Pancreatic pseudocyst infection due to multidrug resistant acinetobacter baumannii
Pancreatic pseudo cyst infection due to mdr-a. Baumannii

La infección pseudocista pacreática causada por el resistente a las drogas múltiples acinetobacter baumannii.[1]

Cruz N. Rodríguez,[1] Alfonso J. Rodríguez-Morales,[2@] Ada García,[1] Bileida Pastran,[1] Ivette Jiménez,[1] Pilar Meijomil.[1]

[1]Lab. Microbiología, Hospital General del Oeste, Caracas, Venezuela; [2]Instituto Experimental José Witremundo Torrealba, NURR-ULA, Trujillo; [@]Email: ajrodriguezm_md@hotmail.com

Abstract
Background: Pancreatic infections due to Acinetobacter baumannii, a wider drug resistant gram negative bacillus, are unusual. Its management is complicated and should include surgical and antimicrobial treatments. Methods: A case report and review of the pertinent English-language literature are presented. Results: We report a case of a patient with a previous acute pancreatitis that developed an infected pancreatic pseudocyst requiring surgical and antimicrobial treatment. Conclusions: This etiology is unique in the literature, and given the emergence of multidrug resistance pattern, worldwide, initial empiric therapy with drugs such as quinolones and carbapenems is recommended, jointly with proper surgical management.
Key Words: Acinetobacter baumanii, pancreatic pseudocyst, infection.

Resumen
Introducción: Las infecciones del páncreas producidas por Acinetobacter baumannii, un bacilo gram-negativo ampliamente resistente, son infrecuentes. Su manejo es complicado y debe incluir tratamientos quirúrgicos y antimicrobianos. Métodos: En el presente artículo reportamos un caso y su revisión de la literatura. Resultado: Se reporta el caso de un paciente previamente con cuadro de pancreatitis aguda que desarrollo pseudoquiste pancreático infectado requiriendo tratamiento quirúrgico y antimicrobiano. Conclusiones: La etiología aquí reportada es infrecuente de la literatura, y dada la emergencia de patrones de multiresistencia mundial, el manejo empirico inicial con drogas como las quinolonas y carbapenems es recomendado, en conjunto con el manejo quirúrgico apropiado.
Palabras Clave: Acinetobacter baumanii, pseudoquiste pancreático, infección.

Introduction

[1] Recibido: Marzo - 2005 Aprobado: Junio - 2006

Pancreatic pseudocysts (PPC) are common in acute and chronic pancreatitis and have numerous potential complications including infection, hemorrhage into the pseudocyst or intestinal tract, free intraperitoneal rupture, and fistulae [1,2].

Infectious complications frequently arise, and especially infection of pancreatic necrosis is an important risk factor for mortality. Several strategies have been devised to reduce this risk, and the use of prophylactic therapy, e.g. selective digestive decontamination, can be considered in patients with documented necrosis of the pancreas.

Pancreatic abscesses and infected pseudocysts arise later in the course of disease, and should be considered as separate entities, due to differences in therapy and outcome of these patients. When infection occurs, source control using either surgical or percutaneous drainage techniques, is essential to avoid systemic severe complications [3]. Etiological agents involved in these infectious complications of pancreatic diseases are: Escherichia coli (44.4%), Enterococcus spp. (28.4%), Staphylococcus aureus (14.8%), Klebsiella spp. (16.0%), Pseudomonas spp. (4.9%), Proteus spp. (3.7%), among others [4,5].

In The literature is not reported either in case or in case reports, Acinetobacter baumannii as etiological agent of infected PPC, for this reason we report a case of patient with a previous acute pancreatitis that developed an infected PPC requiring surgical and antimicrobial treatment.

Case report

The patient, a 34-year-old man with a past history of acute pancreatitis in the three previous months worsening in the three previous days before hospitalization, when presented intense right hypochondrial pain and nausea. No other any notable medical history was found. He was no taking any medications and denied drug or alcohol use.

Physical examination revealed a temperature of 37.8ºC, pulse of 92 beats/min, blood pressure of 120/80 mm Hg, and respirations of 24 breaths/min. Cardiopulmonary exam was within normal limits. The abdomen was diffusely tender but otherwise soft and non-distended, with audible bowel sounds. Rectal exam revealed guaiac-negative stool. Peripheral pulses were palpable bilaterally.

