CASE REPORT | PANCREAS



Necrotizing Pancreatitis After Cardiac Arrest With Cardiopulmonary Resuscitation

Christine Donat, MD¹, Michel Farah, MD², and Kathryn Jobbins, DO, MS¹

¹Department of Medicine, University of Massachusetts Chan Medical School-Baystate, Springfield, MA ²Department of Cardiology, University of Massachusetts Chan Medical School-Baystate, Springfield, MA

ABSTRACT

Acute pancreatitis has been reported as a complication of cardiac arrest and cardiopulmonary resuscitation. However, necrotizing pancreatitis as a subsequent complication has not. Because pancreatic necrosis develops 7–10 days after the initial episode of pancreatitis, it may be difficult to identify and, therefore, diagnose. This case details the course of a patient who developed infected necrotizing pancreatitis after receiving cardiopulmonary resuscitation after cardiac arrest.

KEYWORDS: acute necrotizing pancreatitis; cardiopulmonary resuscitation; ischemia; wounds and injuries

INTRODUCTION

Necrotizing pancreatitis is deadly, with approximately 15% mortality overall and 27% mortality in those requiring surgical intervention for suspected infected necrosis.¹

In post-cardiac resuscitation patients, this diagnosis can be missed given many remain intubated and limited in ability to communicate their symptoms.

Thus far, few cases of necrotizing pancreatitis after cardiopulmonary resuscitation (CPR) have been reported. In one case, direct compression over the xiphoid process was noted by the code leader and the resulting pancreatitis was believed to be traumatic.² In another case, automatic chest compressions using a LUCAS device resulted in traumatic pancreatic fracture and necrotizing pancreatitis.³ Another reported case cites the development of pancreatitis after prolonged CPR, believed to be due to ischemia in the setting of cardiac arrest.⁴

In our case, the patient presented without signs of pancreatitis before her cardiac arrest. She was later diagnosed by computed tomography (CT), which confirmed necrotizing pancreatitis, subsequently found to be infected. The pancreatitis was unfortunately not recognized before the patient's clinical decompensation secondary to infected necrosis.

CASE REPORT

Our patient is a 51-year-old woman admitted after an out-of-hospital hypoxic cardiac arrest secondary to asthma exacerbation. She had unknown downtime and received 5 rounds of CPR with pulseless electrical activity rhythm. Return of spontaneous circulation was achieved with epinephrine, and the patient was intubated and transferred to the intensive care unit where she remained transiently febrile and was suspected to have aspiration pneumonia based on CT findings. Her CT on admission also showed a contracted gallbladder without inflammation and no intra or extrahepatic biliary dilation. She was treated with 5 days of ampicillin/ sulbactam without improvement and transitioned to piperacillin/tazobactam and vancomycin, later transitioning to vancomycin with cefepime because of hypernatremia. On day 9, she was able to be extubated. Secondary to persistent fever, antibiotic coverage was rebroadened to piperacillin/tazobactam from cefepime.

ACG Case Rep J 2023;10:e01223. doi:10.14309/crj.000000000001223. Published online: December 20, 2023 Correspondence: Christine Donat, MD (christine.donat@baystatehealth.org).



Figure 1. Severe acute pancreatitis with small acute peripancreatic fluid collection.

Our patient was now able to describe right upper quadrant (RUQ) tenderness, and a RUQ ultrasound was performed, without abnormal findings. Over the next 36 hours (hospital day 11), the patient became hypotensive and a CT was performed, which showed necrotizing pancreatitis without other significant findings, including an unremarkable gallbladder and no biliary ductal dilation (Figure 1). On chart review, initial laboratory tests drawn in the emergency department showed a lipase of 199 (upper limit of normal 60 IU/L). She had no history of alcohol use, hyperlipidemia, or hypercalcemia and had not received any drugs at home or during her hospital stay known to cause pancreatitis.⁵ Total IgG and subclasses were also normal, except for IgG1 at 835 (upper limit of normal 810 mg/dL). Aspartate transaminase/alanine transaminase were initially elevated in the setting of cardiac arrest, peaking at 7,380/4,292 on hospital day 2, slowly trending down to normal by day 10, and remaining normal thereafter despite RUQ pain. Bilirubin remained normal throughout.



Figure 2. Necrotizing pancreatitis with worsening acute and chronic collections nearly encasing the entire pancreas. Large subcapsular fluid collection overlying the left hepatic lobe and small subcapsular fluid collection overlying the posterior right hepatic lobe.

Despite a normal biliary system on imaging throughout her hospital stay, repeat biliary ultrasound on hospital day 13 showed dilation of the common bile duct (CBD) to 1.3 cm. Given concern for choledocholithiasis, the surgery team took the patient for cholecystectomy within 24 hours. There was no dilation of the biliary tree on intraoperative cholangiogram and no stone identified. There was also no evidence of intrinsic or extrinsic compression of the CBD. Given its transience, the most likely etiology of the CBD dilation seems to be opioid medication because the patient had been receiving frequent morphine doses for several days.

