

Acute pancreatitis mimicking myocardial ischemia: A case report and a review of the literature

Antonio Villa¹, Francesca Campagna¹, Giulia Gallotta¹,
Riccardo Bennicelli², Paola Sartori³

Affiliations:

¹ M.D., Department of Emergency, ASST Monza, PO Desio, Monza, Italy.

² M.D., Department of Cardiology, ASST Monza, PO Desio, Monza, Italy.

³ M.D., Department of Surgery, ASST Monza, PO Desio, Monza, Italy.

Corresponding author:

Dr. Antonio Villa, ASST Monza, PO Desio, Via Fiuggi, 56 20159 Milan, Italy.

E-mail: antonio_villa@fastwebnet.it

Abstract

In literature, acute pancreatitis has been shown to be associated with a wide range of electrocardiographic changes. In the present report, we describe a case of a young woman who presented to our emergency room with chest pain and electrocardiographic alterations suggestive of myocardial ischemia; laboratory data showed elevated amylase and lipase levels. A computed tomography (CT) scan of the abdomen confirmed the diagnosis of acute pancreatitis. In the next day, she repeated the electrocardiogram, which showed a regression of the ST-segment depression. T-waves changes and ST-segment depression are common in acute pancreatitis and, therefore, emergency physicians should consider acute pancreatitis in the differential diagnosis of patients presenting with chest pain and electrocardiographic changes.

KEY WORDS: Acute pancreatitis; electrocardiographic changes; myocardial ischemia.

Riassunto

In letteratura, la pancreatite acuta è stata associata ad un'ampia gamma di alterazioni elettrocardiografiche. In questo lavoro presentiamo il caso di una giovane donna giunta alla nostra osservazione con dolore toracico ed alterazioni elettrocardiografiche suggestive di ischemia miocardica; i dati di laboratorio mostravano elevati livelli di amilasi e lipasi. Una TC dell'addome ha confermato la diagnosi di pancreatite acuta. Un ECG ripetuto il giorno successivo evidenziava una regressione del sottoslivellamento del tratto ST. Le modifiche dell'onda T ed il sottoslivellamento del segmento ST sono comuni nella pancreatite acuta e, pertanto, i medici che lavorano in emergenza dovrebbero considerare la pancreatite acuta nella diagnosi differenziale dei pazienti con dolore toracico ed alterazioni elettrocardiografiche.

TAKE-HOME MESSAGE

Acute pancreatitis has been shown to be associated with a wide range of electrocardiographic alterations and symptoms mimicking myocardial infarction. Emergency physicians should consider acute pancreatitis in the differential diagnosis of patients presenting with chest pain and electrocardiographic changes.

Competing interests - none declared.

Copyright © 2017 Antonio Villa et al. FS Publishers

This is an open access article distributed under the Creative Commons Attribution (CC BY 4.0) License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. See <http://www.creativecommons.org/licenses/by/4.0/>.

Cite this article as: Villa A, Campagna F, Gallotta G, Bennicelli R, Sartori P. Acute pancreatitis mimicking myocardial ischemia: A case report and a review of the literature. J Health Soc Sci. 2017;2(2):209-214

DOI 10.19204/2017/ctpn7

Received: 28/04/2017

Accepted: 18/06/2017

Published: 15/07/2017

INTRODUCTION

Acute pancreatitis (AP) is an acute inflammatory process of the pancreas that is associated with variable involvement of multiple organ system dysfunctions, in which cardiovascular manifestations are frequent [1]. A wide range of electrocardiographic (EKG) changes including arrhythmias, bradycardia, T-wave changes, intraventricular conduction disturbances, and ST-segment elevation or depression have also been described [2–11]. In the present report, we describe a case of a young woman who arrived at our department of emergency with chest pain and EKG alterations suggestive of myocardial ischemia.

CASE REPORT

A 21-year-old woman presented to our emergency room with lower mild sternal chest pain radiating posteriorly and associated with vomiting and diarrhea. She denied any type of alcohol or drug usage; moreover, she did not have any cardiovascular risk factors, except for estroprogestinic anti-contraceptive therapy. Physical examination showed a blood pressure of 110/65 mmHg and a heart rate of 101 beats per minute (BPM). Her chest was clear to auscultation and heart sounds were normal. Her abdomen was soft and nontender with adequate bowel sounds. There were no guarding, rebound tenderness, or organomegaly. A 12-lead EKG showed diffuse ST-segment depression with T negative (Figure 1). Laboratory data showed: Potassium 3,4 mMol/L, calcium 2,12 mMol/L, aspartate aminotransferase 44 U/L, amylase 1136 U/L, and lipase 1116 U/L; HS-troponin T was negative. Echocardiogram was normal. A computed tomography (CT) scan of the abdomen revealed a focal acute edematous pancreatic tail pancreatitis. She was treated with intravenous fluids and pain medication (nothing by mouth). In the next day, a repeat ECG showed regression of the ST-segment depression.

