SURGICAL TREATMENT OF THE COMPLICATIONS OF ACUTE PANCREATITIS, OUR 3 YEARS EXPERIENCE

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Abstract

Aim: The pancreatic pseudocysts treatment includes open surgery, percutaneous drainage, endoscopic trans enteric drainage, and the trans papillary one. None of these techniques can guarantee 100% success, and without any complication. We describe our experience in pancreatic pseudocysts treatment.

Material and method: This retrospective descriptive study enrolled 170 patients from 2008-2011. Out of 170 patients, 59 patients were diagnosed with postpancreatitis pseudocyst. Ranson criteria, APACHE II points, imaging examinations, and laboratory findings are all recorded.

Results: 99 patients were diagnosed with severe acute pancreatitis, whereas 71 patients with mild form of acute pancreatitis. The pancreatic necrosis was encountered in 28%, sepsis in 37%, pseudocysts in 34.7%, and other minor complications in 0.3% of all cases. In majority of cases the most important clinical features were epigastric pain, nausea, vomiting, fever, increased amount of white blood cells, and stable high amylase level. The ultrasound examination and computerized tomodensitometry examination confirmed the diagnose. Pseudocysts over 6 cm width, and their complications (over infection, gastrointestinal obstruction, peritonitis, recurrences, and fistulas) are consider to be treated. 16 patients were spontaneously resolved, 36 patients undergone to open surgery (cyst-enteric anastomosis), and finally in 7 patients external drainage was performed. The patients suffering with pancreatic necrosis and sepsis were medically treated close to intensive care unit.

Conclusions: The surgery is essential in pancreatic pseudocyst treatment. The drainage procedures (percutaneous and endoscopic one) can be considered as well. Endoscopic treatment remain our future challenge.

General Considerations

Acute pancreatitis definition (PA) represents an acute inflammation of the pancreas, which is associated with its autolysis and involvement in different scales of other tissues around it, or the organs or systems remotely.

Epidemiology of acute pancreatitis (PA)

Incidence and etiology of acute pancreatitis varies in different countries of the western world. A small number of Published Studies show conflicting results in the rate of incidences of pancreatitis ranging from 20 to 30 new cases per 100,000 inhabitants per year. A 2009 European study showed that people over 60 years have an increased incidence of pancreatitis from gall stone. Incidence (PA) varies by geography. The incidence of acute pancreatitis is different in different countries. In Albania, the incidence is 1,042 per 3,544,808 inhabitants. In 80% of cases with acute pancreatitis causes are alcohol and gall stones. The third cause is

the ERCP procedure but this pancreatitis is a light form.

Anatomical and physiological data

Anatomically pancreas is a body bent over the convexity of the vertebral column at the level of L2-L3 with oblique direction up and left by duodenum d2 until the spleen. It is located in lesser sac over the transverse mesocolon, fixed to the right from ligament of Treitz and to the left from Told fascia. Pancreas weighs 70 grams and is composed of 4 parts: the head continues with the neck, the body and the tail which continues with the pancreato - splenic ligament. Normally is white color in pink, with lobular appearance rolled out by a thin transparent capsule. Pancreas is a gland with dual secretion exocrine and endocrine.

Pancreas plays an important role in digestion (exocrine function) and in maintaining the glucose level (endocrine function). About 85% of the function is exocrine and only 2% endocrine function.

There are about 40 acinar cells that make up an acinus which relates centric acinar cells with pancreatic duct. Endocrine part consists of about a million islets of Langerhans mainly in the tail of the pancreas that secrete insulin and glucagon.

