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Case report ■

Early Peritoneal Lavage in the Treatment of Acute Pancreatitis - Case Report

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SUMMARY

Acute pancreatitis (AP) is an inflammatory process of the pancreatic parenchyma resulting in glandular autodigestion initiated by pancreatic enzymes. At the onset of AP, parenchymal edema and adiponecrosis occur (acute edematous pancreatitis). If necrosis spreads to the parenchyma, accompanied by hemorrhage and dysfunction of the gland, the condition progresses to the stage of hemorrhagic and necrotizing pancreatitis. The only way of treatment of severe forms was surgery for a long period of time. Nowdays, 80% of patients are treated with conservative therapy.

The aim of our study was to establish weather early peritoneal lavage (EPL) as minimally invasive procedure can significantly reduce mortality among patients with AP. Prospectively, we analyzed the patients treated at Surgery Clinic in Niš during the year 2005.

During this period, we treated six patients for severe hemorrhagic and necrotizing pancreatitis using EPL. In this paper, two cases in detail are reported. In addition to intensive therapy and reanimation, EPL was performed in the first 48-72 hours from the onset of disease. After abdominocenthesis, two drains were placed in the abdominal cavity: the first into the left subphrenial space and the second into the Douglas recessus. A continues lavage was performed 24h a day during 3-5 days, using solutions for peritoneal dialysis (Peristeril).

All patients treated with EPL survived, developing well-known complications of AP like pancreatic pseudocyst and chronic pancreatitis.

The mortality rate in cases of mild clinical forms has fallen to 5% (3.8-7%), while it is still high in cases of severe forms, about 20% (15-25%). In the first week of disease, most of deaths occurred as a result of multiple organ failure, while in upcoming stages of disease infection played a bigger role. The key point of treatment is to prevent development of systemic inflammatory response and MOF in the first three days. By means of peritoneal lavage, toxins are eliminated from the abdominal cavity, which is the main factor for successful treatment of AP.

Key words: **acute pancreatitis, peritoneal lavage**

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INTRODUCTION

Acute pancreatitis (AP) is an inflammatory process of the pancreatic parenchyma resulting in glandular autodigestion initiated by pancreatic enzymes. The pancreas constitutes 0.1% of the total body weight (BM) and has 13 times higher protein output than the liver and reticuloendothelial systems combined (4% BM). Owing to the fact that pancreas is located in the retroperitoneum and does not possess a capsule, the inflammatory process spreads easily and rapidly. At the onset of AP, parenchymal edema and adiponecrosis occur (acute edematous pancreatitis). If necrosis spreads to the parenchyma, accompanied by haemorrhage and disfunction of the gland, the condition progresses to the stage of haemorrhagic and necrotising pancreatitis.

Incidence: Documented results vary. In the USA, the incidence of AP is 19.5, while 8.3 out of 100.000 patients are registered with chronic pancreatitis (1). Registered cases of AP in 1993 amounted to 215.000, while in 1998 the number was 183.000. In Germany, the incidence rate is 17.5 patients in the population of 100.000, while in Finland 73.4:100 000 (2). AP is 3 times more frequent in black people compared to white people (20.7 versus 5.7 patients in a population of 100.000). AP is also more frequent in men than in women.

Mortality: The total mortality rate in patients with mild clinical forms of AP is 15%, while in patients with severe forms of the disease the mortality rate amounts up to 30% (3). During the past 20 years, owing to aggressive intensive care treatment and the introduction of new therapeutic strategies, the mortality rate in cases of mild clinical forms has fallen to 5% (3.8-7%), while it is still high in cases of severe forms, about 20% (15-25%) (3).

During the first week following the onset of the disease, most cases of patient death are a result of multiple organ dysfunction, while in later stages infection is the main risk factor for a fatal outcome. However, destruction of the pancreas is the main cause of death.

CASE REPORT

We present the case of the patient S.B., male, aged 41 years, admitted complaining of abdominal pain which started the day prior to hospitalisation, increasing in intensity, accompanied by nausea, vomiting. He had a temperature of 37.8°C, puls of 93 beats/min and blood pressure of 170/105 mmHg. He was a smoker and had no family history of pancreatitis.

On physical examination he was conscious, oriented, with pallour of skin and mucoses, respiratory rate was normal, he had a fever and moderate epigastric tenderness without peritoneal signs.

