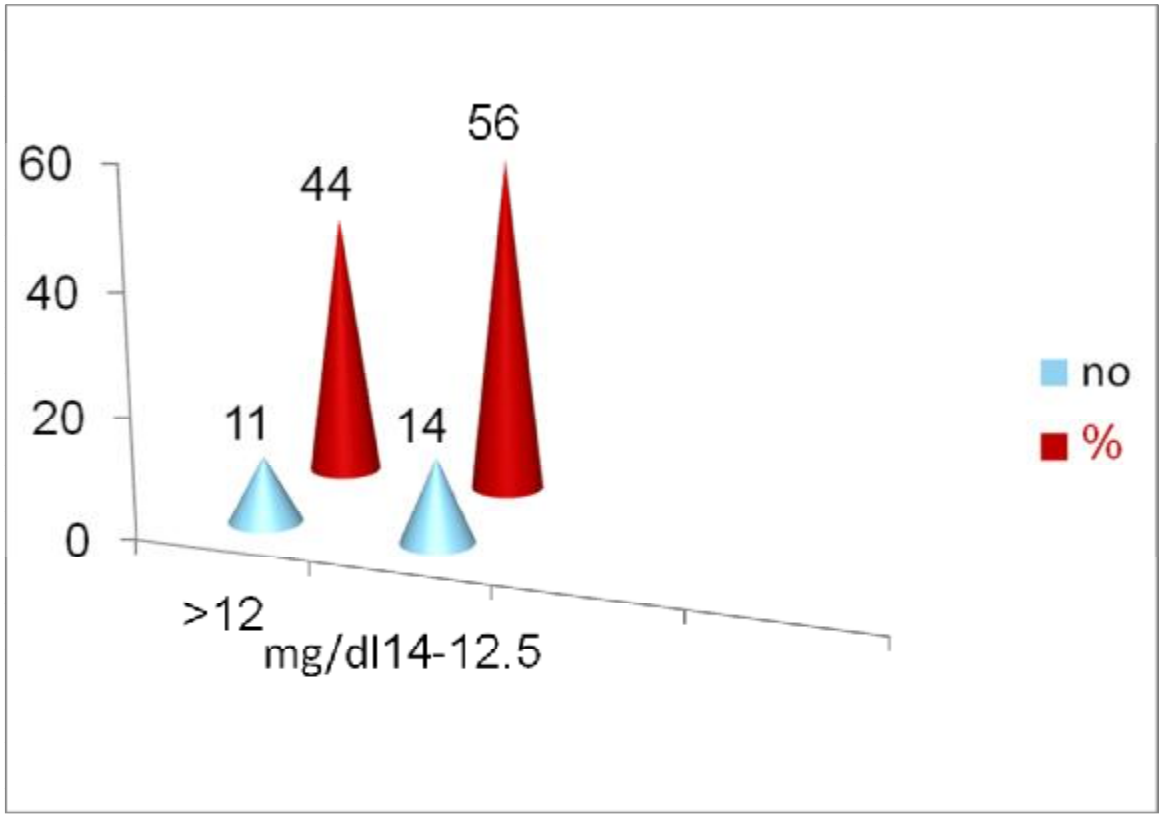


Figure no 15

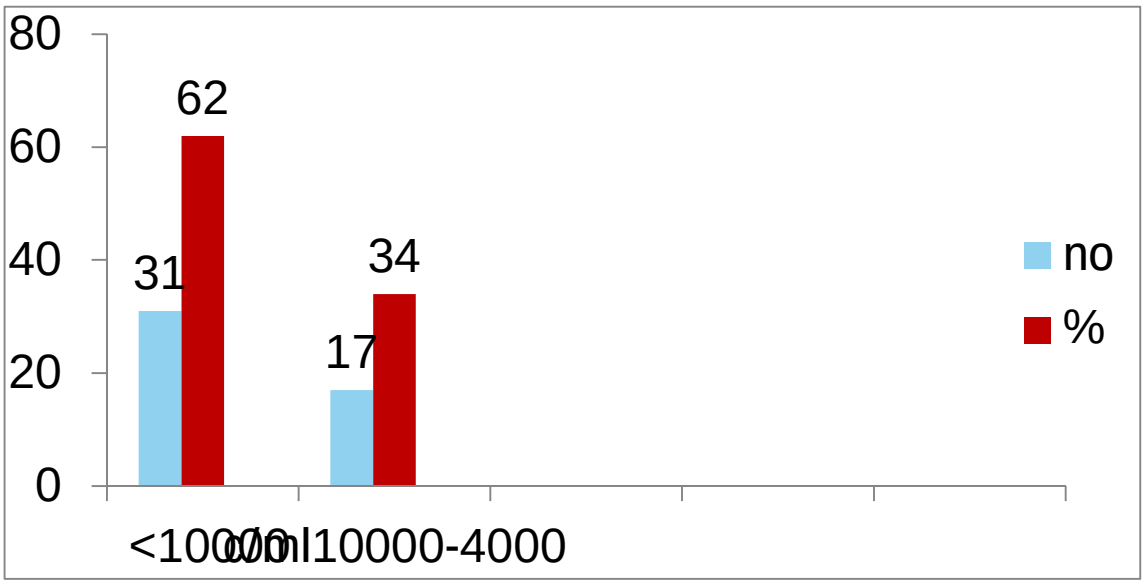
Distribution of patients according to hemoglobin level in female.



White blood cell in about 62% is high than normal range 4-11x10^3/cc

Figure no 16

Distribution of patients according to WBC.



We used the serum and urine amylase as indicator of acute pancreatitis especially when found it very high among normal range 30 -170 we found about 60% 0f patient has high reading of serum amylase>2000iu as urine amylase 60%geeting high level >1100iu as we know both investigation are not specific but they are sensitive

Figure no. 17

Distribution of patients according to level of serum amylase .