Exocrine pancreas secretes 1000ml per day basic pancreatic juice, without color, isosmotic juice, consisting of enzymes secreted by acinar cells and rich in bicarbonates secreted by pancreatic ductal cells. The enzymes comprise mainly proteases and enzymes that serve to digest lipid and glycogen. They are secreted in the form of inactive pro enzymes to prevent autolysis of pancreatic parenchyma and become active after reach the duodenum in the form of trypsin, elastase, chemotrypsin, carboxypeptidase and phospholipase A. Hormones that promote secretion of pancreatic enzymes are secretin, cholecystokinin, gastric peptide, substance P, acetylcholine. Endocrine part produces hormones are presented in Table nr.1.

Table nr.1. Hormones that are produced in the endocrine part of the pancreas

The hormone	Islet cell	Function
Insulin	β cells	Reduce blood glycemia
Glucagon	α cells	Increase blood glycemia
Somatostatin	δ cells	
Pancreatic polypeptide	Pp cells	Inhibit the secretion of all the other Gastro intestinal hormones
Amilin\Islet Amyloid Polypeptide	β cells	Inhibit the secretion of exocrine pancreas and insulin secretion
Grelin	ε cells	Inhibit release and the function of insulin

Physiopathology of acute pancreatitis

Pancreatitis is an acute inflammatory process characterized by activation inside the gland of pancreatic enzymes, the recruitment of inflammatory cells in the pancreas and blood microcirculation changes in the pancreas. The degree of inflammatory changes varies from a short episode of clinical symptoms to a systemic inflammatory response with multi organ failure. Two concepts more accepted explaining the pathogenesis of biliary pancreatitis are: Common channel hypothesis and the hypothesis of ductal hypertension. Common channel blocking brings reflux of bile in the pancreas gland causing pancreatitis. Increased pressure in the channel and not just

pancreatic biliary reflux brings destruction of small ducts and outspreading of pancreatic enzymes in pancreatic parenchyma. Moreover ductal hypertension blocks acinar release enzymes facilitating intra acinar activation of them. In particular bile acids increase intracellular calcium concentration that facilitates activation of trypsinogen to active trypsin. An episode with excessive drinking of alcohol n does not cause acute pancreatitis. Alcohol increases the mobility of the sphincter of ODDI which inhibits pancreatic secretion, but ther is no experimental full assurance to the people about this effect of alcohol in the sphincter of ODDI. Henri Sarles proposed that alcoholic pancreatitis is caused by precipitation of secreted proteins in pancreatic small ducts which forms a nucleus for calcification and stone formation in the pancreatic ducts. For more alcohol destabilizes zymogens granules and the membrane of lysosomes. Conversion of alcohol in acetaldehid increases the cellular concentration of calcium ions that causes depolarization of mitochondria resulting in cell death.

Prognostic criteria of acute pancreatitis

A number of scoring systems are used to determine the gravity of the AP and its prognosis. In the beginning were used Ranson criteria presented in Table nr.2.

Table nr.2. Ranson criteria

In emergency room	Within 48 hours
Age >55 years old	Hematocryt decrease > 10%
Leucocyte >16.000	Increase of blood urea > 2 mg/l
LDH >600UI/l	Calcemia < 8 mg/dl
AST >250UI/l	Deficit i bazave > 5mmol/l
Glycemia >220mg/dl	PO_2 < 60 mmHg
	Liquid lack > 4 l

Atlanta revised classification provides four categories of patients with acute pancreatitis and presented in Table nr.3.

Table nr.3. Classification of Atlanta

Gravity of pancreatitis	Locale complications		Sistemic complicationes
Mild	Without pancreatic and peripancreatic complications	and	Without multi organ failure
Moderated	Sterile Pancreatic and peripancreatic complications	Or	Multi organ failure Temporarily
Severe	Infected Pancreatic and Peripancreatic complications	Or	Multi organ failure Permanent
Critical	Infected Pancreatic and Peripancreatic complications	And	Multi organ failure Permanent

By Apache II clinical classification includes the assessment of temperature, cardiac frequency, respiratory frequency, average arterial pressure, oxygenation, arterial pH, potassium and sodium serial, creatinine, hematocryt, and the number of leucocytes.