Laboratory findings on admission (2nd day of illness): The red-cell count was $5{,}25x10^{12}$/L, white-cell count was $20{,}2x10^{9}$/L. The glucose level 13 mmol/L, calcium 2,29 mmol/L, serum amylase level was 4035.0 U/L, urinary amylase level was 41200 U/L.

Laboratory findings on admission (2nd day of illness): the red-cell count was 5 250 000, white-cell count was 20 200. The glucose level 13, calcium 2.29, serum amylase level was 4035.0, urinary amylase level was 41 200.

Based on the clinical examination, laboratory and ultrasonography findings, an intensive care treatment was initiated. During hospitalization, the patient's vital signs were continuously monitored, as well as laboratory follow-up with control ultrasonography readings and CT scans.

Based on the above results, the presenting patient's pancreatitis can be scored according to the Balthazar scale: Gr. D/E, Ranson's score: II/III (Table 1).

Initially, on admission, the serum alanine and aspartat aminotransferase levels were elevated, conjugated bilirubin was 80 μmol/L, urea and creatinine discretely elevated. The red-cell count showed a tendency to fall during disease progression- 3000000, hematocrit was 31%, sodium 131 mmol/L, K 3.2 mmol/L, Cl 90 mmol/L (Figures 1, 2).

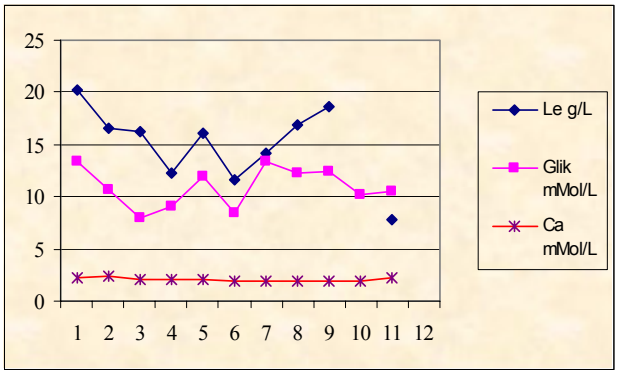


Figure 1. *White-cell count, serum glucose and calcium levels (according to days of disease)*

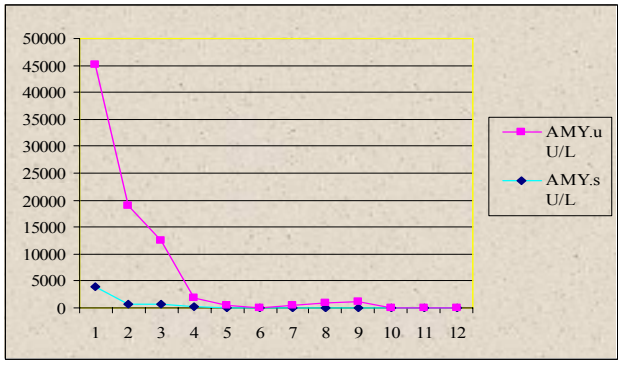


Figure 2. *Serum and urinary amylase levels*

Table 1. *Screening laboratory findings*

Day	White-cell	Glukose mMol/L	Amylase(s) U/L	Amylase(u) U/L	Ca mMol/L	Ta mmHg	Puls /min.	Temp. °C
II	20.2	13.4	4035.0	41200.0	2.29	170/105	93	37.8
III	16.6	10.7	696.0	18370.0	2.35	200/120	120	37.5
IV	16.3	8.0	692.0	11740.0	2.12	210/120	140	38.2
V	12.2	9.1	220.0	1520.0	2.0	180/100	90	37.6
VI	16.08	11.9	106.0	437.0	2.0	190/130	85	37.7
VII	11.7	8.5			1.92	170/90	80	36.5
VIII	14.1	13.4	73.0	496.0	1.90	170/110	110	37.5
IX	16.9	12.3	76.0	772.0	1.94	150/100	90	37.6
X	18.7	12.5	98.0	963.0	1.94	140/90	80	36.5
XI		10.2	89.0		1.86	130/90	85	
XVI	7.8	10.5	72.0		2.22			

Abdominal ultrasonography screening

Within 48 hours after presentation: microlithiasis within the gallbladder; extrahepatic bile-ducts normal; pancreas was swollen, edematous, the anteroposterior diameter of the pancreatic head was 35 mm, while that of the body 25 mm.

