Case report **UDC: 616.37-002.1:616.37-003.4-073**

PANCREATIC PSEUDOCYST AS A COMPLICATION OF ACUTE ALCOHOLIC PANCREATITIS – CASE REPORT

Snežana Tešić-Rajković[1], Biljana Radovanović-Dinić[1] and Miroslav Stojanović[2]

Pancreatic pseudocysts are localized collections of fluid enclosed by fibrous wall, which may arise in association with acute or chronic pancreatitis, pancreatic trauma, or pancreatic duct obstruction. During the evolution of a pancreatic pseudocyst, a rupture may occur as an acute complication. The basic diagnostic procedures which allow visualization of the pseudocyst are ultrasonography and computed tomography (CT). We report the clinical, ultrasonographic and CT presentation of the pancreatic pseudocyst in a 41-year-old man suffering from alcoholic pancreatitis. *Acta Medica Medianae 2010;49(2):44-47.*

Key words: *pancreatitis, pancreatic pseudocyst, abdominal ultrasonography, computed tomography*

Gastroenterology and Hepatology Clinic, Clinical Center Niš, Serbia[1]
Surgery Clinic, Clinical Center Niš, Serbia [2]

Contact: Snežana Tešić Rajković
Klinika za gastroenterologiju i hepatologiju
Klinički centar u Nišu
Bul. dr Zorana Đinđića 48 Niš
E-mail: snezanatesic@yahoo.com

Introduction

Pancreatic pseudocysts are localized collections of fluid enclosed by non-epithelized wall of fibrous or granular tissue that may arise in association with acute or chronic pancreatitis, pancreatic trauma or pancreatic duct obstruction. Pseudocyst may be localized within the pancreas or in its immediate vicinity in the small omentum, although there is a possibility of spreading to the neck, mediastina, pelvis and scrotum (1).

Pseudocysts usually develop in patients with alcoholic pancreatitis. In countries where alcohol consumption is high, in 59-78% of patients pseudocysts develop along with alcoholic pancreatitis (2).

Pancreatic pseudocysts can be asymptomatic, however, they can often be manifested by persistent abdominal pain, anorexia, nausea and vomiting. The existence of pancreatic pseudocysts should be suspected of in the case of verifying the palpable tumor in the epigastric or left upper quadrant, four weeks after the attacks of acute pancreatitis (3). During the evolution of pseudocysts, acute complications are possible in the form of acute hemorrhage (usually from pseudo aneurysm arising from splenic artery), infection, penetration into the spleen and liver and rupture. Chronic complications include gastric obstruction, biliary obstruction and lienal thrombosis or portal vein with development of gastric veins varicosity (4). Basic diagnostic procedures which allow visualization of a pseudocyst are ultrasonographic (US) abdomen examination and computed tomography (CT).

Case report

A 41-year-old patient M.D., a musician, was admitted to the hospital for intense pain in the bowel and under the right rib arc. The pain occurred a few days prior to admission and was intensified at breathing in and movement. He said that for the previous 5 years he had suffered from high blood pressure which had been poorly regulated. He is a perennial smoker, smokes a pack of cigarettes per day. He has regularly consumed alcohol for years, about 100g a day, sometimes much larger quantities.

On admission, the patient was oriented, afebrile, eupnoic, of medium osteomuscular build and moderate nutrition status. Skin and visible mucous membranes are normally colored, the action of the heart rhythmic with occasional extrasystoles, sounds clear, without accompanying noise TA-150/120 mmHg. Abdomen is in the plane of the chest, palpatory tender, moderately painfully sensitive in the left hypochondrium, superficially and at deep palpation, without signs of peritoneal irritation. The liver and spleen are within physiological limits. Renal succession is negative. Extremities without swelling ECG: sinus rhythm, with occasional VES, SF 83/min. Laboratory findings indicate the existence of leucocytosis $11.4 \cdot 10^9$ /L, increased values of urea 10.8 mmol/L and creatinine 155.9 mmol/L, normal values of serum transaminases, high serum amylase values 980.4 U/L respectively, urine 6440 U/L, slightly higher levels of serum triglycerides 2.27 mmol/L, gamma GT 30 U/L. Sedimentation rate was 20 in the first hour, the value of C reactive protein 54 mg/L. Abdominal ultrasonography validated regular clinical appearance of the liver, gallbladder, head

and body of the pancreas and spleen. Both kidneys were with polycystic changes, of vague contours. The largest cyst in the right kidney was 89mm, and 69mm in the left. Free fluid was not verified in the abdominal cavity and / or pelvis. A plain film of the abdomen was within normal limits. The patient was discharged from hospital after seven days with a recommendation to comply with the hygienic-dietetic regime and to apply the therapy.

