Acute Pancreatitis with Abdominal Bloating and Normal Transabdominal Ultrasound

Silvikarina Erfanti Dewi Halim
Division of Internal Medicine, Eka Hospital, Pekanbaru

Corresponding author:
Silvikarina EDH. Division of Internal Medicine, Eka Hospital. Jl Ir Soekarno Hatta, Tengkerang Baru, Pekanbaru. Phone: 07616989999. E-mail: silvikarinaerfantidewihalim@gmail.com

ABSTRACT

The diagnosis of acute pancreatitis (AP) is a topic of interest for clinicians, as it is an inflammatory disorder of the pancreas. A 44-year-old man presented with bloating for one month and it worsened one day before admission to the ER, accompanied by discomfort in the upper left abdomen. The patient had abdominal distension, but there was no vomiting or severe abdominal pain. The gallbladder had polyps on the ultrasound transabdominal, but there were no stones or pancreatitis. Magnetic resonance imaging (MRI) 2 months ago showed multiple stones in the gallbladder and normal pancreas. The laboratory results were leukocytes: 15x103/L, SGPT: 262, SGOT: 249, GGT: 1369 U/L, total bilirubin: 2.44, direct bilirubin: 2.08, lipase: 14,690 U/L, amylase: 3,693 U/L, and triglyceride: 56 mg/dl. The patient refused a CT scan or a repeated MRI. The levels of SGPT, SGOT, GGT, lipase, and amylase showed an improvement after 3 days. Based on the Atlanta classification, AP is diagnosed if at least 2 of the following 3 criteria are fulfilled: abdominal pain, serum lipase, or amylase at least 3 times the upper limit of normal (ULN) or characteristic findings on imaging. Gallstones are the most common cause of AP. Acute pancreatitis was suspected due to abdominal bloating that couldn't be explained by common causes, such as ascites, bowel edema, hematoma, and ileus. CT-scan contrast or MRI was necessary for patients who had normal USG and no classic sign of abdominal pain.

Keywords: *acute pancreatitis, abdominal bloating, normal transabdominal ultrasound*

ABSTRAK

Pankreatitis akut (PA) adalah gangguan inflamasi pankreas, dan diagnosis yang benar merupakan bidang yang menarik bagi dokter. Laki-laki 44 tahun, datang dengan keluhan kembung selama 1 bulan dan memburuk 1 hari sebelum masuk ke IGD, disertai rasa tidak nyaman di perut kiri atas. Pasien mengalami distensi perut, tidak ada muntah atau sakit perut yang parah. Ultrasonography (USG) transabdominal ditemukan polip kandung empedu, tidak ada batu atau pankreatitis. Magnetic resonance imaging (MRI) 2 bulan sebelumnya, menunjukkan banyak batu di kandung empedu dan pankreas normal. Hasil lab pasien menunjukkan leukosit 15x10^3/L, SGPT 262, SGOT 249, GGT 1369 U/L, total bilirubin 2,44, direk bilirubin 2,08, lipase 14.690 U/L, amilase 3.693 U/L dan trigliseride 56 mg/dl. Pasien menolak computerized tomography scan (CT scan) atau MRI berulang, berdasarkan kriteria Atlanta, ketidaknyamanan perut dengan amilase/lipase tinggi dan riwayat batu empedu, diagnosis ditegakkan. Setelah 3 hari, SGPT, SGOT, GGT, lipase, amilase mengalami perbaikan. Berdasarkan klasifikasi Atlanta, AP didiagnosis jika setidaknya 2 dari 3 kriteria berikut terpenuhi: sakit perut, lipase serum, atau amilase setidaknya 3x upper limit of normal (ULN) atau temuan karakteristik pada pencitraan. Batu empedu adalah penyebab pancreatitis terbanyak. Perut kembung pada pasien ini tidak dapat dijelaskan oleh penyebab umum, seperti asites, edema usus, hematoma, ileus yang membuat kami mencurigai adanya pankreatitis. Kontras CT-scan atau MRI sangat penting pada pasien dengan USG normal dan tidak ada tanda klasik nyeri perut.