72 hours after presentation: pancreas was swollen, edematous, with wavy contours.

3rd day after presentation: Pancreas was swollen, wavy outer contours. The anteroposterior diameter of the pancreatic head was 50 mm. A moderate amount of fluid was present in the Bursa omentalis, subhepatic and Morrison's space. Douglas remains unchanged.

3rd day after presentation: Pancreas with minor transonic zones (up to 4 mm), resulting from necrosis and haemorrhage. A large fluid collection was present in the Bursa omentalis, subhepatic and Morrison's space. Douglas without change.

4th day: Pancreas was 35 mm in diameter, of semiliquid consistency. Peripancreatic large fluid collection was registered approximately 95x42 mm in size and also subhepatically.

Day 6: Pancreatic head measures 41 mm in diameter, the body 27 mm. Analysis of organ structure from the head towards the body reveals an area with liquid and solid content measuring 53x28 mm. Fluid content is detected in the subhepatic, Morrison's, para/prevesicular space and among the intestinal folds.

Computed tomography of pancreas

3rd day after presentation: pancreatic head 45 mm in diameter, tail 35 mm. Parenchyma in the region of the pancreatic head not homogenous. In the pancreatic head, a thick fluid collection was revealed and areas of necrosis occurring periferally and centrally.

Day 8: pancreatic head 49 mm in diameter, tail up to 53 mm. Pancreatic parenchyma more homogenous compared to the previous finding, with no evidence of necrosis; however, a fluid collection was registered.

Day 16: pancreatic head was of greater dimensions, not homogenous. Uncinat processus was 35 mm in diameter with a pseudocyst 40x7 mm present, spreading to the head and neck. No fluid collections were detected in the abdominal cavity.

TREATMENT

On admission (2nd day of the clinical condition), intensive care treatment was undertaken including the application of parenternal solutions, antibiotics (III

generation cephalosporins), spasmolytics, analgesics, somatostatin, ranitidine, as well as symptomatic cardiovascular therapy. Oral intake was withheld, a nasogastric tube was placed, diuresis monitored.

Following ultrasonography and CT on the 3rd day following onset of the disease, peritoneal lavage (within 72 hours of admission) was started. It was performed according to standard principles of peritoneal dialysis. Two drainage tubes were placed: one in the Bursa omentalis, the other in Douglas's space. Before the commencement of lavage, the content drained from Douglas's space is biochemical analysis: amylase level 12890.0 U/L; Na 139.0 nmol/L; Ca 1.70 mmol/l; Cl 103 mmol/L. For the purpose of lavage Peristeril solution 2500 L/2h was used. During lavage of Douglas's space, a change in the macroscopic aspect of the drain effluent was observed, whereas after 12 hours it was clear in appearance.

Abdominal cavity lavage lasted four days. The quantity of effluent drained from Douglas's space diminished over time. Drainage tubes were removed on the 7th postoperative day.

The general condition of the patient, laboratory and ultrasonography findings indicate that if peritoneal lavage is undertaken within the first 72 hours, this will induce a more rapid reduction in the level of urinary and serum amylase, white cell count and promote an improvement in the patient's clinical condition irrespective of the fall in red cell count, calcium, potassium and chloride levels.

Length of hospital stay was 17 days, during which there was no need for open drainage of the pancreas and necrosectomy. Control follow-up was scheduled five days later. The general condition of the patient and laboratory findings were within normal range. After the first control visit, on the 11th day following discharge, the patient was once again admitted to hospital due to epi/mesogastric pains accompanied by a fair increase in serum amylase. Substitutional, supportive treatment, as well as prophylactic antibiotics, analgesics and spasmolytics were administered. Ultrasonography did not show any significant changes to the pancreas. The second hospitalisation lasted eight days.

Patient follow-up after hospital discharge was carried out regularly, every 5-8 days. The general condition of the patient was good, laboratory and ultrasonography findings within normal range.

Six months later, the patient once again presented with epi/mesogastric pains. Laboratory analyses revealed a moderate rise in white cell count, blood glucose, urea, creatinine and serum amylase (about 112 U/L). Ultrasonography and CT scan revealed the presence of pseudocysts in the pancreas (Figure 3).