DISCUSSION

Drummond first reported EKG changes in AP in 1934 [1]. Tachycardia and fibrillation

were noted and suspected to be secondary to shock. Over the years, other authors also identified EKG abnormalities in patients with AP [4–7, 12–16]. Different types of tachyarrhythmias and bradyarrhythmias, such as atrial flutter and fibrillation, and supraventricular premature contractions, shortened PR interval, QRS prolongation, various bundle-branch blocks, non-specific changes in repolarization, decrease T-wave voltage, T-wave changes, and ST-segment abnormalities were often seen, in approximately 50% of patients affected by AP. Most frequently associated changes with AP are T-wave inversion and ST-segment depression [1]. New onset ST-elevation in leads V2 and V4 with diffusely inverted T waves mimicking acute myocardial infarction have been reported with mild AP [4, 17, 18]. Although very rare, AP may even be complicated by myocardial infarction secondary to hypovolemia, coagulation abnormalities, or coronary artery spasm [19].

Studies evaluating EKG changes in AP have yielded varying results. Buch et al. [3] reported that 57% of patients with AP had transient EKG abnormalities, which consisted mainly in non-specific T-wave changes and accelerated atrial or nodal rhythms.

Mautner et al. [20] also reported that significant EKG alterations during AP usually occur in patients with previous cardiac abnormalities.

Rubio-Tapia et al. [7] reported EKG abnormalities in 55% of patients affected by AP, consisting in non-specific changes of repolarization, sinus tachycardia, and left anterior hemiblock being the most frequent abnormalities.

Pezzilli et al. [2] also reported that 51.8% of the patients with AP had EKG abnormalities, and electrolyte alterations were present in about one-half of these.

Nadkarni et al. [21] found EKG abnormalities in more than 80% of patients with AP, and the 68% of patients with severe pancreatitis had prolonged QTc as compared to 35% of patients with mild pancreatitis. Furthermore, all the patients who succumbed had pro-

longed QTc. Similar results were reported by Ates et al. [6], who evaluated QTc dispersion in 32 patients with acute biliary pancreatitis. No cardiac pathology was demonstrated in patients who had EKG changes by echocardiography or coronary angiography [4, 22, 23]. The EKG changes return to their baseline once the patient recovers from AP [7, 11, 17, 18, 23–25].

The reason why EKG changes are seen with AP is still unclear. There are multiple hypotheses proposed, such as electrolyte abnormalities, coronary vasospasm, fat embolism, myonecrosis from release of pancreatic enzymes, and hypovolemia [14, 17, 22, 26, 27]; it has been also hypothesized that the EKG changes might be due to underlying ischemic heart disease unmasked by the stress of AP and/or imbalance of the autonomous nervous system [16, 20], endogenous insulin-induced hypoglycemia [28], hemodynamic instability, cardiobiliary reflex [16, 29], exacerbation of underlying cardiac disease, coagulopathy, coronary artery spasm, and drug use such as vasopressors [4, 5, 13, 14]. ST-segment elevation and increase in creatine phosphokinase MB fraction are thought to be secondary to a direct cardiotoxic effect of proteolytic pancreatic enzyme [5].