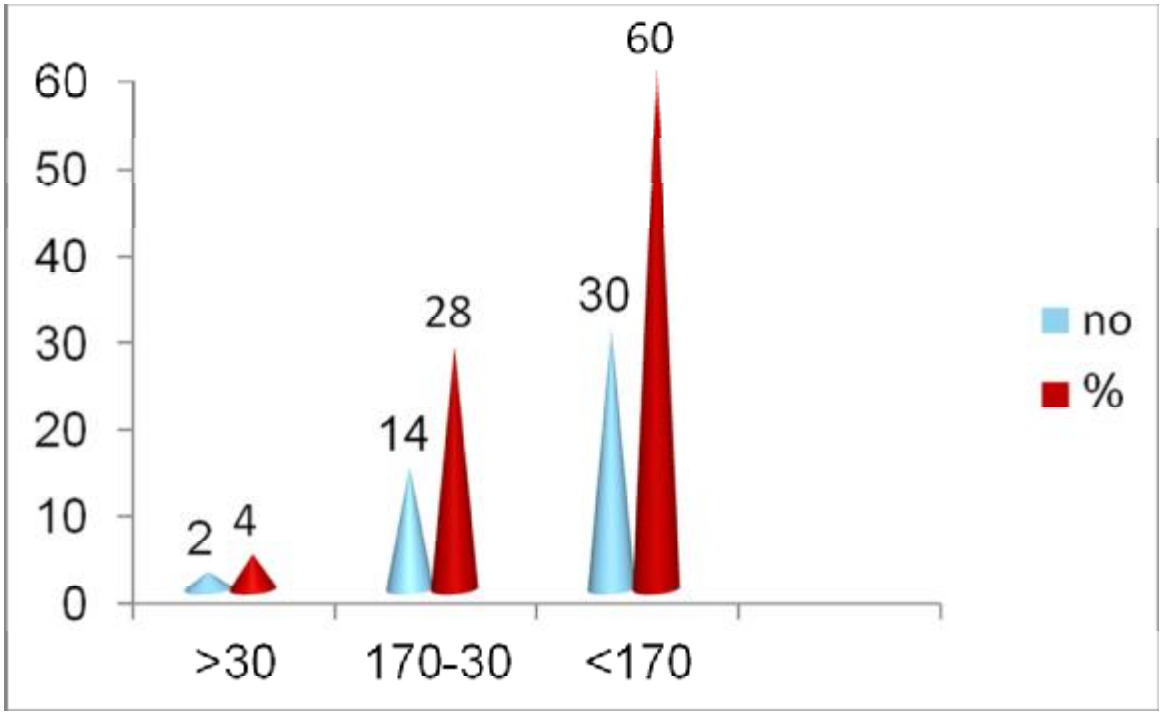
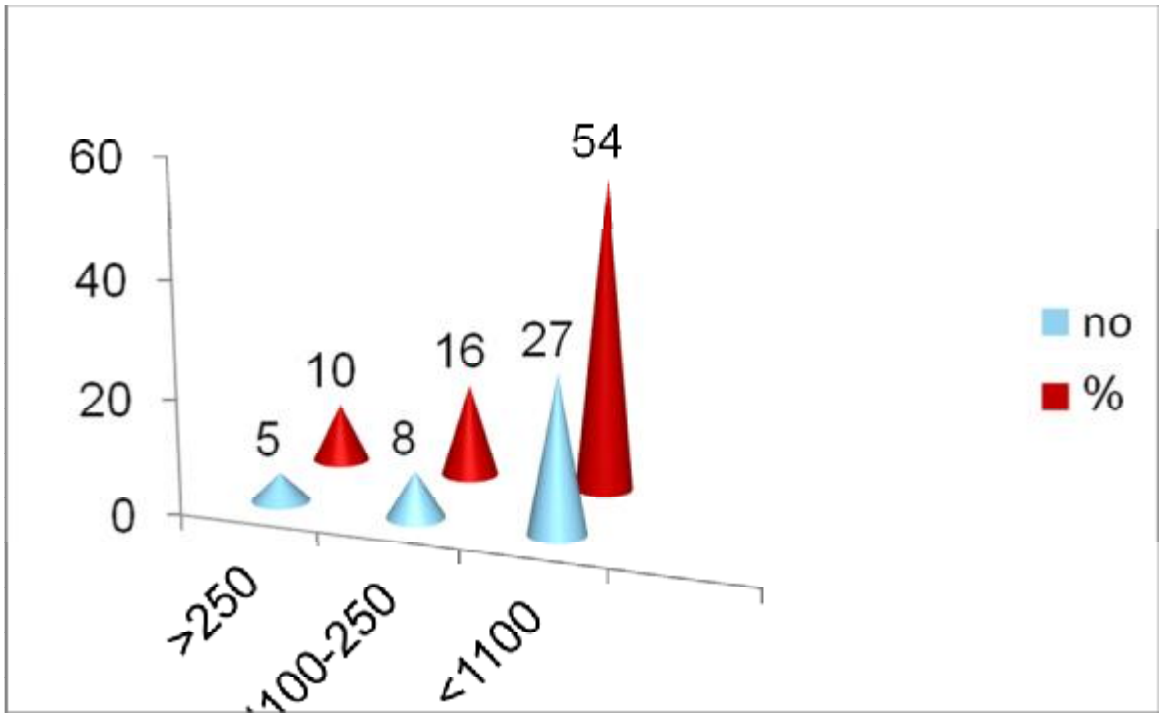


Figure no.18

Distribution of patients according to level of urine amylase



Serum level of calcium, blood sugar , urea ,lactate dehydrogenase and alkaline phosphates about 34% of patient had low serum ca than normal range while blood sugar and urea had no significant high level in this study group but lactate dehydrogenase and alkaline phosphates 26%and 52% elevation in this group study respectively than normal range

Figure no.19

Distribution of patients according to calcium level .

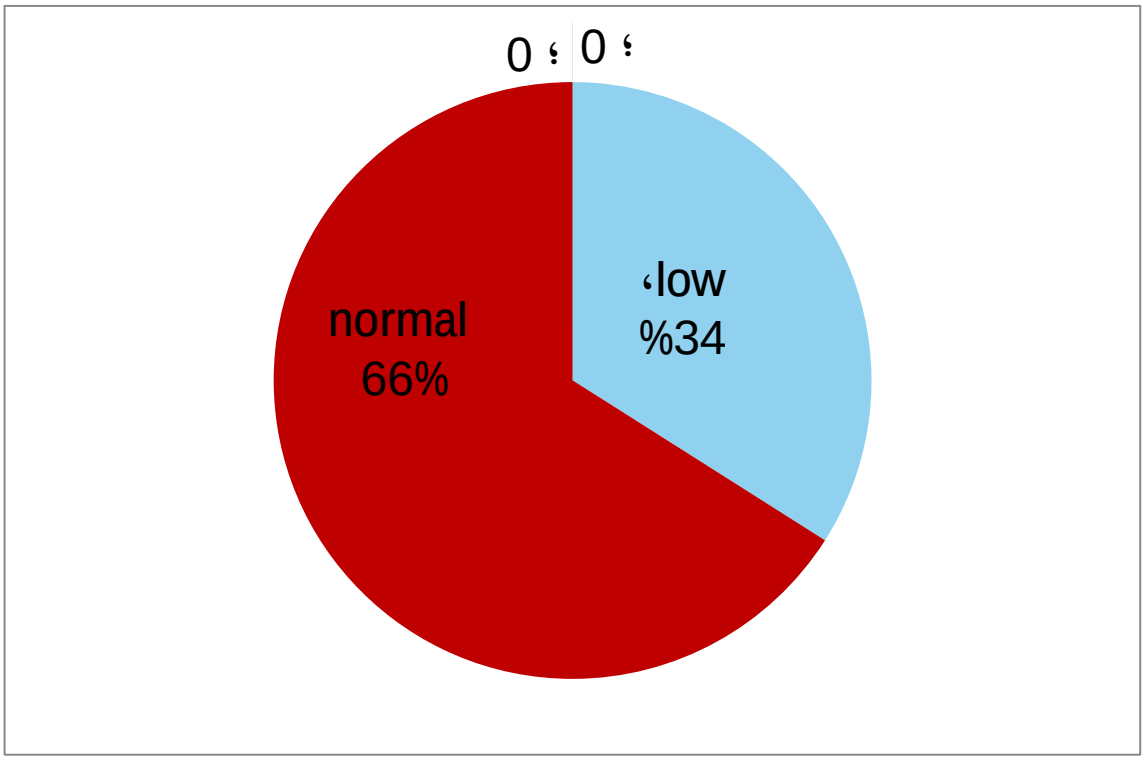


Figure no. 20

Distribution of patients according to fasting blood sugar level .