After one month, the patient was again admitted to hospital for severe abdominal pain. The pain began in the bowel, but quickly spread to the entire abdomen and both loins, accompanied by sensation of being short of breath, choking and unformed bowel movement without blood and mucus. During the physical examination, high blood pressure 175/120 mmHg was verified as well as palpatory diffuse abdominal pain sensitivity to deep palpation, with no signs of peritoneal irritation. The liver and spleen were within normal limits. Laboratory findings showed leucocytosis $8.7 \cdot 10^9$ /L, high values of serum amylase 1231 U/L respectively, in the urine of 6030 U/L, increased values of urea 10.8 mmol/L and creatinine 164.7 mmol/L. CRP was 38.7 mg/L. Radiography of the heart, lungs and abdomen showed normal findings. The ultrasonographic examination of the abdomen verified the existence of free fluid in the abdominal cavity. The patient was observed with the diagnosis *Pancreatitis acuta, Policystismus renii bill. et Hypertensio arterialis.* With the assigned therapy accompanied with compensation of fluids, analgetics, proton pump blockers and vitamins, a satisfactory symptomatic effect was reached and the patient was discharged after 7 days to home treatment with the recommendation to apply the therapy, diet and have regular examinations by a gastro-entero-hepatologist and nephrologist.

However, after two weeks, the patient was again hospitalized at the Clinic for Gastro-enterology and Hepatology because of abdominal pain that began 4 days prior to admission. The pain was intense, localized throughout the abdomen and spread in a form of a belt. On admission, the stomach was below the plane of the chest, palpatory tender, painfully sensitive to deep palpation in the epigastrium, without signs of peritoneal irritation. The liver and spleen were within normal limits, with negative renal succussion. Findings of extremities were normal.

Laboratory findings showed: leukocytosis $11.1 \cdot 10^9$ /L, elevated values of serum amylase 925 U/L and urine of 861.1 U/L, increased urea 10.4 mmol/L, creatinine 164.9 mmol/L and elevated C-reactive protein 152.5 mg/L. Ultrasonography of the abdomen verified the normal-sized liver with a liquid collection in the VIII segment (cyst?) (Figure 1), gall bladder with echogenic change without acoustic shadows (Figure 2). In the projection of the tail, a liquid collection 103.3x58.4mm was noticed (Figure 3). The spleen was voluminous, 141mm and homogeneous. Both kidneys were polycystically changed. The moderate amount of ascites was verified (Figure 4).

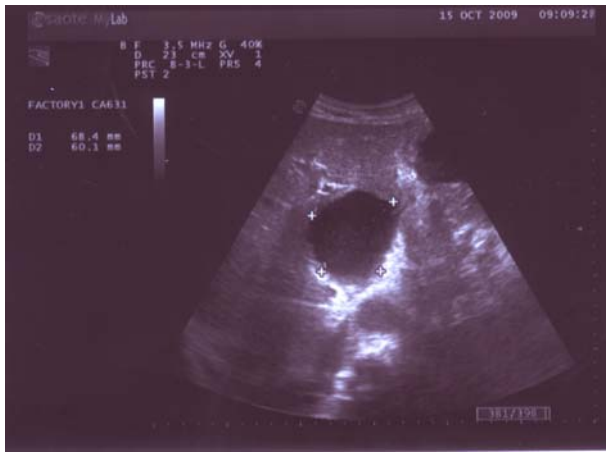

Figure 1.