General information on conservative treatment of AP

Main principles of treatment of AP include correcting volemia, controlling pain, oxygenotherapy, replacement of electrolytes (potassium, calcium), correction of metabolic disorders (Hyperglycemia), nutrition, and antibiotic prophylaxy.

Apache II on 8 points, Ranson over 3 points, and c reactive protein 150 mg/dl are indicative of severe acute pancreatitis. Oxygenotherapy given by nasal oxygen tube or mask to maintain oxygen saturation 95%. In severe cases patients are intubated and assisted by ventilation with positive pressure at the end of expiration (Peep). Treatment of pain is necessary and can be done with pethidin and non steroide antinflammatory drugs. Preferably no morphine is used because it promotes spasms of the sphincter of Oddi. The first days the pain makes intolerable the enteral nutrition, so fluid and small amounts of caloric foods were given to the patients which are increased gradually with the decrease of pain, cessation of nausea to vomiting, and restoring the noise of intestinal motility. Enteral nutrition is preferred because it is physiological, prevents intestinal mucosa atrophy and infections, costs less, and finally gives less complications. Of course the beginning of intestinal food has the condition that the enteric competence or the intestinal transit is restored therefore the patient is supported with parenteral nutrition. Fluid therapy is monitored by the amount of urine over 0.5 ml/kg weight, venous central pressure measurement, and clinical signs as arterial pressure, pulse, elasticity of the skin, uremia and creatinine levels, hematocryt, the lack of hemoconcentration. Fluid excess therapy varies depending on the case, but reported figures from 250-1000 ml/h. Is important to avoid conditions as hypovolemia and hypervolemia. Often they have even hypocalcemia as a result of necrotic fatty tissue, so it must be corrected depending on the serial level. Hyperglycemia is a common finding and correcting with insulin is done due to glycemia levels and glucose load. Preferably we use imipenem and ciprofloxacin because they have better penetration into the pancreatic tissue.

Complications of AP

AP local complications rank collections, pancreatic necrosis, abscess, pseudocyst colonic obstruction, and pleural fistulas. Acute collections of liquid are displayed early in the course of acute pancreatitis and reside near or within the pancreas. This is a frequent complication in patients with severe form of pancreatitis in 30-50% of cases. Half of these lesions spontaneously regression. Acute collections of liquid which do not reduce turn in pseudocysts or abscesses. Pancreatic necrosis is a focal area or diffuse of the parenchyma of the pancreas that typically is associated with peripancreatic fat necrosis. The amount of necrosis increases with worsening clinic and is diagnosed by contrast scanner, which looks as well limited hypodense area, greater than 3cm, which comprise over 30% of the pancreas. The pseudocyst is a limited pancreatic fluid collection with granulation fibrous tissue walls and formed not only during acute pancreatitis but also during trauma pancreas or chronic pancreatitis. Pseudocysts of acute pancreatitis can be palpated but usually are diagnosed by imaging. They are usually round or ovoid and have a clear wall that is identified by ultrasound or scanners. Pancreatic abscess is a collection of intra abdominal pus usually limited, near the pancreas, containing little or no pancreatic necrosis. D'Egidio and Schein, in 1991, described a classification of pancreatic pseudocyst based on the underlying etiology of pancreatitis (acute or chronic), the pancreatic ductal anatomy, and the presence of communication between the cyst and the pancreatic duct and defined three distinct types of pseudocysts. Type I, or acute "postnecrotic" pseudocysts that occur after an episode of acute pancreatitis and are associated with normal duct anatomy, rarely

communicates with the pancreatic duct. Type II, also postnecrotic pseudocysts, which occurs after an episode of acute-on-chronic pancreatitis (the pancreatic duct is diseased but not strictured, and there is often a duct-pseudocyst communication). Type III, defined as "retention" pseudocysts, occurs with chronic pancreatitis and is uniformly associated with duct stricture and pseudocyst duct communication. Another classification, based entirely on pancreatic duct anatomy, is proposed by Nealon and Walser.