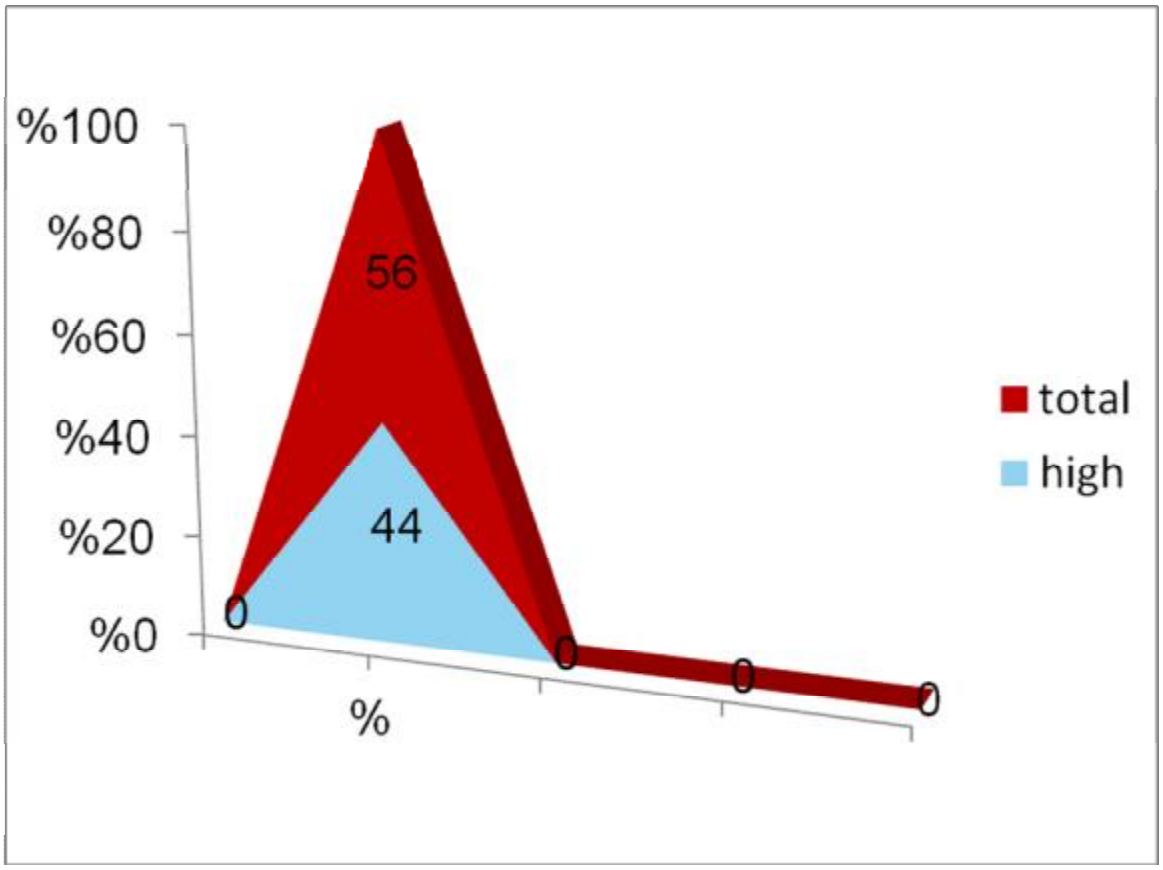


Figure no. 21

Distribution of patients according to urea level .

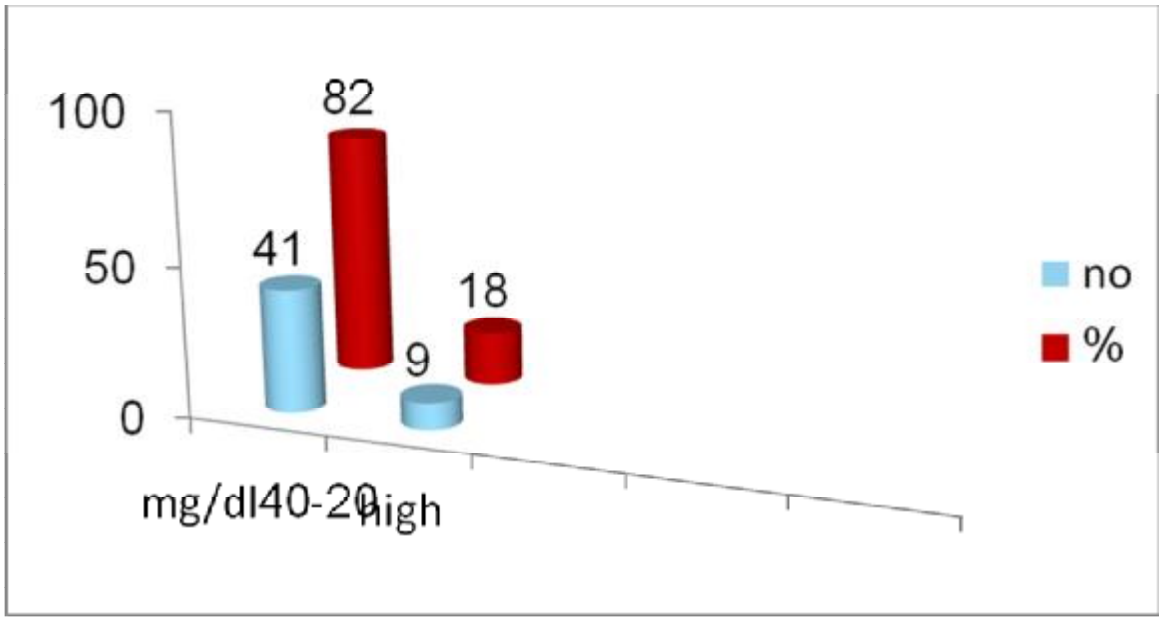


Figure no. 22

Distribution of patients according to level of lactic dehydrogenase /IU/L.