Kata kunci: *pankreatitis akut, perut kembung, USG transabdominal normal*

INTRODUCTION

Acute pancreatitis is an inflammatory condition of the pancreas characterized by abdominal pain and elevated levels of pancreatic enzymes in the blood. Acute pancreatitis is a leading gastrointestinal cause of hospitalization in the United States. Several conditions are associated with acute pancreatitis. Gallstones and chronic alcohol use disorder are responsible for approximately two-thirds of these cases.[1] The reported annual incidence of acute pancreatitis in the United States ranges from 4.9 to 35 per 100,000 population.[2] The incidence of acute pancreatitis is increasing worldwide due to increased rates of obesity and gallstones.[3]

According to the 2012 revised Atlanta classification, the diagnosis of acute pancreatitis requires at least 2 of the 3 following criteria: abdominal pain consistent with pancreatitis, serum amylase and/or lipase of at least 3 times the upper limit of the normal value or findings consistent with acute pancreatitis on imaging contrast-enhanced CT (CECT), Magnetic resonance imaging (MRI) or ultrasound.[4]

We present a unique case of acute pancreatitis with abdominal distension, high lipase, and amylase, and no imaging ultrasound findings that suggest acute pancreatitis. Early diagnosis and supportive treatment led to the patient's recovery

CASE ILLUSTRATION

A 44-year-old man presented with bloating for one month and it worsened one day before admission to the ER, accompanied by discomfort in the upper left abdomen. The patient had abdominal distension, but there was no vomiting or severe abdominal pain. He denied any history of diabetes or prediabetes, obesity, abdominal trauma, and any surgical history, but he said he had stones in his gallbladder since 2 months ago from an MRI examination. Ultrasonography (USG) transabdominal was done in the ER and revealed a polyp gallbladder, with no stones or pancreatitis. But an MRI scan 2 months ago, showed multiple stones in the gallbladder and normal pancreas. The laboratory results were leukocytes: 15x10^3/L, SGPT: 262, SGOT: 249, GGT: 1369 U/L, total bilirubin: 2.44, direct bilirubin: 2.08, lipase: 14,690 U/L, amylase: 3,693 U/L, and triglyceride: 56 mg/dl. The patient refused a CT scan or a repeated MRI. Based on the Atlanta classification, AP is diagnosed if at least 2 of the following 3 criteria are fulfilled: abdominal pain, serum lipase, or amylase at least 3 times the upper limit of normal (ULN) or characteristic findings on imaging. During the first three days of treatment, the patient must be fasting and receive treatment such as parenteral nutrition smolfkabiven 1448ml/day, infusion of levofloxacin 1x500mg, infusion of metronidazole 3x500mg, esomeprazole injection 1x40mg, primperan injection 2x10mg and infusion of the hepatoprotection glycyrrhizin 2x1 ampoule. Abdominal distension was significantly alleviated after 3 days of treatment. The laboratory evaluation revealed an improvement in leukocytes, neutrophils, direct bilirubin, GGT, SGPT, amylase, and lipase. After improving, the patient was able to start drinking milk and eating porridge on the third day of treatment. We continued to administer the patient an infusion of levofloxacin 1x500mg, an infusion of metronidazole 3x500mg, an esomeprazole injection 1x40mg, and an infusion of the hepatoprotector glycyrrhizin 1x1 ampoule. On the sixth day of treatment, the patient fully recovered and the laboratory evaluation showed that leukocytes, neutrophils, SGPT, and direct bilirubin were normal, so the patient was discharged on the sixth day of admission.

Table 1.Hematological and biochemical investigations

Parameter	First day	Third day	Sixth day
Hemoglobin (g/dL)	13.6	13.1	12.4
Hematocrit (%)	39.5	38.1	36.7
Leukocyte (10^3/L)	15.2	11.4	8.5
Neutrophil (%)	81	76	65
Platelets (10^3/L	374	358	388
Creatinin (mg/dL)	0.8		
Amylase (U/L)	3963	38	
Lipase (U/ml)	14690	46	
Blood glucose	120	112	110
SGOT (U/L)	249	19	20
SGPT (U/L)	262	88	44
Gamma GT (U/L)	1369	1052	732
Albumin (g/dL)	3.7		
Total bilirubin (mg/dL)	2.44	1.36	0.90
Direct bilirubin (mg/dL)	2.08	1.12	0.50
Indirect bilirubin (mg/dL)	0.36	0.24	0.30
Total Cholesterol(mg/dl)	203		
Triglyceride (mg/dl)	56		
Cholesterol LDL (mg/dl)	151		
Cholesterol HDL (mg/dl)	56		
Ca 19-9 (U/ml)	9 (normal <37)		