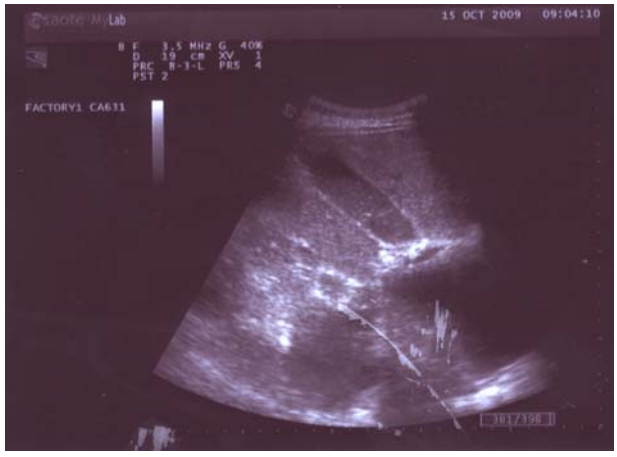

Figure 2.

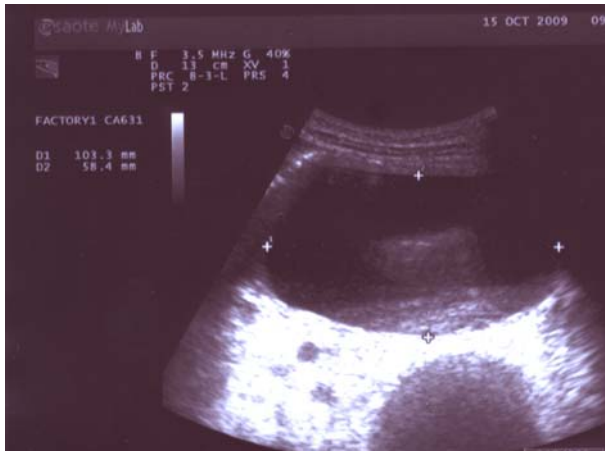

Figure 3.

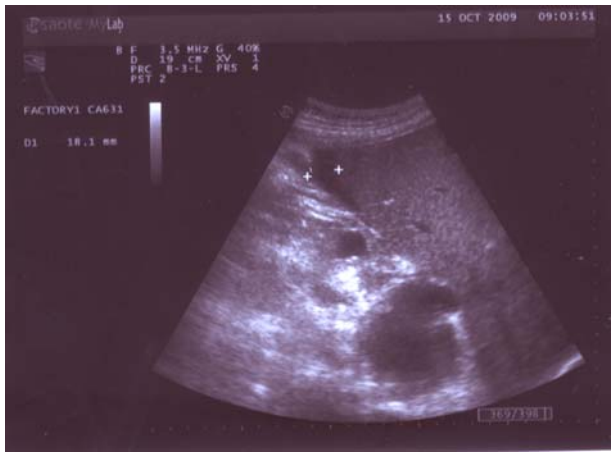

Figure 4.

Radiography of the heart and lung verified the presence of accentuated pulmonary vascular markings on both sides, with no collapse and consolidation of the parenchyma. The right costofrenic sinus was shaded.

At esophagogastroduodenoscopy the esophagus findings were normal. Lumen of the stomach was partially distorted with the impression of the external compression. Gastric mucosal tissue in general was slightly hyperemic with hypertrophic folds in the corpus. No erosion and ulceration and proliferation were verified. Pylorus was slightly distorted, and the bulbus and the postbulbar part of the duodenum were hyperemic, edematous mucosa with no erosion, ulceration and proliferation. At computed tomography (CT) examination of the abdomen, a cystic formation dimensions 76x46x42mm (KK LL AP) was observed in the liver lobe in caudatus, of densymetric values of tenous fluid. There was no dilatation of intra and extrahepatic biliary tract. The findings of gall bladder were normal. Pancreas morphology was normal, without focal lesions. Between the stomach, pancreas and spleen, in bursa omentalis, a large multilocular, barriered cystic change was found, size 84x62mm (measured in the axial plane), of densymetric values of tenous fluid. The spleen morphology was normal, without focal lesions. Polycystically changed kidneys were seen on both sides. Adrenal glands were of normal presentation. A free fluid was found in the abdominal cavity, in both subhepatic and perisplenic areas. No retroperitoneal linfoadenomegalia was verified. The native abdominal radiography detected the presence of certain quantities of gas in the intestines. After consultations with a hepatobiliary surgeon, the patient was tranferred to the clinic for a surgery under the diagnosis - Pseudocystis pancreatis.