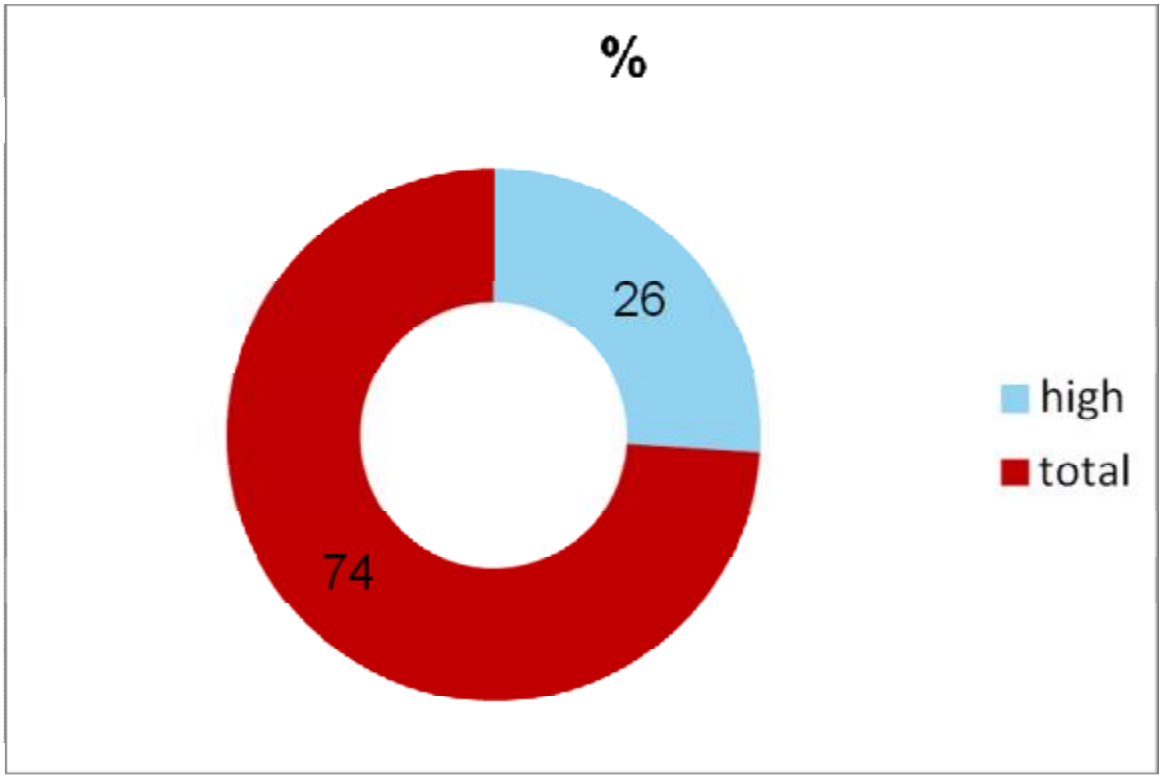
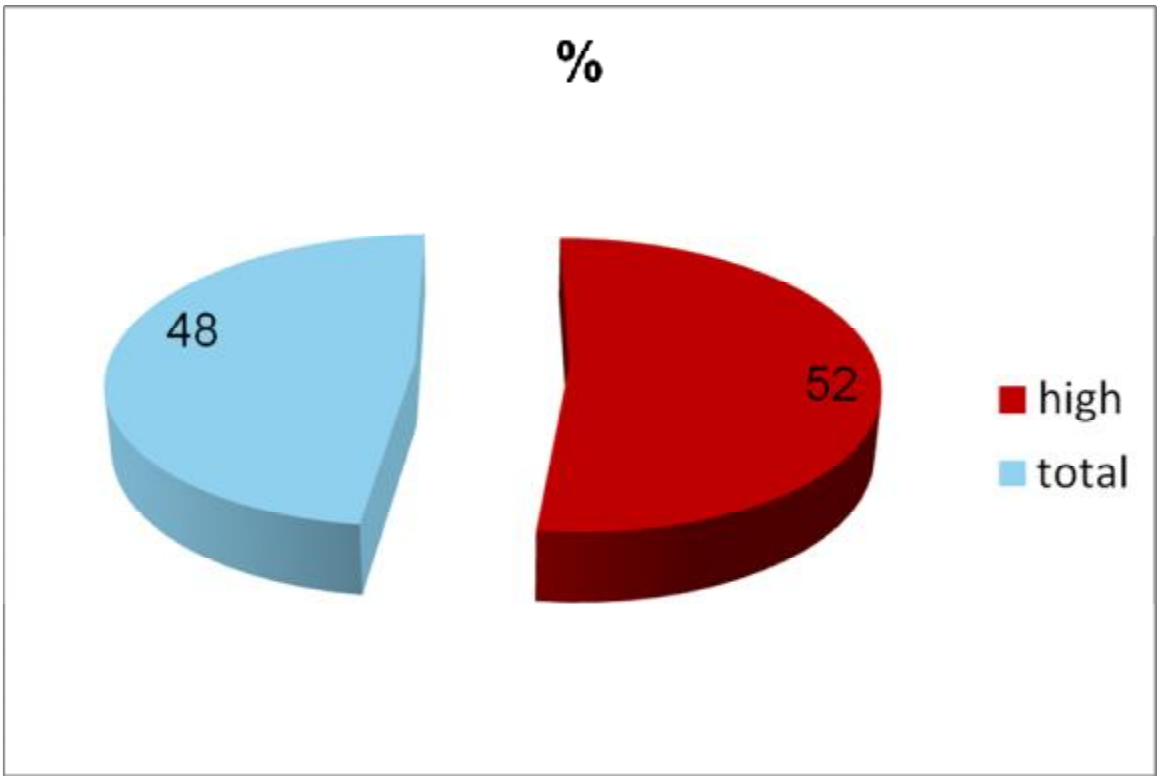


Figure no 23

Distribution of patients according to alkaline phosphatase level



As part of investigation X ray ,US ,CT scan abdomen and pelvis we found the following illus in 38% of patient by X ray ,76% finding by US indicated the diagnosis of acute pancreatitis like edematous bulky pancreas with free fluid in peritoneal cavity and illus.