Type I: normal duct/no communication with the cyst.

Type II: normal duct with duct-cyst communication.

Type III: otherwise normal duct with stricture and no duct-cyst communication.

Type IV: otherwise normal duct with stricture and duct-cyst communication.

Type V: otherwise normal duct with complete cutoff.

Type VI: chronic pancreatitis and no duct-cyst communication.

Type VII: chronic pancreatitis with duct-cyst communication [10].

Abscesses is formed in acute pancreatitis or pancreas trauma. Appear after 4 weeks from the beginning of severe acute pancreatitis. Colon complications during the course of acute pancreatitis are rare but fatal for life. Pathogenetic mechanisms are explained by the spread of pancreatic enzymes through retro peritoneum in mesocolon causing pericolitis, inflammatory external compression from mesocolic mass formed after necrotizing of adipose tissue, hypotension and shock from mesenteric artery thrombosis. This leads to failure of the colon, fistula formation, perforation, and obstructing during the course of acute pancreatitis. Fistulas can be pancreato-Pleura, pancreato-colonic, and splenic infiltration. Table nr.4 illustrates the prevalence of local complications of AP.

Table nr.4. Local complications of AP

Echographic findings in acute pancreatitis	Prevalence
Peripancreatic inflammation	60%
Heterogeneous parenchyma	56%
Decrease of echogenicity of pancreas	44%
Unclear ventral borders	33%
Enlarge of the pancreas	27%
Intra pancreatic focal changings	23%
Peri pancreatic liquid collections	21%
Hypoechogen focal mass	17%
Venous thrombosis	4%

AP treatment modalities

Modalities include endoscopy and open intervention. Endoscopic modalities included ERCP, sphincterotomy, endoscopic cyst gastrostomy or rarely a cyst duodenostomy, endoscopic necrosectomy. Success varies 80-93%, and complications in 7-23%. Open surgical intervention may be of interest in the case of large liquid collections or in cases of extended pancreatic necrosis or in the case of their over infection at the time when the

abscess is formed. Open surgery is usually a last resort. If pseudocysts shown in acute pancreatitis are drained with surgical routs a cyst gastroanastomosis or cyst duodenoanastomosis is made for the pseudocysts of the head of the pancreas or a cyst jejuno anastomosis is made for the pseudocysts of the tail of the pancreas. Indications for surgery: 1) sepsis deteriorating clinic plus temperature and leukocytosis with or without multi organ

failure; 2) infected pancreatic necrosis confirmed by fine needle aspiration cytology; 3) scanner with contrast showing lying pancreatic necrosis with air pockets which are pathognomonic for infected necrosis 4); The poor condition overall with persistent abdominal pain, nausea, anorexia, progressive decline in weight.

The aim of the study

The first goal is to assess the clinical data, laboratory, imaging in determining the gravity and identifying acute pancreatitis complications, as well as evaluating the prognosis associated with these data. The second goal is the identification of our experience in endoscopic and surgical treatment.

Material and methods

This is a descriptive, retrospective study of General Surgery clinic during the period 2008-2011. The study included 170 patients with acute pancreatitis and of these 59 patients with pseudocyst of acute pancreatitis. Were evaluated Ranson criteria and Apache 2, as well as the formation of pseudocysts (based on the criteria of imaging, laboratory, and clinics). There have also been assessed opportunities handling pseudocysts. There are studied 59 patients with pseudocyst and the study included only those patients who needed surgery because of acute complications of pseudocysts as infection, peritonitis, or had a symptomatic pseudocyst, large and persistent. They analyzed data such as age, sex, associated diseases, clinical data, surgical procedure used, post operative course.