SGOT: serum glutamic oxaloacetic transaminase, SPGT: serum glutamic pyruvic transaminase

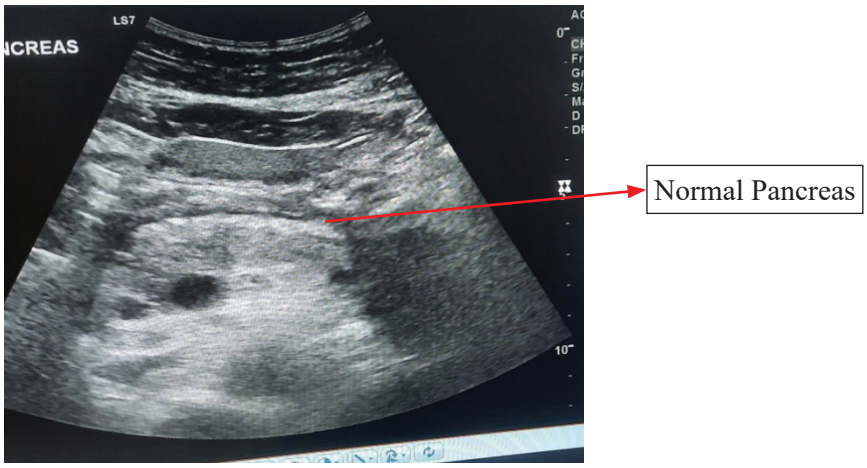

Figure 1. Ultrasonography abdominal (at ER) showed normal pancreas

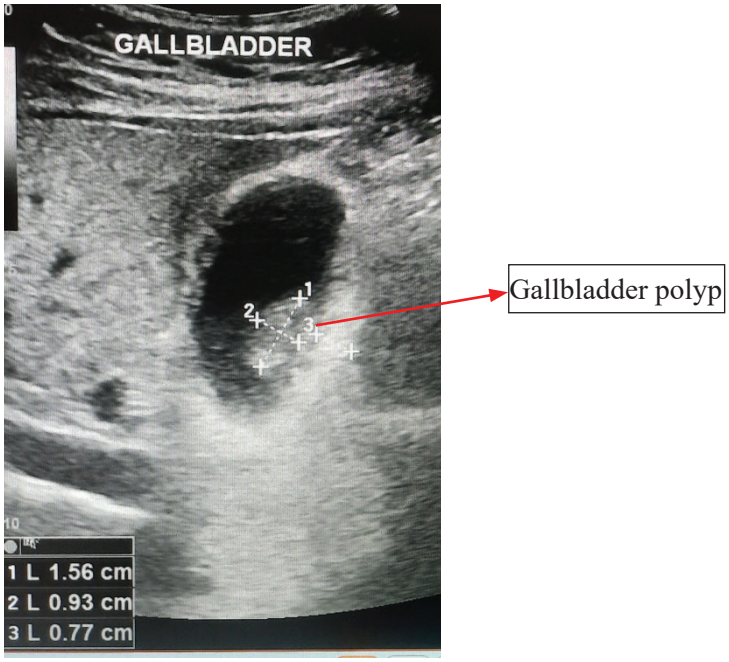

Figure 2. Ultrasonography abdominal (at ER) showed multiple polyps in the gallbladder and no stone