while only 36% support the diagnosis by CT scan

figure no 24

Distribution of patients according to finding of erect abdominal X - ray

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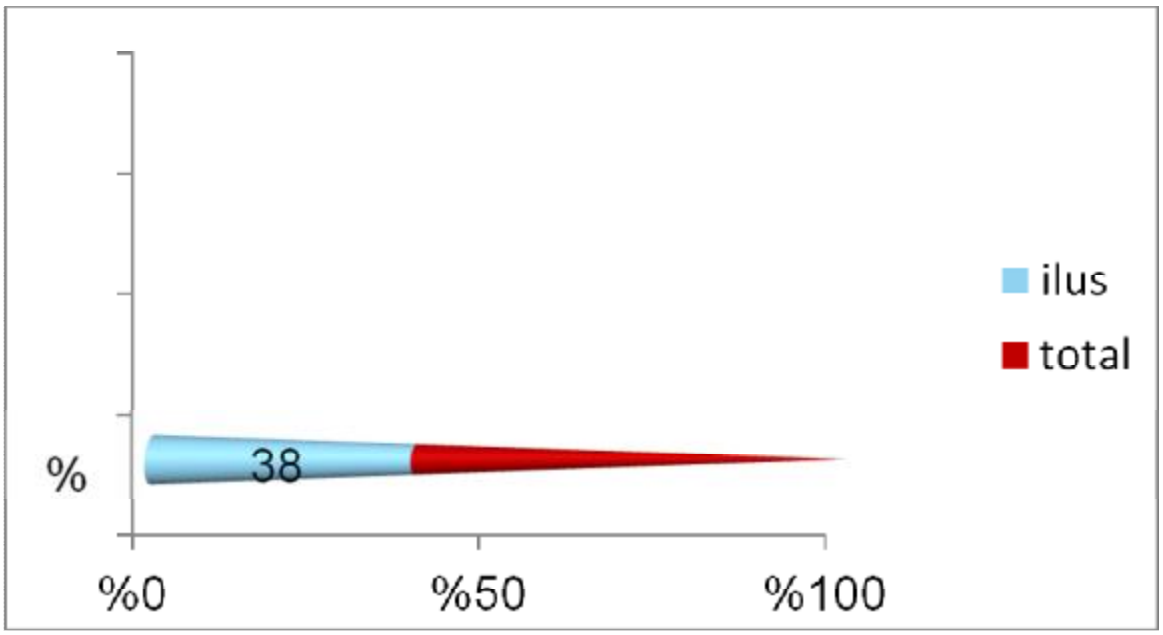


Figure no. 25

Finding of ultra sound abdominal and pelvic .

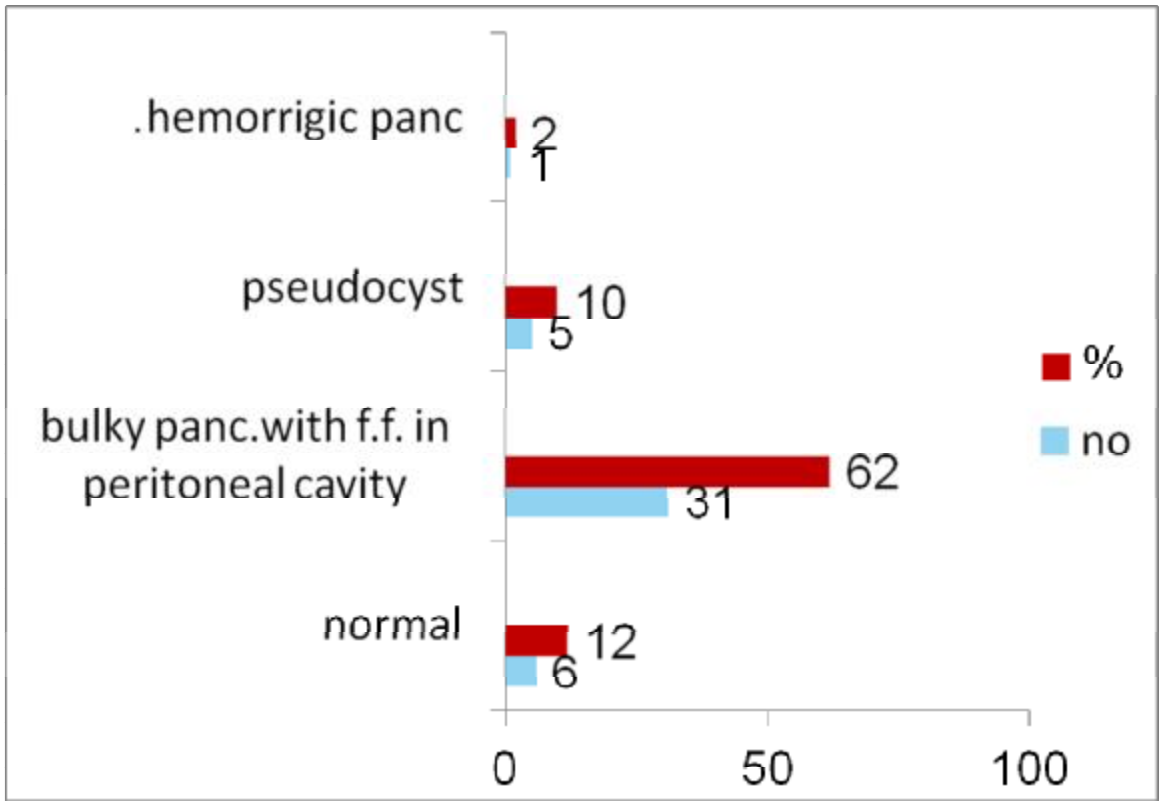
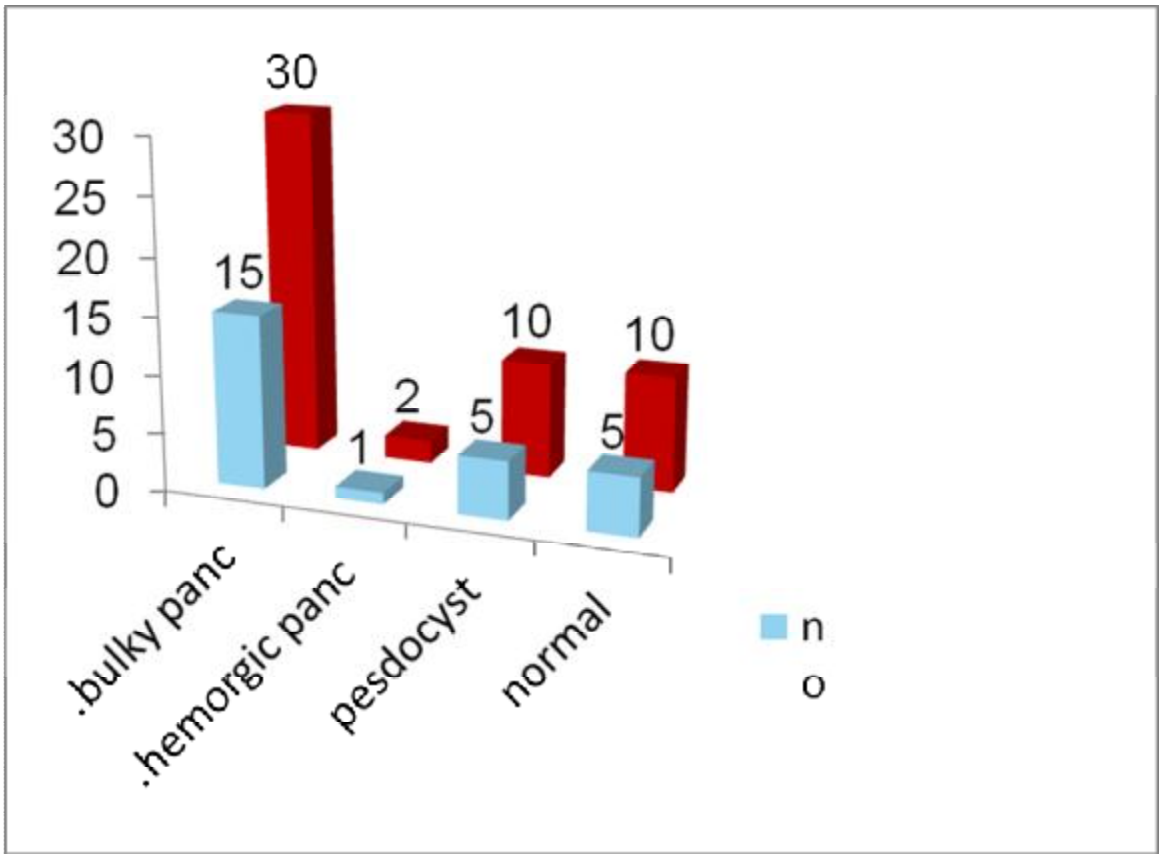


Figure no. 26

Distribution of patients according to result of CT/ SCAN .