Choledocholithiasis was unlikely believed to be the etiology of her pancreatitis, given the pancreatitis far preceded the CBD dilation (13 days), with a normal liver panel and normal prior imaging. Furthermore, given the lipase was elevated immediately after her arrest, this would rather suggest pancreatic trauma or ischemic injury.

The patient's hospital course was further complicated by the development of pancreatic and hepatic abscesses (Figure 2) managed surgically, bacteremia, reintubation, and a second cardiac arrest with return of spontaneous circulation. After a prolonged hospital stay, she was able to be discharged to a rehab facility and then home where she continues to recover.

DISCUSSION

This case highlights the possibility of necrotizing pancreatitis as a complication of CPR, the etiology of which may be related to direct trauma, ischemia, or, most likely for this patient, a combination of these factors.

The incidences of both traumatic and ischemic pancreatitis after CPR are low, with only 3 case reports found on literature review for either etiology. For ischemic pancreatitis alone, the overall incidence is more common. In a retrospective analysis of acute pancreatitis in intensive care unit patients, 22% were identified as having ischemia as the etiology.⁶ In addition, a retrospective autopsy study in patients who died after elective resection and grafting of abdominal aortic aneurysm showed an 11% incidence of pancreatitis compared with a 3% pancreatitis rate in all autopsies.⁷

Given the rarity of pancreatitis after CPR, it is difficult to recognize. In several cases where acute pancreatitis was recognized after CPR, the diagnosis was discovered on imaging while 1 case was diagnosed from elevated lipase.

To better identify cases, recognition of risk factors of pancreatitis is beneficial. These include shock/ischemia, trauma, drug toxicity, alcohol use, gallstones, autoimmune disease, hyperlipidemia, and hypercalcemia. In this case, the trauma and ischemia associated with CPR were not initially considered, and therefore, higher suspicion for pancreatitis in these patients may be warranted. It is not currently the standard of care to check lipase after CPR. However, this test is easy to perform and if pancreatitis is addressed early, increased morbidity may be avoided. Measurement of lipase after CPR remains a possible direction for further investigation.

Another highlight of this case is the question of surgical vs endoscopic necrosectomy for the treatment of infected necrosis. In our patient, surgical necrosectomy was likely chosen because of the presence of multiple hepatic abscesses traversing where the root of the mesenteric vessels would lie deep in the posterior right lobe, difficult to access through endoscopic means. However, in other circumstances, endoscopic pancreatic necrosectomy may be superior when compared with surgical means because endoscopic necrosectomy had a lower rate of pancreatic fistulas and shorter length of hospital stay compared with surgery.⁸

DISCLOSURES

Author contributions: All authors made substantial contributions to the conception or design of the work; the acquisition, analysis, or interpretation of data; drafting or revising the manuscript critically for important intellectual content; final approval of the version to be published; and agree to be accountable for all aspects of the work. Christine Donat, MD, is the article guarantor.

Financial disclosure: None to report.

Informed consent was obtained for this case report.

Received June 6, 2023; Accepted November 6, 2023

REFERENCES

- Van Santvoort HC, Bakker OJ, Bollen TL, et al; Dutch Pancreatitis Study Group. A conservative and minimally invasive approach to necrotizing pancreatitis improves outcome. *Gastroenterology*. 2011;141(4):1254–63.
- Aziz M. Traumatic pancreatitis: A rare complication of cardiopulmonary resuscitation. *Cureus*. 2017;9(8):e1574.
- Deras P, Manzanera J, Millet I, Charbit J, Capdevila X. Fatal pancreatic injury due to trauma after successful cardiopulmonary resuscitation with automatic mechanical chest compression. *Anesthesiology*. 2014;120(4): 1038–41.
- Piton G, Barbot O, Manzon C, et al. Acute ischemic pancreatitis following cardiac arrest: A case report. JOP. 2010;11(5):456–9.
- Wolfe D, Kanji S, Yazdi F, et al. Drug induced pancreatitis: A systematic review of case reports to determine potential drug associations. *PLoS One*. 2020;15(4):e0231883.
- Baldursdottir MB, Andresson JA, Jonsdottir S, et al. Ischemic pancreatitis is an important cause of acute pancreatitis in the intensive care unit. J Clin Gastroenterol. 2023;57(1):97–102.
- Warshaw AL, O'Hara PJ. Susceptibility of the pancreas to ischemic injury in shock. Ann Surg. 1978;188(2):197–201.
- van Brunschot S, van Grinsven J, van Santvoort HC, et al; Dutch Pancreatitis Study Group. Endoscopic or surgical step-up approach for infected necrotising pancreatitis: A multicentre randomised trial. *Lancet.* 2018; 391(10115):51–8.

Copyright: © 2023 The Author(s). Published by Wolters Kluwer Health, Inc. on behalf of The American College of Gastroenterology. This is an open access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.