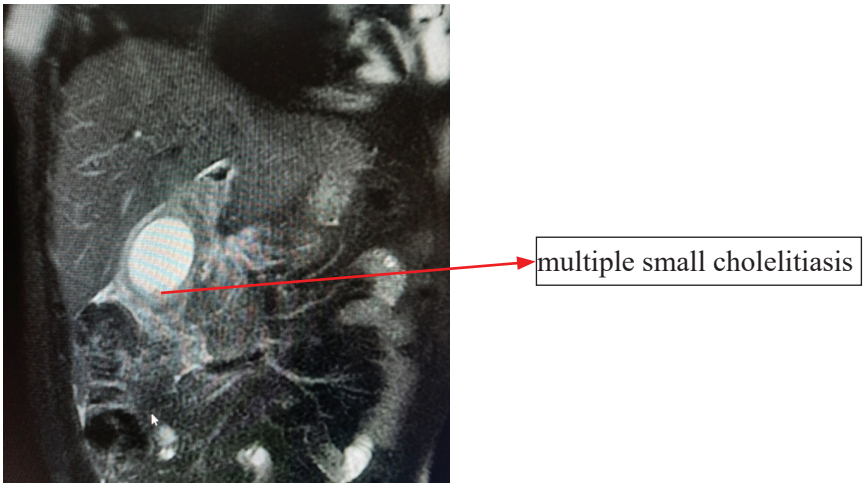

Figure 3. Abdominal MRI (2 months ago), showed multiple stones in gallbladder and normal pancreas

DISCUSSION

The annual incidence of acute pancreatitis ranges from 4.9 to 35 per 100,000 population, and it is rising globally.[2,5] Acute pancreatitis is among the most common gastrointestinal conditions requiring acute hospitalization.[9] Gallstones (including microlithiasis) are the most common cause of acute pancreatitis accounting for 40 to 70 percent of cases. However, only 3 to 7 percent of patients with gallstones develop pancreatitis while less frequent causes of acute pancreatitis include medication, endoscopic retrograde cholangiopancreatography, hypercalcemia, hypertriglyceridemia, surgery, and trauma.[8] The mechanism by which the passage of gallstones induces pancreatitis is unknown. Two factors have been suggested as the possible initiating event in gallstone pancreatitis: reflux of bile into the pancreatic duct due to transient obstruction of the ampulla during passage of gallstones; or obstruction at the ampulla secondary to stones or edema resulting from the passage of a stone.[11]

Based on the revised Atlanta classification, acute pancreatitis (AP) is diagnosed if at least 2 of the following 3 criteria are fulfilled: acute onset upper abdominal pain radiating to the back, serum lipase or amylase at least 3 times the (upper limit of normal) ULN or characteristic findings on imaging.[1,2]

Bloating and abdominal distension, are both common functional-type complaints encountered daily by gastroenterologists and healthcare providers.[4] Abdominal bloating is a subjective sensation that is commonly associated with objective abdominal distension. Bloating and distension can be functional or may be the manifestations of organic disorders which should be diagnosed and treated separately.[14] We should gather detailed dietary history, physical examination by assessing bowel movement frequency and stool consistency, and special imaging techniques to measure abdominal shape during episodes of distension. Causes of abdominal distension are related to an increase in intra-abdominal volume: ascites, bowel edema, hematoma, bowel distension, or ileus.[4] Several underlying mechanisms have been proposed and may coexist in an individual patient with abdominal bloating. First, intraluminal content includes gas, air, water, and fecal material. Air and gas may become abundant within the lumen through aerophagia and potentially from overproduction of gas by the colonic or small intestine. Second, bacteria or alteration in colonic microbacteria may lead to increased production of colonic gas by fermentation or decreased gas consumption, leading to increased colonic gas content and bloating. Collins et al found that interruption of the host–microbiota equilibrium affects the intestinal immune system and leads to inflammation. This, in turn, leads to gut sensory and motor dysfunction which may contribute to bloating.[14] In acute pancreatitis, a person may develop some swelling too besides bloating in the upper abdomen. This is because of compression of the duodenum and gastric distension so intestinal contents have stopped moving, causing the intestines to swell.[15]

In this case, there was no nausea, vomiting, or diarrhea, defecation and vent were normal and from USG abdominal we didn't find any fluid in the abdominal cavity, or swelling in the pancreas. The abdominal distension of this patient could not be explained by common causes: ascites, bowel edema, hematoma, bowel distension, or ileus, leading us to suspect pancreatitis.