This complication following severe acute pancreatitis (necrotising-haemorrhagic) is frequent and to be expected. The patient was treated conservatively: oral intake was withheld, fluids and electrolytes were supplemented (crystalloid solutions), analgesics, spasmolytics, somatostatin and antibiotics (III generation cephalosporins) were administered. The patient's vitals were continually monitored. Contol ultrasonography was performed every other day, a control CT scan on the 6th day. Following conservative treatment a very mild improvement of the patinet's clinical condition and laboratory findings was noted, except for serum amylase which showed a tendency to rise. This was the reason why we decided to undertake percutaneous drainage of the pancreatic pseudocysts (Figures 4, 5, 6).

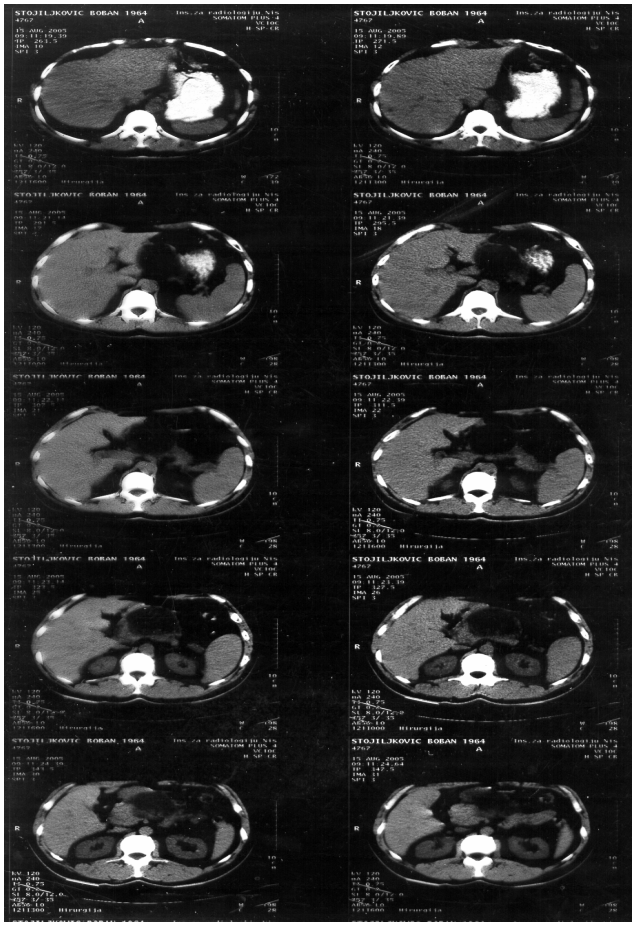
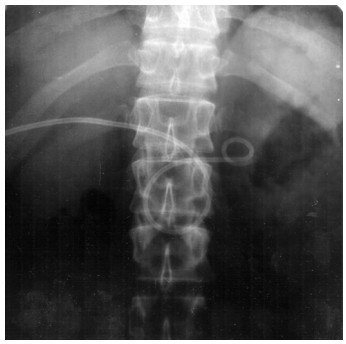
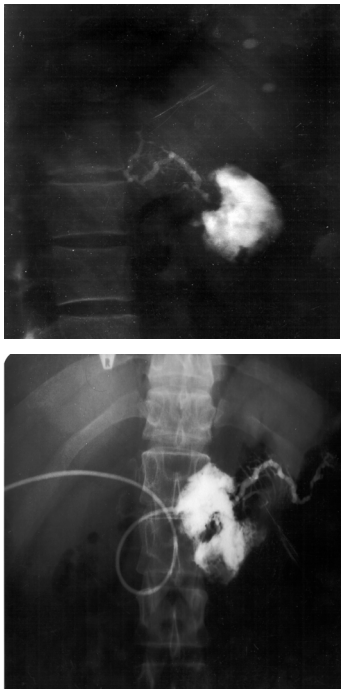


Figure 3. *CT scan - presence of pseudocysts in the pancreas*





Figures 4, 5 and 6. *Percutaneous drainage of the pancreatic pseudocysts*

Percutaneous drainage lasted eight days, however, the amount of evacuated fluid was constant on all days. The pseudocysts did not show a tendency to regress, nor was there any improvement in follow-up laboratory analyses. Surgical treatment of the pseudocysts with cystojejunostomy according to Brown was performed. The abdominal cavity was drained threefold. The postoperative period went well. Laboratory findings, control ultrasonography and CT scans showed great improvement as early as the second postoperative day. The patient was discharged from hospital on the 11th postoperative day in a good general state.