Results

There have been studied 170 patients with acute pancreatitis of our surgical service and of these 59 patients with pseudocyst of acute pancreatitis in our clinic. Male female ratio was 14 to 8. Average age 38.2 years. 99 patients belonging to the form of severe acute pancreatitis and 71 patients with acute pancreatitis belonging to the easy form. Pancreatic necrosis group meets at 28.0%, sepsis in 37.0%, pseudocysts 34.7%, other 0.3%. Dominant symptoms in the majority of patients were epigastric pain 95.5%, palpable mass 12.5%, nausea, vomiting, temperature and leucocytosis 45.5% as well as the high value of amylase in plasma. Figure nr.1 presented etiological factors of acute pancreatitis predisposing thus in pseudocyst formation.

Table nr.5. Etiological factors and complications of acute pancreatitis

Echographic findings in acute pancreatitis	Prevalence
Peripancreatic inflammation	60%
Heterogeneous parenchyma	56%
Decrease of echogenicity of pancreas	44%
Unclear ventral borders	33%
Enlarge of the pancreas	27%
Intra pancreatic focal changings	23%
Peri pancreatic liquid collections	21%
Hypoechogen focal mass	17%
Venous thrombosis	4%

Ultrasound turned positive in 77.2% of patients, screened in 81.8% of patients. 16 pseudocysts were reduced spontaneously and were followed with ultrasound and scanners.

Surgical route were treated 36 patients (61%) through the formation of cyst-entero anastomosis for internal drainage. By patients treated surgically, 18 patients (50% of cases)

underwent cyst-entero anastomosis, which are simple (11 patients), or Roux en y (7 patients). 13 patients (36.1% of cases) underwent formation of cyst-gastro anastomosis. 5 patients (13.9%) of cases resolved surgically) underwent formation of cysto duodeno anastomosis for internal drainage of pseudocysts. With external drainage were treated forms of pseudocysts turn into abscess through percutaneous drainage. In this way were treated 7 patients with pseudocysts (11.9%). 16 patients (27.1%) of pseudocysts enjoyed spontaneous regression. In Table nr.6 are summarized surgical techniques used.

Table nr.6. Operator techniques followed in our series

Types of treatment		Number of patients
Spontaneous regression		16 patients
Cyst-entero anastomosis	Simple ones	11 patients
	Roux en y	7 patients
	Cyst-gastro anastomosis	13 patients
Cyst-duodeno anastomosis		5 paciente
Percutaneous drainage		7 paciente

Pseudocysts treated surgically were about 6 cm, with the average size about 8.2 cm. In the postoperative period, in patients treated with external drainage were found 2 recurrences which were selected respectively, each forming cyst-gastro anastomosis during surgical internal drainage by road, and one again with internal drainage, but formed cyst-jejuno anastomosis Roux en y. In patients with percutaneous drainage of pseudocysts we had a trans gastric fistula formation formed after the drainage of a pseudocyst turn into an abscess as shown below. In patients with spontaneous regression of pseudocysts was realized their fistulization in neighboring cavities of the body, namely 3 cases. A fistulization was formed between the pseudocysts of the body of the pancreas and jejunum, while two other fistulizations were created between pseudocysts of the head of the pancreas and stomach.

Patients with systemic complications of acute pancreatitis as sepsis and multiorgan or insufficiency, including the left pleural versament were treated in intensive service units. In Table nr.7 are shown in each chosen treatment tactical complications.

Table nr.7. Complications after treatment

Spontaneous regression	Perkutaneous drainage	Surgery
Pseudocyst-jejunum fistula Pseudocyst – stomach fistula (2)	Recurences (2)	No complications