As part of management we received the severe cases in ICU and Put them in NPO/ NGT ,vital mentoring, at least 5 days staying and given antibiotics and analgesia as part of regime of management we found 36% of group study admitted to ICU and transfer to ward and about 92% taken the mentioned regime the final outcome only one patient expired because of hemorregic pancreatitis and 5 others complicated

as psedeocyst and all others discharges after revived optimal management with good condition

Figure no. 27

Distribution of patients according to duration of stay in ICU.

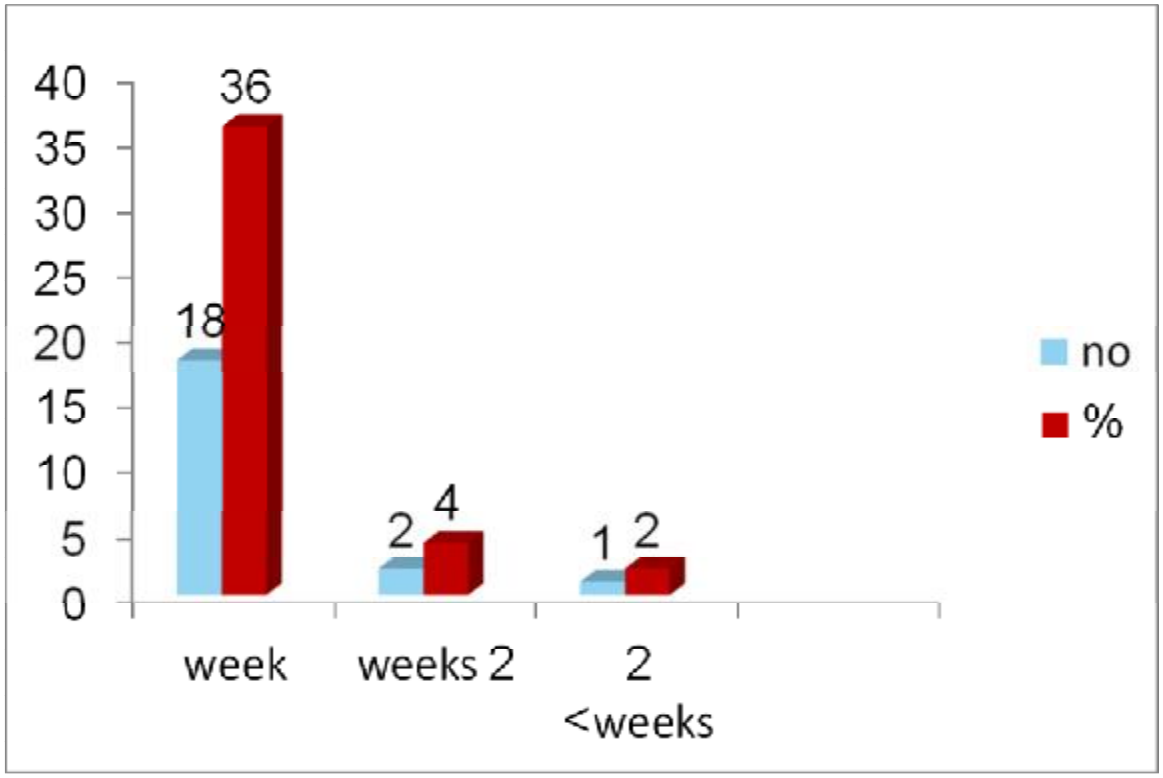


Table no 12

Distribution of patients according to NPO/NGT(naso-gastric tube).

Naso-gastric tube	No.	%
Not done	4	8
Yes	46	92
Total	50	100

Figure no 28

Distribution of patients according to duration of stay in hospital.

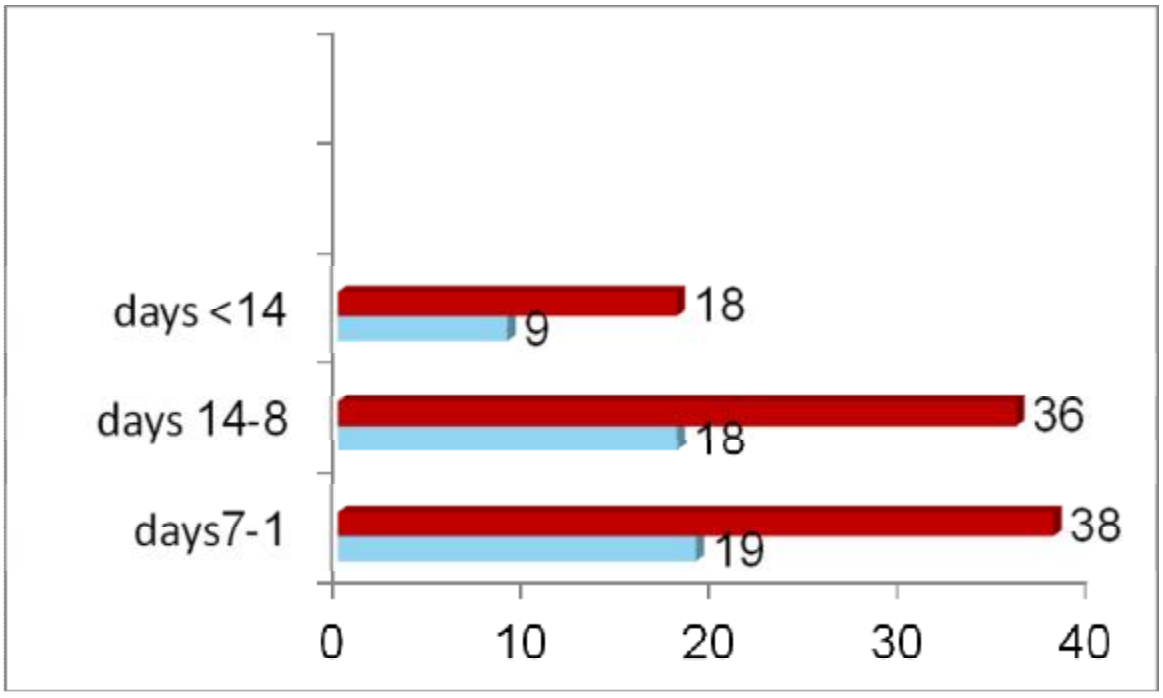


Figure no 29

Distribution of patients according to general condition at discharge from hospital.