b. Case report of patient T.B., male, aged 43 years, worker, who was first admitted to hospital due to a severe form of pancreatitis (haemorrhagic-necrotising), where clinical and laboratory findings, as well as ultrasonography readings were similar to the previous patient. Substitutional and pharmacotherapy, supportive treatment, intensive care monitoring were undertaken immediately following hospitalization. Peritoneal lavage and drainage were carried out within the first 48 hours following the onset of abdominal pains. Lavage accompanied by drainage lasted six days. During lavage, serum and urinary amylase levels, white cell count and blood glucose returned to normal values.

Control abdominal ultrasonography and CT of the pancreas showed a moderate enlargement of the pancreas with no visible pathological features. The length of hospital stay was 12 days.

The patient's 2nd hospitalization lasted longer due to complications arising from the acute haemorrhagic-necrotising pancreatitis. The patient was admitted to our clinic complaining of epigastric and mesogastric pains, weakness, fever (up to 38 ^{0}C), nausea, vomiting. Laboratory findings on presentation were Le 18 000, glucose 10,6; proteins 50, serum amylase 46; Na 132; Ca 1,95; Cl 2,2. On admission, all oral intake was withheld, a nasogastric tube placed, fluids and electrolytes supplemented, and treatment with analgesics, spasmolytics, somatostatin and antibiotics (III generation cephalosporin combined with metronidazol) started.

Monitoring of the patient's vital signs was constant. One month later abdominal ultrasonography revealed: a swollen pancreas with the head more enlarged - 37 mm than the body - 25 mm; presence of fluid collection in the peripancreatic area - 97x36 mm, as well as in the pelvic cavity - 85x65 mm. Administered conservative treatment did not yield any improvement, the patient had a persisting fever.

Abdominal CT revealed a fluid collection in the abdominal cavity, retroperitoneum, exudates in the bursa omentalis, paracolic and subhepatic areas. The pancreas was swollen, edematous with the presence of peripancreatic exudate.

Drainage of the abdominal abscess was undertaken using the methodology according to Seldinger. In the abscessal cavity located pararenally, an 8Fr catheter was placed. The catheter drained 600 ml/24h purulent effluent. Drainage lasted 4 days, without a reduction in the quantity of the effluent drained.

After drainage procedure, a control CT scan of abdomen was performed: exudate was still present in the Bursa omentalis, peri/subhepatic space, in the perisplenic and peripancreatic spaces. A capsulated collection had developed in the projection of the linea alba, approximately 10x9 cm in size, which was subjected to percutaneous drainage. After control imaging with urografin, the location of the drainage catheter was adjusted. The cavity was washed out with saline solution and iodine in the ratio 5:1. The same procedure was applied to the previous pararenal abscess in the left pararenal space, by means of another drainage catheter. Treatment was modified by the addition of semisynthetic penicillins, antibiotics were given in larger doses, parenteral intake of fluids was increased, electrolyte disbalance was corrected.

The general condition of the patient following these surgical procedures showed an improvement, he no longer had a fever, an increase in the red cell count and a decrease in white cell count were evidenced, as well as a normalisation of the electrolyte status, serum urea and creatinine. The drain effluent became serous clear in appearance and less in quantity over time.

CT scan after one week shows the regression of fluid in the abdominal cavity and normalization in pancreatic size. The patient was in good clinical condition on discharge two days following the removal of drainage tubes.

DISCUSSION

The clinical course of AP varies from a mild, transitory illness to severe, rapid, fatal disease. Severe forms of AP have poor prognosis and high mortality rate. The disease goes through two phases. The first days of this clinical condition are characterized by systemic inflammatory response syndrome (SSIO). The second phase arises within two weeks following the onset of the disease, with the domination of septic complications which are the result of infection and pancreatic necrosis. This type of infection occurs in 40-70% patients and represents the most important risk factor for a fatal outcome. About 20% patients who go through the SSIO phase die due to septic complications (infected pancreatic pseudocyst, pancreatic necrosis or multiple organ dysfunction).