Initial laboratory studies revealed a white blood cell count of 14,500/mm^3 (normal: 5,000-10,000), with a left shift of 81.2% neutrophils (55-70). The hematocrit was 41.1% (42-52), the platelet count was 487 × 10^3/mm^3 (150-350), prothrombin time was 9.5 sec (8-12), activated partial thromboplastin time was 33.6 sec (24.0-36.0), fibrinogen concentration was 210 mg/dL (200-400), and lactate concentration was 710 U/L (130-500). The serum total bilirubin concentration was 0.83 mg/dL (0.2-1.0). Urea nitrogen concentration was 10.5 mg/dL (10-520) and a creatinine concentration of 0.8 mg/dL (0.5-1.3). Urinalysis was normal. An arterial blood gas was also normal. Chest and supine abdominal X-rays were also normal. Abdominal CT showed a collection with a fluid component that have apposition to the gastric wall, this fluid-containing cystic mass suggested a pancreatic pseudocyst (class D, Balthazar classification) [6]. Abdominal ultrasound showed also the pancreatic pseudocyst under the lower wall of the stomach (Fig. 1), additionally gallbladder lithiasis was also diagnosed.

Three set of blood and stool cultures were obtained. Empiric antibiotic therapy was initiated with ciprofloxacin. The patient was transferred to the surgical ward and prepared for an endoscopic retrograde cholangiopancreatography (ERCP) that reveals no apparent pancreatic alterations. The patient underwent pancreaticocystogastrostomy, cholecystectomy, hepaticojejunostomy-en-Y, gastroenterostomy and enteroenterostomy. During surgical procedure, PPC liquid samples were taken for cultures. Blood and stools cultures were negative, but in PPC liquid a gram-negative bacilli grew, being identified by biochemical and automated methods as Acinetobacter baumannii. In vitro antimicrobial susceptibility of this isolate was assessed by an agar disk diffusion method using Mueller-Hinton agar as recommended by the National Committee for Clinical Laboratory Standards (NCCLS) (now Clinical and Laboratory Standards Institute, CSLI) [7].

Isolate was tested against 24 drugs, including: ampicillin/sulbactam, piperacillin, piperacillin/tazobactam, ceftazidime, cefoperazone, amikacin, gentamicin, tobramicin, ciprofloxacin, cefepime, aztreonam, meropenem and imipenem, among others. This isolated strain of A. baumannii showed resistance to all tested drugs except for imipenem. A course of 2-weeks therapy with imipenem (0.5 g each 6h) was given. Postoperative recovery was unremarkable. After 3 weeks, he was tolerating a normal diet and was discharged. There were no further complications.

Discussion

In the clinical setting of acute pancreatitis, postacute pseudocyst and pancreatic abscess are late consequences of the disease [1,4,8,9]. Both features are characterized by an inflammatory wall, which delineates the process from the surrounding tissue and is located within or around the pancreas. Pancreatic pseudocysts may have a connection with the pancreatic duct system, which has a major impact on treatment. Postacute pseudocyst and pancreatic abscess are late consequences of acute, mostly necrotizing pancreatitis. It has been convincingly established that postacute pseudocyst is a late complication of necrotizing pancreatitis with sterile necrosis. Pancreatic abscess appears as a collection of pus with little or no necrosis, and cultures reveal bacteria or fungi. It should not be confused with infected necrosis, as both features differ in clinical expression and associated mortality [8].

The therapeutic role of antibiotics in acute pancreatitis is controversial. Three early controlled trials using ampicillin in patients with mild pancreatitis showed no effect on the course of the illness [10-14]. Several studies have shown that ampicillin is a poor antibiotic choice for acute pancreatitis. First, ampicillin does not cover many of the common organisms infecting the pancreas in acute pancreatitis, particularly some strains of Escherichia coli, Klebsiella, and Enterobacter [15]. Second, ampicillin does not penetrate the pancreas. Some studies have evaluated the concentrations of several antibiotics in the blood and pancreatic tissue [16]. Ciprofloxacin, ofloxacin, and imipenem were found to have high pancreatic tissue levels and high bactericidal activity [17], against organisms known to cause infected necrosis and sepsis in patients with acute pancreatitis. In this setting, administration of these antibiotics to patients with severe acute (necrotising) pancreatitis and pancreatic pseudocysts reduces the risk of serious complications [18]. Due to the emergence of multidrug resistant strains of A. baumannii as well of other enterobacteria and gram negative bacilli

[19-21], especially in the surgical setting [20,21], is important to consider the use of wide spectrum antimicrobial drugs in the initial empiric therapy of suspected infected pancreatic pseudocysts as well the importance of microbiological cultures and drug susceptibility tests.