Discussion

80-85% of cases of acute pancreatitis are light form without displaying multi organ failure. Mortality of this group is 1%. 15-20% of cases of acute pancreatitis are more severe with the appearance of pancreatic necrosis and multi organ failure. In these patients remains sterile necrosis in 60% and infected in 40% of cases. The last category of patients have higher mortality by 25-70%. In our study of patients with acute pancreatitis mortality varies by age group, hospitalization and based

on the form of treatment (conservative or surgical). More specifically, it results a 6% mortality in patients aged 20-29 years, 9% in the age group 30-39 years, 10% 40-49 years, 18% 50-59 years old, 9% in 60-69 years, and 48% in people over 70 years old. So with increasing age also increases mortality from acute pancreatitis and its complications. In patients who stayed in hospital over 6 days, mortality was 33%, whereas patients who have stayed 1-6 day mortality was 67%. This is explained by the fact that there is a high mortality and aggressive forms so hospitalization is shorter and higher mortality. Mortality in conservatively treated resulted in 60%, while those with surgical treatment 22%. To patients with pancreatic necrosis which were operated, results in 18% mortality. This supports the fact that if the indication is placed properly and intervened in time, surgery significantly reduces mortality from acute pancreatitis.

Scanning is the examination of choice for the diagnosis of acute pancreatitis and its complications. In our study computerized tomography has succeeded in diagnosis in 81.8%, and examination ultrasonic in only 71.2%. Fluid scanner can not distinguish from solid detritus distinguished better with Magnetic Resonance. This fact supports the studies of several authors in the literature (1,2,3). According to some other authors (4) distinct resonances with Fluid good solid detritus from and replaces the contrast scanner.

Imaging can not distinguish sterile collections from infected while the air in infected collections is present only in 20% of the time. Screened within 72 hours from the installation of the disease has no value and the diagnosis made through clinic and laboratory data.

One of the complications that occur during the course of acute pancreatitis is forming of pseudocysts of various magnitude, etiology and localization in the gland of pancreas. Pseudocyst is a pancreatic fluid collection surrounded by fibrous wall and surrounding viscera without a surrounding epithelium. The discussion about the treatment of pseudocysts displayed during the course of acute pancreatitis is based on the fact that

when is the optimal time for intervention, which is the best intervention and how operators will manage complications of these techniques. Pseudocyst are seen in 10% of patients with acute pancreatitis as a result of inflammation, trauma and ductal obstruction (5,6). Diagnosis is established clinically and through imaging. In ERCP are given data for the course of pseudocyst and are made the selection of patients suitable for treatment not with open laparotomy. ERCP adds risk for infections of pseudocyst, perforations, intestinal bleeding. If ERCP shows no communication of pseudocyst with pancreatic ductal system has been used percutaneous drainage. When ERCP shows communication between the pseudocyst and the ductal system have been used pancreatic stents trans papillary whuch are preferred also in patients with pancreas cancer. Our country has used ERCP for diagnosis, but no for treatment of pseudocysts, and here we talk about general principles for planning surgical intervention via gives ERCP assistance. This remains a priority for the future in the treatment of pseudocysts in our country. 50% of pseudocysts are reduced spontaneously. According to Andrea Sanberg and bp (7) pseudocysts can be reduced spontaneously in 70% of cases. Over time, 40% of patients with untreated pseudocyst gives different complications (6).

Surgical drainage is the gold standard for the treatment of pancreatic pseudocysts (8,9,10). We use open surgical intervention in the initial treatment for cases suitable for surgery and anesthesia. Percutaneous drainage is used in pseudocysts that are infected, with thin walls, no ductal leak, the patient is clinically stable (11,12,13). It remains for the future to our country the endoscopic transpapillary drainage, which is used in symptomatic cases with thin walls pseudocysts and ductal leak, or in patients who refuse surgery. Cyst gastrostomies by endoscopy are used in pseudocysts with the wall adherent to the posterior gastric wall. If the wall of the cysts is adherent with duodenum an endoscopic cyst duodenostomy is done. We have successfully treated patients with pseudocyst in surgical route, formed cyst-jejuno anastomosis simple or type roux

en y in 50% of cases. Surgical routs are implemented also in internal drainage in surgical techniques of cyst-gastro anastomosis and cyst-duodeno anastomosis respectively 36.1% and 13.9% of cases.