Even though the aethiologic factors of AP are numerous, in 10-30% of patients, the cause remains unknown. The most commonly known cause is a biliary tract disorder (38%): due to retention of a gallstone in the Oddy sphincter resulting in obstruction of the pancreatic duct, while microlithiasis leads to idiopathic AP.

Abnormalities of the bile ducts (cyst of the choledoch duct, jugstapapilary diverticulum) can also lead to AP. Alcohol abuse is the second most frequent cause of AP (35%), even though it takes a leading place in developed countries. On the cellular level, alcohol causes intracellular accumulation of digestive enzymes and their increased activity. Alcohol also increases the level of proteins in the pancreatic juice and permeability of the ducts, leading to transfer of pancreatic enzymes to the pancreatic parenchyma resulting in its destruction, but reduces the level of bicarbonates and tripsin inhibitor.

Less common causes are: post - ERCP (about 4%), trauma (up to 1%), drugs (up to 1,4%), infection (up to 1%), hereditary pancreatitis (up to 1%), hypercalcemia (up to 1%), various abnormalities of the pancreas (up to 15%), hypertriglyceridemia (up to 1%), tumors (up to 1%), toxins (up to 1%), postoperative AP (up to 1%), idiopathic AP (about 10%) (2).

A number of mechanisms are responsible for the onset of pancreatic autodigestion. Proteins are converted to inactive proenzymes, while cellular Golgi bodies segregate into separate subcellular parts. Lyzozome enzymes are necessary for intracellular recycling, while enzymes of the zimogenic granules are transported to the intestine during digestion of food. Zimogenic granules have acidic pH and a low calcium concentration which prevent untimely secretion of pancreatic enzymes, and they represent the extracellular promoters of the activation cascade.

Under the influence of various factors, these defense mechanisms are inhibited, which leads to intracellualr activation of enzymes resulting in pancreatic autodigestion and AP. The presence of cytokines (bradikinin and phospholypase A) leads to greater vascular permeability and vasodilation, pain and adhesion of leucocytes to the walls of blood vessels. Necrosis of adipose tissue (peripancreatic fat) can lead to hypocalcemia, while the destruction of pancreatic B cells leads to hyperglycemia.

Diagnosis: Before AP can be diagnosed with certainty, apart from data obtained during history taking and physical examination, the following must be analyzed everyday:

1. Laboratory findings: white cell count (over 12 000) and red cell count (shows a tendency to fall in cases of haemorrhagic pancreatitis), blood glucose (elevated when necrosis of pancreatic B cells occurs), fall in electrolyte levels (Na, K, Ca, CO_2, P, Mg) due to translocation of fluid to the third compartment, increase in serum amylase (if the serum concentration at least triples, this is a sure sign of AP), lipase is high during the first 12 days, especially in chronic pancreatitis due to alcohol abuse, while hyperbilirubinaemia, elevated AST and ALT levels are indicators of biliary pancreatitis.
2. Abdominal and pancreatic ultrasonography as a screening test.
3. Abdominal computed tomography is most important in diagnosing AP.
4. Criteria for ranking AP according to its severity vary. According to Balthazar 5 levels/grades exist:

Grade (level) A - normal pancreas

Grade B - focal or diffuse pancreatic inflammation

Grade C - Pancreatic inflammation and peripancreatic enlargement (intrinsic abnormality of the gland).

Grade D - Peripancreatic fluid collection - localized collection or phlegmona.

Grade E - 2 or more peripancreatic collections and/or retroperitoneal presence of air (Figures 7, 8 and 9).