Figure 1. Abdominal ultrasound of patient at income, showing the pancreatic pseudocyst behind the lower wall (pared) of the stomach.

Figure5

Referentes

1. Grace P, Williamson R. Modern management of pancreatic pseudocysts. Br J Surg 80:573–581, 1993.

2. Heider R, Behrns Ke. Pancreatic pseudocysts complicated by splenic parenchymal involvement: results of operative and percutaneous management. Pancreas 23:20-25, 2001.

3. De Waele J, Vogelaers D, Decruyenaere J, De Vos M, Colardyn F. Infectious complications of acute pancreatitis. Acta Clin Belg 59:90-96, 2004.

4. Beger Hg, Rau B, Mayer J, Pralle U. Natural course of acute pancreatitis. World J Surg 21:130-135, 1997.

5. Rau B, Uhl W, Buchler Mw, Beger Hg. Surgical treatment of infected necrosis. World J Surg 21:155-161, 1997.

6. Balthazar Ej, Robinson Dl, Megibow Aj, Ranson JH. Acute pancreatitis: value of CT in establishing prognosis. Radiology 174:331-6, 1990.

7. National Committee for Clinical Laboratory Standards. Performance Standards for Antimicrobial Susceptibility Testing. Information Supplement. NCCLS, Villanova, 2003.

8. Bittner R, Block S, Buchler M, Beger Hg. Pancreatic abscess and infected pancreatic necrosis. Different local septic complications in acute pancreatitis. Dig Dis Sci 32:1082-7, 1987.

9. Bradley EL 3rd. A clinically based classification system for acute pancreatitis. Summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 through 13, 1992. Arch Surg 128:586-590, 1993.

10. Tenner S, Banks Pa. Acute pancreatitis: nonsurgical management. World J Surg 21:143-148, 1997.

11. Craig Rm, Dordal E, Myles L. The use of ampicillin in acute pancreatitis. Ann Intern Med 83:831-832, 1975.

12. Cameron Jl, Howes R, Zuidema Gd. Antibiotic therapy in acute pancreatitis. Surg Clin North Am 55:1319-1324, 1975.

13. howes r, zuidema gd, cameron jl. Evaluation of prophylactic antibiotics in acute pancreatitis. J Surg Res 18:197-200, 1975.

14. Finch Wt, Sawyers Jl, Schenker S. A prospective study to determine the efficacy of antibiotics in acute pancreatitis. Ann Surg 183:667-671, 1976.

15. Rodriguez Aj, Rodriguez Cn, Garcia A, Duque C, Molina N, Barbella R, Lakatos M, Meijomil P. Antibiotic susceptibility of Enterobacteriaceae species isolated in Venezuela over ten years. J Chemother 13:450-452, 2001.

16. Buchler M, Malfertheiner P, Friess H, Isenmann R, Vanek E, Grimm H, Schlegel P, Friess T, Beger Hg. Human pancreatic tissue concentration of bactericidal antibiotics. Gastroenterology 103:1902-1908, 1992.

17. Rodriguez Cn, Rodriguez-Morales Aj, Garcia A, Pastran B, Meijomil P, Barbella Ra, Blanco Jj, Vargas Ja, Gutierrez G. Quinolones antimicrobial resistance in certain Enterobacteriaceae: A ten-year evaluation study in a general hospital of Venezuela. Int J Antimicrob Agents 25:546-50, 2005.

18. Bosscha K, Vos A, Visser Mr, Berger P, Van Dullemen H, Ploeg Rj, Gooszen Hg. Reduced risk of complications associated with severe acute (necrotizing) pancreatitis by administration of antibiotics; results from a literature review. Ned Tijdschr Geneeskd 145:1982-1985, 2001.

19. Rodriguez Cn, Rodriguez-Morales Aj, Garcia A, Pastran B, Meijomil P. Comparative study of antimicrobial drug resistance of Acinetobacter baumannii inside and outside the ICU at West General Hospital, Caracas, Venezuela. Acta Cient Venez 51:165-166, 2000.

20. Maragakis Ll, Cosgrove Se, Song X, Kim D, Rosenbaum P, Ciesla N, Srinivasan A, Ross T, Carroll K, Perl Tm. An outbreak of multidrug-resistant Acinetobacter baumannii associated with pulsatile lavage wound treatment. JAMA 292:3006-3011, 2004.

21. El Shafie Ss, Alishaq M, Leni Garcia M. Investigation of an outbreak of multidrug-resistant Acinetobacter baumannii in trauma intensive care unit. J Hosp Infect 56:101-105, 2004.