The risk of infection increases with time and occurs in 10 to 15% of the patients or by the displacement of intestinal bacteria, or infection of any hematoma inside the cyst, or by the use of ERCP. In these cases it becomes external drainage that has the risk of fistula formation in 12 to 20% of patients. We suggest internal draining training cyst entero anastomosis in infected pseudocyst only in clinically stable patients with a mature walled pseudocyst. This conclusion is also supported by foreign authors (14,15).

External or percutaneous drainage mortality was 10% and for that internal surgical way is lower 3%. Recurrences for external or percutaneous drainage are 18%, while surgical intern drainage are again lower by 8%.

Persistent pancreatic fistula formation after percutaneous external drainage of pseudocysts of acute pancreatitis requires simple internal drainage cyst enteric or drainage type roux en y. External drainage in our study had the highest morbidity then internal drainage (60% vs. 40%) which is consistent with the data of foreign authors (16).

Concluding that is required a multidisciplinary team for the treatment of pancreatic pseudocysts comprising surgeons, endoscopic specialists and radiologe. 70 up 80% of the fistula closed spontaneously within 4 months of training, with a performance by prolonged recovery in cases of fistula with high debit(more than 200 ml per day). In our study had fistula formation after percutaneous drainage of a form suppurated pseudocyst of pancreas, who, after drainage made recurrence and just p recurrent pseudocyst was drainaged spontaneously in the stomach through the formation of a trans gastric fistulas.

There was no fistula formation after surgical treatment of pseudocysts. The incidence of repeated open surgical intervention in patients with external drainage is lower than in those with internal drainage (16.6% versus 75%). This is supported by the results of the literature (15,16).

The most important strategy of the treatment of pancreatic fistulas can be a combination of early total parenteral nutrition (but not extended in time) with nutritional jejunostomies. The same recommendations given by other authors (17).

The use of non-absorbable suture provides better haemostasis and avoids haemorrhage. Recent mortality has fallen from 8.6 to 16% boundaries within the limits 0 to 5%.

In the case of pancreatic necrosis observed during the course of acute pancreatitis indications for surgery and the optimal time for surgical intervention provided as general principles below, given the track record foreign surgical encountered in the literature (18). In our study is described a case with infected pancreatic necrosis treated surgically through open nekrosectomy or laparotomic late (after day 21-t), while talking about primacy of antibiotic therapy in preventing pancreatic necrosis infection of acute pancreatitis and prevention with through appropriate antibiotics and efficient surgical treatment of infected pancreatic necrosis.

Is preferred delayed necrosectomy approximately after the 21-t day because of the following circumstances exist: create better lines of demarcation to distinguish necrotic tissue, operated on a patient stable clinically after rehydration treatment, haemorrhage is low since it leaves only necrotic tissue, leaving only necrotic tissue while maintaining maximum normal pancreatic tissue, reduce local complications such as angio erosion or in the small intestine leading to postoperative hemorrhage or fistula creation. Treatment should be conservative as long as possible, especially during the first two weeks.

Conclusions

Gall stone and alcohol are the most common causes of acute pancreatitis. Ultrasound is the first step for diagnosing patients with acute pancreatitis. Management of acute pancreatitis is done while maintaining circulatory volume, e nteral nutritional therapy and begins with the identification of patients with severe forms

of acute pancreatitis. Always performed scanner with contrast and do not perform it in the first 72 hours. Resonance has been used to identify liquid content from solid peripancreatic collections. An attempt with percutaneous drainage or surgery is made in patients with clinical deterioration and multi organ failure, but not without being demarcated the necrotic pancreatic tissue. Full treatment of pseudocysts formed during the course of acute pancreatits is done by multidisciplinary teams consisting of surgeons, endoscopic specialists and radiologists. The best treatment of complicated pseudocysts with hemorrhage, fistulization, and/overinfection is the, surgical drainage through cyst-entero or cyst-gastro anastomosis.

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