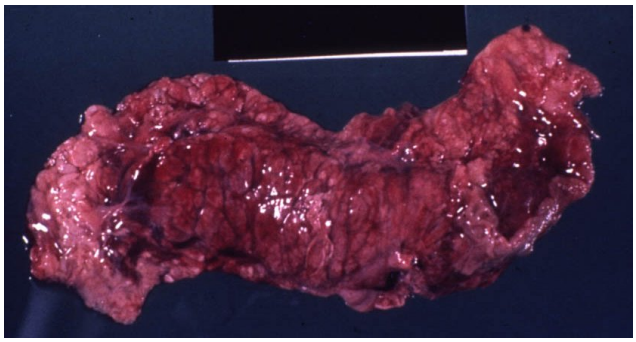


Figure 7. *Diffuse pancreatic inflammation*

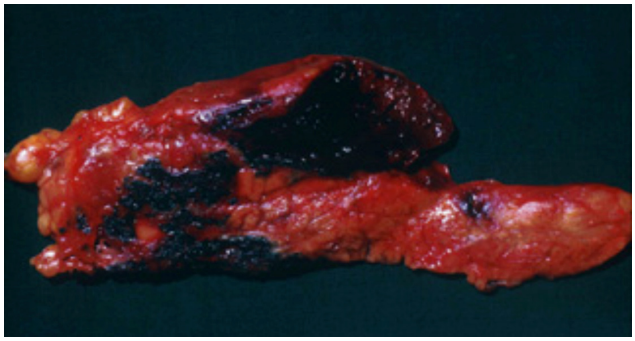


Figure 8. *Pancreatic-necrotising-haemorrhagic presentation*

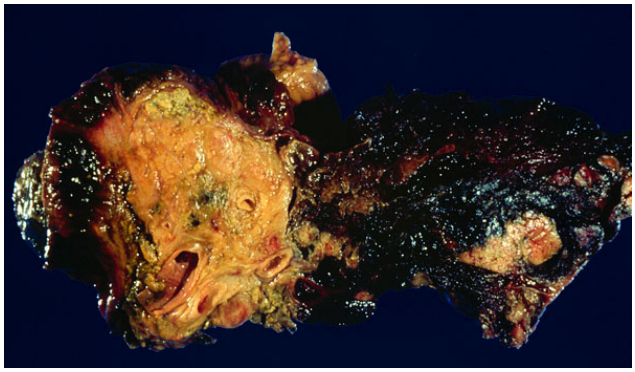


Figure 9. *Pancreatic necrosis*

According to some authors, valid prognosis cannot be based on these criteria, whereby they recommend determining the severity of the disease based on the extent of necrosis:

Ranking AP	Scoring	Extent of necrosis	Scoring
Gr.A	0	Absence of necrosis	0
Gr.B	1	30% necrosis	2
Gr.C	2	50% necrosis	4
Gr.D	3	> 50%	6
Gr.E	4		

Scoring	Mortality	Morbidity
0-1	0%	0%
2-3	3%	8%
4-6	6%	35%
7-10	17%	92%

Today, clinical systemic scoring methods are mostly used (based on various biochemical parameters, APACHE-II, APACHE-III) to classify the severity of the illness (4).

Treatment: in 80% cases it is conservative and comprises the following:

- Supplementing fluid under constant monitoring of input/output of fluid, and maintaining an electrolyte balance; crystalloid solutions, other parenteral solutions, blood transfusion, especially in cases of haemorrhagic pancreatitis;
- Enteral feeding through a nasoenetric tube aimed to reduce gastrointestinal secretion;
- Endoscopic placement of nasopancreatic tube within the first 48h following the onset of the condition accompanied by drainage of pancreatic juices (5);
- Administration of analgesics, especially meperidine (Demerol 15-35mg iv. Or 50-150mg/3-4 im.) due to its spasmolytic effect on the Oddy sphincter;
- Administration of antibiotics, in severe forms of AP complicated by septic shock or when CT shows a pancreatic phlegmona. III generation cephalosporins are recommended (ceftriaxone 2g im./iv.) in combination with ampicillin (Marcillin 500 mg/6h);
- Somatostatin (octreotide) reduces mortality;
- Gabexate mesilate in preventing complications with no effect on mortality (6);
- Continous oxygen supply in case of acidosis; intubation - in cases of tachipnea and respiratory insufficiency;
- Total parenteral nutrition, in cases with bad prognosis or if no clinical improvement is observed after four days of treatment;
- ERCP, if removal of gallstones from the duct is required;
- Aspiration of necrotic tissue under CT guidance, if necessary;
- Peritoneal lavage within the first 72h of disease onset (laparoscopic closed peritoneal lavage). This period is regarded as optimal since cytokine secretion does not reach peak values in the first 48 hours, thereby lavage carried out in this interval can prevent the development of SSIO and MODS (multiple organ dysfunction syndrome).

According to clinical investigations, early debridment and drainage of necrotic tissue or inflammatory exudate increases mortality rate in severe cases of AP. However, in cases of extensive pancreatic necrosis, early peritoneal lavage and drainage are able to maintain an intact pancreas and drain the peripancreatic area, which considerably improves outcome (7).