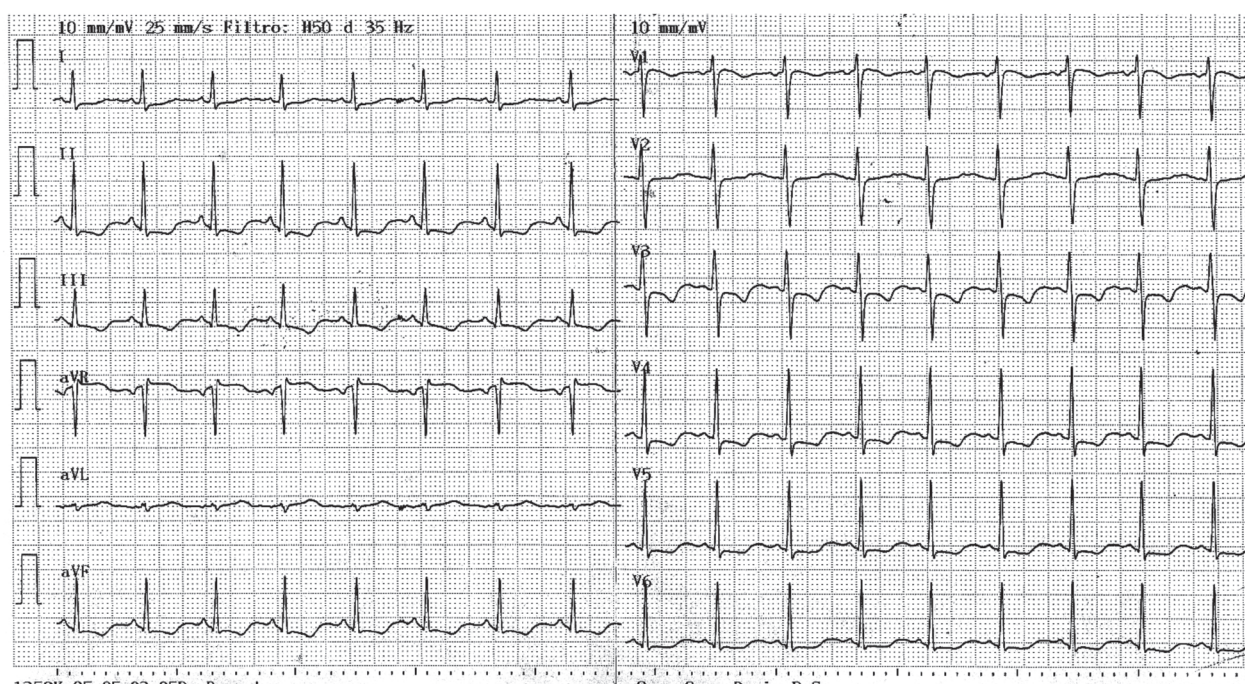
Electrolyte abnormalities have been implicated as a possible cause of EKG changes [2].

AP is associated with some metabolic abnormalities [1], such as hypocalcemia, hypophosphatemia, and hyperkalemia. Effects of low calcium on EKG consist of QT prolongation, ST-segment changes, and T-wave abnormalities [30]. It is known that variations on the level of ionized calcium have been directly correlated to clinically significant changes in myocardial contractility [31].

Hypomagnesemia associated with AP has been implicated to play a role causing coronary vasospasm resulting in stunning of the myocardium and EKG changes. The phenomenon is known as the ‘cardiobiliary reflex’ [2, 4, 5, 14, 27, 32].

Hypomagnesemia is associated with concomitant metabolic changes and EKG abnormalities [32]. Atrial fibrillation, atrial flutter, supraventricular tachycardia, *torsade de pointes*, prolonged PR interval, widened QRS complex, and prolonged QT interval are some of the changes [32]. Hypomagnesemia is also implicated in coronary artery spasm [32]. Other EKG abnormalities were found to be associated with lower levels of phosphorus and calcium [7].

Figure 1. EKG showing ST depression in leads II, III, aVF, and V3-V6.



CONCLUSIONS

T-waves changes and ST-segment depression are common in AP and, therefore, AP should be considered by emergency physicians in the

differential diagnosis of patients presenting with chest pain and EKG changes mimicking myocardial ischemia.

References

1. Yegneswaran B, Kostis JB, Pitchumoni CS. Cardiovascular manifestations of acute pancreatitis. *J Crit Care*. 2011;26(2):225e11–18.
2. Pezzilli R, Barakat B, Billi P, Bertaccini B. Electrocardiographic abnormalities in acute pancreatitis. *Eur J Emerg Med*. 1999;6(1):27–29.
3. Buch J, Buch A, Schmidt A. Transient ECG changes during acute attacks of pancreatitis. *Acta Cardiol*. 1980;35(5):381–390.
4. Khairy P, Marsolais P. Pancreatitis with electrocardiographic changes mimicking acute myocardial infarction. *Can J Gastroenterol*. 2001;15(8):522–526.
5. Albrecht CA, Laws FA. ST segment elevation pattern of acute myocardial infarction induced by acute pancreatitis. *Cardiol Rev*. 2003;11(3):147–151.
6. Ates F, Kosar F, Aksoy Y, Yildirim B, Sahin I, Hilmioglu F. QT interval analysis in patients with acute biliary pancreatitis. *Pancreas*. 2005;31(3):238–241.
7. Rubio-Tapia A, Garcia-Leiva J, Asensio-Lafuente E, Robles-Diaz G, Vargas-Vorackova F. Electrocardiographic abnormalities in patients with acute pancreatitis. *J Clin Gastroenterol*. 2005;39(9):815–818.
8. Makaryus AN, Adedeji O, Ali SK. Acute pancreatitis presenting as acute inferior wall ST-segment elevations on electrocardiography. *Am J Emerg Med*. 2008;26(6):734.e1–4.
9. Meuleman VG, Schinkel AF, Vos J. Electrocardiographic abnormalities caused by acute pancreatitis. *Neth Heart J*. 2011;19(3):137–139.
10. Sokmen M, Bugdaci MS, Oztekin E. Electrocardiographic changes and importance of repolarization changes in cases with acute pancreatitis. *Turk J Gastroenterol*. 2011;22(3):315–320.
11. Sethi P, Murtaza G, Sharma A, Paul T. ST segment elevation with normal coronaries. *Case Rep Med*. 2016. Vol 2016; Article ID 3132654, 4 pages. doi: 10.1155/2016/3132654.
12. Faintuch JJ, Abrahao MM, Giacaglia LR, Junqueira PC, Salgado LF. Electrocardiographic changes in pancreatitis. *Arq Bras Cardiol*. 1989;52(5):259–260.
13. Wang K, Asinger RW, Marriott HJ. ST-segment elevation in conditions other than acute myocardial infarction. *N Engl J Med*. 2003;349(22):2128–2135.
14. Yu AC, Riegert-Johnson DL. A case of acute pancreatitis presenting with electrocardiographic signs of acute myocardial infarction. *Pancreatol*. 2003;3(6):515–517.
15. Sakagami J, Kataoka K, Sogame Y, Usui N, Kanemitsu D, Takada R, et al. Increased QT dispersion in patients with alcoholic pancreatitis. *Pancreas*. 2004;28(4):380–386.
16. Stimac D, Tomulic V, Hauser G, Jaklievic C, Radic M. Is there any connection between severity of acute pancreatitis and electrocardiographic changes? *J Clin Gastroenterol*. 2006;40(6):559–560.
17. Patel J, Movahed A, Reeves WC. Electrocardiographic and segmental wall motion abnormalities in pancreatitis mimicking myocardial infarction. *Clin Cardiol*. 1994;17(9):505–509.
18. Tejada JG, Hernandez F, Chimeno J, Alonso MA, Martin R, Bastante T. Acute pancreatitis mimicking acute inferior myocardial infarction. *Angiology*. 2008;59(3):365–367.