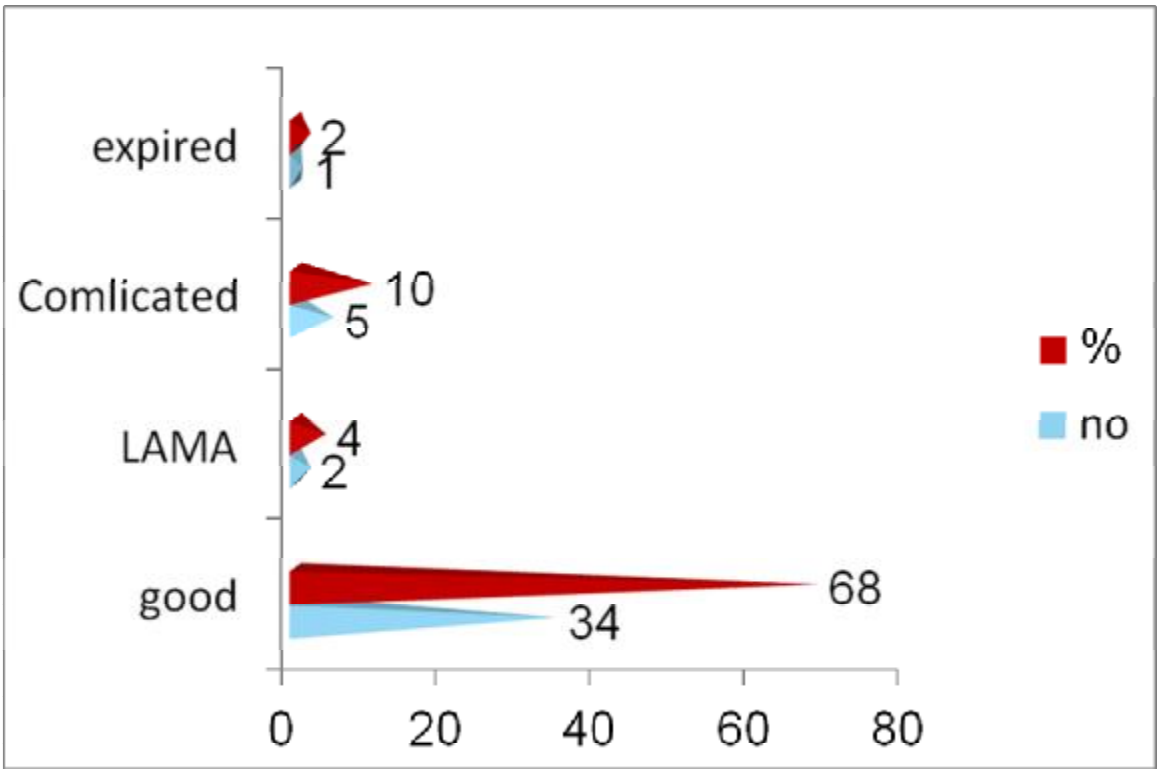
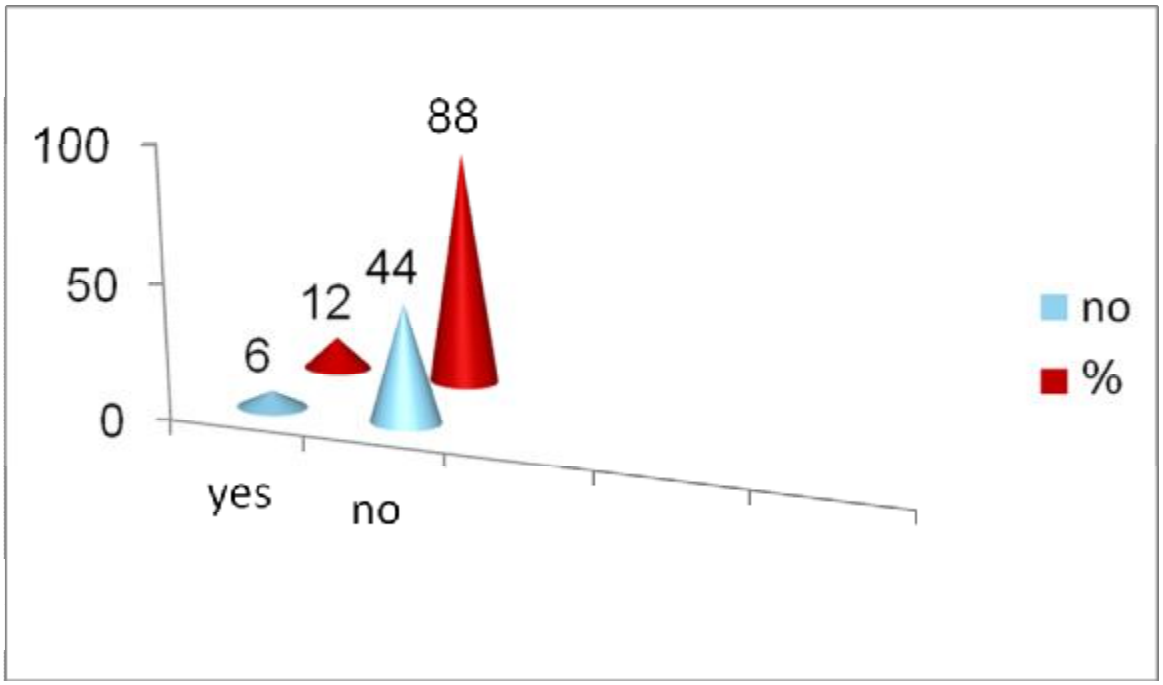


Figure no.30

Distribution of patients according to complication.



Chapter 5

Discussion

Discussions:

During the study period 50 patients were admitted to ALJALA hospital for treatment from acute pancreatitis ,

sex distribution was equal 50% male and 50% female , in other study male were more than female.[32]

Mean age was 55.6±15years, minimum age was 17 years and maximum was 90 years, 40% in age group 21-40years and 48% in age group 41-60years. Mean of age was 46years, it has been recorded that the mean age at onset depends on the etiology.

The mean ages of onset for various etiologies:

Alcohol-related - 39 years, biliary tract–related - 69 years, trauma-related - 66 years, drug-induced etiology - 42 years, ERCP-related - 58 years ,AIDS-related - 31 years, Vasculitis-related - 36 years.[13]

Majority of patients (96%) presented with typical symptoms.