Surgical interventions are indicated in:

- Patients who did not show an adequate response to total conservative treatment and peritoneal lavage and are above index 5 according to Ranson;
- In cases of haemorrhagic pancreatitis, where chemostasis is needed, especially when large blood vessels are damaged with accompanying bleeding;
- In cases of biliary pancreatitis, where sphincterectomy or cholecystectomy is indicated (1).

Early open surgery with drainage and necrosectomy leads to greater damage of the pancreatic parenchyma, requiring repeated necrosectomy (circulus vitiosus) and is accompanied by a high mortality rate.

Ranson suggested a series of different criteria that should be followed during the clinical course of acute pancreatitis (over the years some authors dismissed these criteria, while others have over time requested their reintegration):

Onset of the disease:
Patient age >55 years
White cell count >16 000/mcl
Blood glucose >200/dl

Lactate dehydrogenase >350 IU/l
AST >250 IU/l
Course of the disease during initial 48 hours:
Absolute decrease in hematocrit >10%
Decrease in BUN >8mg/dl
Decrease in serum calcium <8mg/dl
Arterial oxygen saturation <60 mmHg
Base deficit >4mEq/l
Fluid sequestration >6000 ml

According to Ranson's scale patients who have at least two criteria do not require intensive and aggressive treatment (minimal mortality rate); those who fulfill 3-5 criteria require intensive care treatment of acute pancreatitis (mortality rate is 10-20%), while those with more than 5 criteria present, apart from intensive care management require urgent surgical treatment (mortality rate in this group is above 50%).

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RANA PERITONEALNA LAVAŽA U LEČENJU AKUTNOG PANKREATITISA - PRIKAZ SLUČAJA

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Sažetak

Akutni pancreatitis (AP) je zapaljenski proces parenhima pankreasa koji dovodi do glandularne autodigestije uzrokovane enzimima pankreasa. Kod početnih oblika AP, javlja se parenhimalni edem i adiponecroza (acute edematous pancreatitis). Ukoliko je nekroza parenhima pankreasa brza, zbog hemoragije i disfunkcije žlezde, ovakav pankreatitis progredira u stanje hemoragičnog i nekrotizirajućeg oblika pankreatita. Dugi niz godina, jedini način lečenja teških AP bio je hirurški. Danas preovlađuje stav da je lečenje ovakvih pankreatitisa kod 80% bolesnika konzervativno.

Cilj našeg ispitivanja bio je utvrditi da konzervativni način lečenja AP uz primenu rane peritonealne lavaže (RPL), kao minimalno invazivne tehnike, može dovesti do značajnog smanjenja mortaliteta ovih bolesnika. Ispitivanje predstavlja prospektivnu studiju bolesnika lečenih 2005. godine na Hirurškoj klinici u Nišu.

Na našoj Klinici, tokom 2005. godine, konzervativno je lečeno svega šest bolesnika sa teškim hemoragično-nekrotičnim AP. U ovom radu prikazali smo detaljno lečenje dva bolesnika. Kod svih bolesnika, pored mera intenzivne nege i reanimacije, pristupilo se RPL u toku prvih 48-72h od početka bolesti. Kroz abdominocentezu su postavljena dva drena: 1 u levi subfrenični prostor a drugi u Duglasov špag. Lavaža je vršena kontinuirano 24h tokom 3-5 dana rastvorima za peritonealnu dijalizu (Peristeril).

Svi bolesnici lečeni ovom metodom su preživeli uz pojavu poznatih komplikacija koje nastaju nakon AP: pseudocista pankreasa, hronični pankreatitis.

Stopa mortaliteta u slučajevima srednje teških AP je opala i kreće se oko 5% (3.8-7%), dok je i dalje značajno visoka u slučajevima teških oblika oko 20% (15-25%) (3). U prvoj nedelji bolesti, većina smrtnih ishoda je rezultat multiorganskog sistemskog popuštanja, dok u sledećim fazama bolesti infekcija igra mnogo značajniju ulogu, mada je oštećenje pankreasa glavni uzrok smrti. Sprečiti u prva tri dana razvoj Sindroma sistemskog inflamatornog odgovora (SSIO) i MOF-a upravo RPL kojom se otklanjaju toksini iz abdomena ključni je faktor u terapiji teških oblika AP.

Ključne reči: **akutni pankreatitis, peritonealna lavaža**