Upon admission at the Department of Surgery, laboratory findings showed: leukocytosis $15.4 \cdot 10^9$ /L, with a predominance of neutrophils $7.8 \cdot 10^9$ /L, anemia $3.65 \cdot 10^{12}$/L, Hgb 7.9g/L. Increase of creatinine 128.4 mmol/L was verified, decrease in total protein 57.5 g/L respectively, albumin 32.1 g/L, increase in transaminases AST 54 U /L, ALT 64 U/L, increase in gamma GT 50.6 U/L, increased activity of amylase 211 U/L, increased C-reactive protein 78.7 mg/L and increased LDH 619.4 U/L. Two days later the patient suddenly felt severe pain in the abdomen, accompanied by sweating. Physical examination of the abdomen verified severe painful sensitivity of the entire abdomen with signs of peritoneal irritation. Increase in amylase activity in serum to 365 U/L was noticed, increase of CRP to 165.2 mg/L and fall of protein levels in serum of 55.4 g/L, respectively, of serum albumin at 5.28 g/L. Ultrasonography and native radiography of the abdomen verified a rupture of pancreatic pseudocysts. Incision was performed and a drain placed in the abdominal cavity, where the haemorrhagic content was obtained. The value of amylase in the contents of the tube was 4040 U/L. Intensive treatment with antibiotics, fluid and electrolyte supplement, human albumin and plasma transfusions were applied. Sandostatin ampoules were administered subcutaneously every 8 hours. Gradually, there was an improvement of general condition of the patient and laboratory findings, and the patient was discharged with the recommendation for routine examination by a hepatologist, i.e. hepatobiliary surgeon.

Discussion

During the evolution of pancreatitis of alcoholic genesis, a pseudocyst occurs with the pancreatic duct damage and the consequent extravasation of pancreatic secretions. The resulting liquid formed out of the pancreas causes inflammatory response, producing after several weeks the cyst wall composed of fibrous tissue and granulation tissue. The lack of epithelium in the wall is the characteristic of a pseudocyst (1). Thus originated pseudocysts usually contain fluid rich in enzymes and necrotic debris (2,5). The level of amylase and lipase in the pseudocyst fluid is significantly higher than the levels of these enzymes in the blood (2).

In the presented patient, there was a formation of a large pseudocyst of the pancreas after 4 weeks from the first attack of acute pancreatitis. Pseudocyst was localized between the pancreas, stomach and spleen in bursa omentalis, size over 8 cm with compression of the stomach. The disease was initially diagnosed echosonographically, then the exact localization of the pseudocyst and the anatomic relationship to other organs was confirmed during computed tomography. The clinical course of the presented patient, the information on alcoholism and the chronology of events with a worsening of symptoms 4 weeks after the first episode of acute pancreatitis are typical for pancreatic pseudocysts.

The US and CT examination of the abdomen are important both for initial diagnosis of acute pancreatitis and for its further evaluation in terms of developing possible complications such as pseudocysts (6). According to the established guidelines of the international symposium held in Atlanta in 1992, the initial CT should be done in: (a) patients in whom there is doubt regarding the clinical diagnosis of pancreatitis, (b) patients with clinically severe hyperamilasemia pancreatitis, abdominal distension, painful sensitivity of the abdomen, fever and leukocytosis, (c) patients with the Ranson score > 3 or APACHE score > 8, (d) patients who do not show rapid clinical improvement within 72 hours after initiation of the conservative medical therapy and (e) patients who showed improvement during the initial conservative treatment and subsequent deterioration in clinical status indicating the development of complications (7).

In detection of pancreatic pseudocysts, sensitivity of US and CT examination of the abdomen is 75-90%, i.e. 90-100%. For detection of pancreatic pseudocysts, CT is a better choice, because significant quantities of gases reduce the sensitivity of ultrasonographic examination. Identification of the thickened wall surrounded by

fluid collection in the vicinity of the pancreas on abdominal in CT findings, in patients with history of acute or chronic pancreatitis, is virtually pathognomonic for pancreatic pseudocyst (3). In addition, CT can provide detailed information about the surrounding anatomy and can show additional pathology, including dilatation and calcification of the pancreatic duct, dilatation of the common gall and spreading of pseudocysts omentalis outside bursa omentalis (8).

The evolution of a pseudocyst may lead to its spontaneous rupture, which is exactly what happened in the case of the patient presented. A large pseudocyst ruptured into the abdominal cavity, causing peritonitis. An urgent surgical intervention was performed with lavage of abdominal cavity and external drainage.