Ultrasound has little value in the diagnosis of pancreatitis or its complications, but USG can give us the early identification of gallstones and biliary dilatation caused by stones. CT scan-contrast is the most clinically useful investigation, a CT scan can give information about peripancreatic inflammation, focal fluid collections, or necrosis pancreas. MRI may help in the differentiation of fluid collections from partly liquefied necrotic tissue.[13] Therefore, CT-scan contrast or MRI was essential in this patient with normal USG transabdominal and no classic sign of acute pancreatitis

Acute pancreatitis causes a local and systemic inflammatory response syndrome. Although the majority of patients have a mild disease course, around 20% will develop moderate or severe pancreatitis, with necrosis of the (peri) pancreatic tissue and/or (multiple-) organ failure. Supportive care with the use of intravenous fluid hydration is a mainstay of treatment for acute pancreatitis in the first 12–24 hours. Early fluid resuscitation is required to correct intravascular depletion to reduce morbidity and mortality associated with acute pancreatitis.[12] This patient was discharged on the sixth day of admission.

REFERENCES

1. Peery AF, Crockett SD, Murphy CC, Lund JL, Dellon ES, Williams JL, et al. Burden and cost of gastrointestinal, liver, and pancreatic diseases in the United States: update 2018. Gastroenterology 2019;156:254-72.
2. Vege SS, Yadav D, Chari ST. Pancreatitis. GI Epidemiology. Talley NJ, Locke GR, Saito YA (ed): Blackwell Publishing, Malden, MA. 2007; DOI:10.1002/9780470692189.

3. Toouli J, Brooke-Smith M, Bassi C, Carr-Locke D, Telford J, Freeny P, et al. Guidelines for the management of acute pancreatitis. J Gastroenterol Hepatol 2002;17 Suppl:S15-39.
4. Malagelada JR, Accarino A, Azpiroz F. Bloating and abdominal distension: old misconceptions and current knowledge. Am J Gastroenterol 2017;112:1221–31.
5. Roberts SE, Morrison-Rees S, John A, Williams JG, Brown TH, Samuel DG. The incidence and etiology of acute pancreatitis across Europe. Pancreatology 2017;17:155–65.
6. Yadav D, Lowenfels AB. Trends in the epidemiology of the first attack of acute pancreatitis: a systematic review. Pancreas 2006;33:323–30.
7. Nesvaderani M, Eslick GD, Vagg D, Faraj S, Cox MR. Epidemiology, etiology, and outcomes of acute pancreatitis: a retrospective cohort study. Int J Surg 2015;23(Pt A):68–74.
8. Forsmark CE, Baillie J; AGA Institute Clinical Practice and Economics Committee; AGA Institute Governing Board. AGA Institute technical review on acute pancreatitis. Gastroenterology 2007;132:2022-44.
9. Riela A, Zinsmeister AR, Melton LJ, DiMagno EP. Etiology, incidence, and survival of acute pancreatitis in Olmsted County, Minnesota USA. Gastroenterology 1991;100:A296.
10. Moreau JA, Zinsmeister AR, Melton LJ 3rd, DiMagno EP. Gallstone pancreatitis and the effect of cholecystectomy: a population-based cohort study. Mayo Clin Proc 1988;63:466-73.
11. Lerch MM, Saluja AK, Rünzi M, Dawra R, Saluja M, Steer ML. Pancreatic duct obstruction triggers acute necrotizing pancreatitis in the opossum. Gastroenterology 1993;104:853-61.
12. Tenner S, Baillie J, DeWitt J, Vege SS; American College of Gastroenterology. American College of Gastroenterology guideline: management of acute pancreatitis. Am J Gastroenterol 2013;108:1400–15.
13. Brizi MG, Perillo F, Cannone F, Tuzza L, Manfredi R. The role of imaging in acute pancreatitis. Radiol Med 2021;126:1017–29.
14. Mari A, Backer FA, Mahamid M, Amara H, Carter D, Boltin D. Bloating and abdominal distension: clinical approach and management. Adv Ther 2019;36:1075–84.
15. Murray WJ. Massive gastric distention in acute pancreatitis--a report of two cases. Postgrad Med J 1984;60:631-3.