Only 20% of patients were presented to hospital in the first day, result of other study showed that 50% of patients presented within 12 hours of onset,70% within 24 hours, and 30% after 24 hours.[32]

History of chronic disease was positive in 38% of patients, were , 20% is diabetic and 16% hypertensive and 4% hyperlipidemia

Surgical history was recorded positive in 30% o patients , were 22.5% done open cholecystectomy , 12.5%splenectomy and 6.25% laparoscopy cholecystectomy.

Fever was recorded in 34% of the patients , while 36% had tachycardia, they considered as sign of infection. other study found that presence of tachycardia and fever are associated with increased of mortality.[33]

Blood pressure was normal in majority of the patients 94%. According to , the Atlanta classification of acute pancreatitis has been used to differentiate between severe and mild cases of acute pancreatitis. According to this classification, patients are diagnosed with severe acute pancreatitis if they show evidence of organ failure e.g., systolic blood pressure below 90 mm Hg. Also in other study found that the hypotension is associated with increased of mortality.[33]

Abdominal examination finding ,80% feel tenderness and 12%abdomenal guarding which are considered as typical symptoms .

Result of erect abdominal X-ray 38% of patients had ileus.

Finding of ultrasound of abdomen and pelvises , 74% had bulky pancreas with free fluid in peritoneal cavity and 6% was normal.

Level of serum amylase was normal in 32%, while high in 60% of the patients.Serum amylase may be normal (in 10% of cases) for acute or chronic pancreatitis (depleted acinar cell mass) and Hyper triglycerimia.

Reasons for false positive elevated serum amylase include salivary gland disease (elevated salivary amylase), bowel obstruction, infarction, cholecystitis, and a perforated ulcer.[34]

Urine amylase level in 24hr was normal in 26 % and high in 54%. Levels of lactic dehydrogenase was normal only in 10% of the patients ,but high in 26%,which should be measured both at admission and at 48 hours in order to determine the Ranson criteria for survival [35,36] in our study was done only once , and not recorded at what time it had been done.

Mean of WBC,s was 11,000± 5 , it was high in 62% and was normal in 34% of the patients. A complete blood count (CBC) demonstrates leukocytosis white blood cell (WBC) count higher than 12,000/µL) Leukocytosis may represent inflammation or infection , it had been found positive correlation between the leukocytosis and severity of disease.[33]

Mean hemoglobin level was 12.95±1.97 g/dl in males , 44% of them were anemic and 48% were in normal level.

Mean hemoglobin level for the female was12.3 ±2.4g/dl, 44% of women were anemic and 56% was in normal level .

Mean level of urea was 27.3%± 17.7mg/dl, 26% had level range between 10-20 mg/dl, while 46% more than 20mg/dl.

Alkaline phosphatase level was normal level in 10% of the patients , high in 52% and38% was not recorded in the files . Determine alkaline phosphatase level to search for evidence of gallstone pancreatitis.[11]

Calcium level was normal in 14% , while low in 34% , mean value of 8.2± 0.94mg/dl. Measure calcium level to search for an etiology of

pancreatitis (e.g., hyper calcemia) from any cause can lead to acute pancreatitis or complications of pancreatitis (egg, hypocalcemia resulting from saponification of fats in the retro peritoneum).[32]
Fasting blood sugar level was normal in 20%, while high in 44% of patients , with mean level of 171.3±88.3mg/dl. Measure blood glucose level because it may be elevated from B-cell injury in the pancreas.[32]
Lipoprotein level was normal in 10% , while high in 4% , but majority 86% was not recorded .
Although most cases of acute pancreatitis are secondary to biliary disease or excess alcohol consumption[2] ,only 6% of patients were recorded that they drinking alcohol , but majority (74%) of files were had not recorded . any information about alcohol, in similar study suspected etiologies were alcohol in 20.4% .[32] In the United Kingdom, of cases 15–29 % related to alcohol , In audit 20% were, related to alcohol.[32,37,39] Others state that alcohol use is a major cause of acute pancreatitis (accounting for at least 35% of cases).[40]
Result of CT/Scan was not recorded in 64% of files, bulky pancreas with pleural effusion and GBS were in 15 % , and was normal in 8% , in audit study they found that only one third of patients had CT within 10 days, despite the availability of facilities in all hospitals.[32]Although CT is necessary to document the extent of necrosis and fluid collections, it is expensive, not required for diagnosis in most cases,

and is not used in early prediction of severity.[28]Others considered Abdominal CT scans provide prognostic information. [41]

Gall bladder surgery was recorded in 28% of the patients .

36% of patients were admitted to ICU within 7 days, while is more in than half was not recorded in 58% of files.

More than half of patients (68%) was discharged from the hospital in a good health , 30% of the files there were missed information and one patient(2%) expired. In a prospective audit they found the overall mortality rate was 9.2%. This was within the recommended audit standard of10% and is similar to the 8% rate reported in premortem diagnosed patients in north west London (1988–1992)[37] and 8.3% in Nottingham(1987–1993),[42]although values between 3%[31] and 25%[31] have indeed been reported in the UK.

A recent audit in six European countries reported a mean _+(SD) mortality rate of 9.2% (12.1%).[32] Further afield, a retrospective study in Sowetan Africans reported a mortality of 8.1%27 and in the United States, death rates as low as 2% have been reported.[43] These variations are in part due to differences in data collection, autopsy rates, and definitions.[37]

Nasogastric tube was used in 92% of the patients, although it was used in the past, patients were routinely treated by withholding food and placing a nasogastric tube with suction for two to 10 days. This

was done in an attempt to reduce pancreatic stimulation by food, hydrochloric acid, cholecystokinin and secretin.

Withholding food by mouth does reduce pain, but use of a nasogastric tube with suction is no longer advocated as a routine therapeutic measure in acute pancreatitis because it has not been shown to decrease symptoms, mortality or hospital stay. However, a nasogastric tube may be used when the patient has protracted vomiting or if obstruction is seen on the abdominal radiograph.[43,44]

But in our hospital this type of management had been succeed in most of the cases

The complications which is found only one patient had hemorrgic pancreatitis who was expired and 5 other patients were developed psedopancreatic cyst , this result similar to other study result were one patient only had complication.[32]

Chapter 6

conclusion

Conclusions:

Accurate assessment of the incidence and mortality of acute pancreatitis is difficult, as mild pancreatitis may be subclinical and death may occur before the diagnosis is made in sever and fulminante attacks

All junior doctors should suspected acute abdomen for every patient with acute abdomen so to avoid delay in diagnosis

Many important information were not recorded in the files , thus we recommended electronic filing system to be adopted in the hospital .

Prospective study for longer duration is strongly needed.

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Question aire

General data;

age, sex ,symptoms ,which is typical or atypical, duration of symptoms h/o chronic illness, its type e.g. hypertension or diabetes ,surgical history and type of operation

Clinical data;

fever ,tachycardia ,low blood pressure

Investigation;

CBC,FBS, serum Ca, WBC, LDH ,Na, K, urea ,urine and serum amylase ,alkaline phosphates ,lipoprotein…..

Radiological finding;

plain X ray ,US ,Ct scan, ERCP,