19. Hsu PC, Lin TH, Su HM, Lin ZY, Lai WT, Sheu SH. Acute necrotizing pancreatitis complicated with ST elevation acute myocardial infarction: a case report and literature review. *Kaohsiung J Med Sci.* 2010;26(4):200–205.
20. Mautner RK, Siegel LA, Giles TD, Kayser J. Electrocardiographic changes in acute pancreatitis. *South Med J.* 1982;75(13):317–320.
21. Nadkarni N, Bhasin DK, Rana SS, Bahl A, Sinha SK, Rao C, Talwar KK. Diastolic dysfunction, prolonged QTc interval and pericardial effusion as predictors of mortality in acute pancreatitis. *J Gastroenterol Hepatol.* 2012;27(10):1576–1580.
22. Ro TK, Lang RM, Ward RP. Acute pancreatitis mimicking myocardial infarction: evaluation with myocardial contrast echocardiography. *J Am Soc Echocardiogr.* 2004;17(4):387–390.
23. Van de Walle SO; Gevaert SA, Gheeraert PJ, De Pau WM, Gillebert TC. Transient stress-induced cardiomyopathy with an “inverted Takotsubo” contractile pattern. *Mayo Clin Proc.* 2006; 81(11):1499–1502.
24. Altimari AF, Prinz RA, Leutz DW, Sandberg L, Kober PM, Raymond RM. Myocardial depression during acute pancreatitis: fact or fiction? *Surgery.* 1986;100(4):724–731.
25. Lee WK, Frasca M, Lee C, Haider B, Regan TJ. Depression of myocardial function during acute pancreatitis. *Circ Shock.* 1981;8(3):369–374.
26. Kellner A, Robertson T. Selective necrosis of cardiac and skeletal muscle induced experimentally by means of proteolytic enzyme solutions given intravenously. *J Exp Med.* 1954;99(4):387–404.
27. Fulton MC, Mariott HJ. Acute pancreatitis simulating myocardial infarction in the electrocardiogram. *Ann Intern Med.* 1963;59:730–732.
28. Read RC, Doherty JE. Cardiovascular effects of induced insulin hypoglycemia in man during the Hollander test. *Am J Surg.* 1970;119(2):155–162.
29. Krasna MJ, Flancbaum L. Electrocardiographic changes in cardiac patients with acute gallbladder disease. *Am Surg.* 1986;52(10):541–543.
30. Lehmann G, Deisenhofer I, Ndrepepa G, Schmitt C. ECG changes in a 25-years-old woman with hypocalcemia due to hypoparathyroidism. Hypocalcemia mimicking acute myocardial infarction. *Chest.* 2000;118(1):260–262.
31. Lang RM, Fellner SK, Neumann A, Bushinsky DA, Borow KM. Left ventricular contractility varies directly with blood ionized calcium. *Ann Intern Med.* 1988;108(4):524–529.
32. Fox C, Ramsoomair D, Carter C. Magnesium its proven and potential clinical significance. *South Med J.* 2001;94(12):1195–1201.