Rupture of the pseudocyst can have a favorable or unfavorable outcome, depending on whether there has been a rupture in the gastrointestinal tract, the peritoneal cavity or vascular system (9,10). Rupture of a pseudocyst into the gastrointestinal tract may be asymptomatic or with clinically overt bleeding in the form of melena or hematemesis. Rupture into the peritoneal cavity leads to peritonitis, which usually requires urgent surgical exploration during which lavage of the abdominal cavity and external drainage can only be safely applied (3).

Conclusion

Development of complications such as pseudocysts may be expected during the evalu-ation of patients with recurrent alcoholic pancreatitis. In such patients, regular clinical, laboratory and imaging methods of control are necessary in order to obtain timely diagnosis and apply therapeutic treatment to possibly manifested complications that often vitally threaten the patient.

References

1. Agarwal N, Pitchumoni CS. Management of Pancreatic Pseudocysts. Current Treatment Options in Gastroenterology 1999; 2:409–14.
2. Pitchumoni CS, Agarwal N. Pancreatic pseudocysts. When and how should drainage be performed? Gastroenterol Clin North Am 1999; 28: 615-39.
3. Habashi S, Draganov PV. Pancreatic pseudocyst. World J Gastroenterol 2009; 15(1): 38-47.
4. Lu X, Uchida E, Yokomuro S, Nakamura Y, Aimoto T, Tajiri T. Features and choice of treatment of acute and chronic pancreatic pseudocysts--with special reference to invasive intervention. Pancreatology 2008;8(1):30-5.
5. Nealon WH, Walser E. Surgical management of complications associated with percutaneous and/or endoscopic management of pseudocyst of the pancreas. Ann Surg 2005;241(6):948-57.
6. Podgurski L, Hou G, Shaffer K. CT Imaging of a Pancreatic Pseudocyst: Clinical and Anatomic Implications. Radiology Case Reports. [Online] 2007; 2:107.
7. Balthazar EJ, Freeny PC, van Sonnenberg E. Imaging and intervention in acute pancreatitis. Radiology 1994; 193 (2):297-306.
8. Balthazar EJ. Acute pancreatitis: assessment of severity with clinical and CT evaluation. Radiology 2002;223(3):603-13.
9. Martinez-Ramos D, Cifrián-Pérez M, García-Vila JH, Salvador-Sanchís JL, Hoashi JS. Percutaneous drainage treatment of traumatic pancreatic rupture with pancreatic transection. Gastroenterol Hepatol 2010; 33(2):102-5.
10. Mir MF, Shaheen F, Gojwari TA, Singh M, Nazir P, Ahmad S. Uncomplicated spontaneous rupture of the pancreatic pseudocyst into the gut--CT documentation: a series of two cases. Saudi J Gastroenterol 2009;15(2):135-6.
11. Gmijović D, Stojanović M, Višnjić M, Jeremić M, Stojiljković M, Stanojević G, et al. Palliatives of the surgical procedure in the treatment of non-resectable carcinoma of the pancreas head. Acta Medica Mediane 2002;41(4):81-9.

PSEUDOCISTA PANKREASA KAO KOMPLIKACIJA AKUTNOG ALKOHOLNOG PANKREATITISA – PRIKAZ BOLESNIKA

Snežana Tešić-Rajković, Biljana Radovanović-Dinić i Miroslav Stojanović

Pankreasne pseudociste su lokalizovane tečne kolekcije ograđene fibrinskim zidom, koje nastaju kao rezultat akutnog ili hroničnog pankreatitisa, traume pankreasa ili opstrukcije pankreasnog kanala. Tokom evolucije pseudociste pankreasa može nastati akutna komplikacija u vidu rupture pseudociste. Osnovne dijagnostičke procedure koje omogućavaju vizualizaciju pseudociste jesu ultrasonografski pregled abdomena i kompjuterizovana tomografija. Radom se ilustruje klinička, ultrasonografska i CT slika pseudociste pankreasa kod 41-godišnjeg bolesnika sa pankreatitisom alkoholne geneze. *Acta Medica Medianae 2010;49(2):44-47.*

***Ključne reči**: pankreatitis, pseudocista pankreasa, ultrasonografija abdomena, kompjuterizovana tomografija*