OXFORD

Oxford Medical Case Reports, 2023, **6**, 215–220
https://doi.org/10.1093/omcr/omad055
Case Report

Necrotizing acute pancreatitis with electrocardiogram changes masquerading acute coronary syndrome

Nirdosh Kumar [1,*], Shahan Waheed[1], Amnah Siddiqui[1] and Ahmed Tamiz[2]

[1]Department of Emergency Medicine, Aga Khan University Hospital Karachi, Pakistan
[2]Department of Cardiology, Aga Khan University Hospital Karachi, Pakistan

*Correspondence address. Department of Emergency Medicine, Aga Khan University Hospital Karachi, Stadium Road PO Box 3500, Karachi, Pakistan. Tel: +923327468542; Fax: +92 21 3493 4294; E-mail: nirdosh.kumar@aku.edu

Abstract

Acute pancreatitis (AP) refers to the acute inflammation of the pancreas; however, if there is concurrent necrosis, it is called necrotizing acute pancreatitis (NAP). The diagnosis is sometimes difficult because it might mimic acute coronary syndrome (ACS). We report a case of a 28-year-old male, who presented to the emergency department (ED) with severe epigastric pain, shortness of breath and diaphoresis for 4–5 h. The initial electrocardiogram (ECG) showed marked sinus bradycardia with an incomplete left bundle branch block. Considering the clinical presentation and ECG changes, he was managed as ACS and was rushed to catheterization laboratory for a coronary angiogram, which was reported normal. Subsequently, his serum pancreatic enzymes were elevated, and computed tomography of the abdomen showed NAP. In ED settings, it is difficult to differentiate between the two, particularly when AP presents with ECG manifestations masquerading as ACS.

INTRODUCTION

Acute pancreatitis (AP) is a common emergency department (ED) presentation with poor outcomes if the diagnosis is delayed with 1.6 deaths per 100 000 persons per year [1, 2]. It is clinically characterized by epigastric pain with elevated serum pancreatic enzymes (amylase and lipase). As per Atlanta classification AP is divided into two categories: interstitial edematous AP and necrotizing acute pancreatitis (NAP). NAP is characterized by inflammation with necrosis of pancreatic parenchyma and peripancreatic tissues. In acute care settings, sometimes it is difficult to clinically differentiate between AP and acute coronary syndrome (ACS) if the former also presents with electrocardiogram (ECG) findings of ACS. Various ECG changes are recognized in AP in the literature such as sinus tachycardia, atrial fibrillation, supraventricular tachycardias, short PR interval, long QT interval, left bundle branch block (LBBB), left anterior hemiblock, and ST-segment abnormalities are approximately seen in 50% of the cases [3]. Here we present a case of a young male who presented with severe epigastric pain, shortness of breath and diaphoresis with bradycardia and LBBB on the ECG. He was initially managed as ACS, but his coronary angiogram was normal. His serum pancreatic enzymes were elevated, and later, a computed tomography (CT) of the abdomen showed NAP.

CASE REPORT

A 28-year-old gentleman presented to the ED with severe epigastric pain and shortness of breath for the past 4–5 h. The pain was sudden onset, severe in intensity and started after eating. It was localized to the epigastrium, non-radiating, and associated with shortness of breath, vomiting and diaphoresis. He was a regular alcohol user for the last 8 years. On physical examination, he was alert but in distress with hypertension and bradycardia. The abdomen was soft, and there was mild tenderness in the epigastrium. He was given nalbuphine 5 mg intravenously (IV), ringer lactate 1 liter bolus IV, aspirin 300 mg per oral (PO), clopidogrel 300 mg PO and heparin 5000 international units IV in the ED. The ECG showed sinus bradycardia with a heart rate of 45 beats per minute, with an incomplete LBBB and nonspecific ST-T changes as shown in (Fig. 1A). Cardiology was consulted and the patient was taken to catheterization laboratory (Cath Lab) for coronary angiography, which was reported normal as shown in the Fig. 2. The blood workup suggested elevated lipase and amylase. The CT of the abdomen showed features consistent with NAP as shown in (Figs 3A–C). General surgery (GS) was consulted, and the GS took over the patient under their service for further management. The patient was treated for NAP with fluids, antibiotics (meropenem), analgesics and antiemetics for 1 week. The patient's general condition improved, he started tolerating enteral nutrition and remained hemodynamically stable therefore discharged and advised for follow-up.

Investigations

A relevant laboratory workup was done which is shown in Table 1. The patient was moved to Cath Lab before the laboratory workup was reported. The coronary angiogram was normal. Subsequently,

Received: March 1, 2023. **Revised:** April 29, 2023. **Accepted:** May 12, 2023

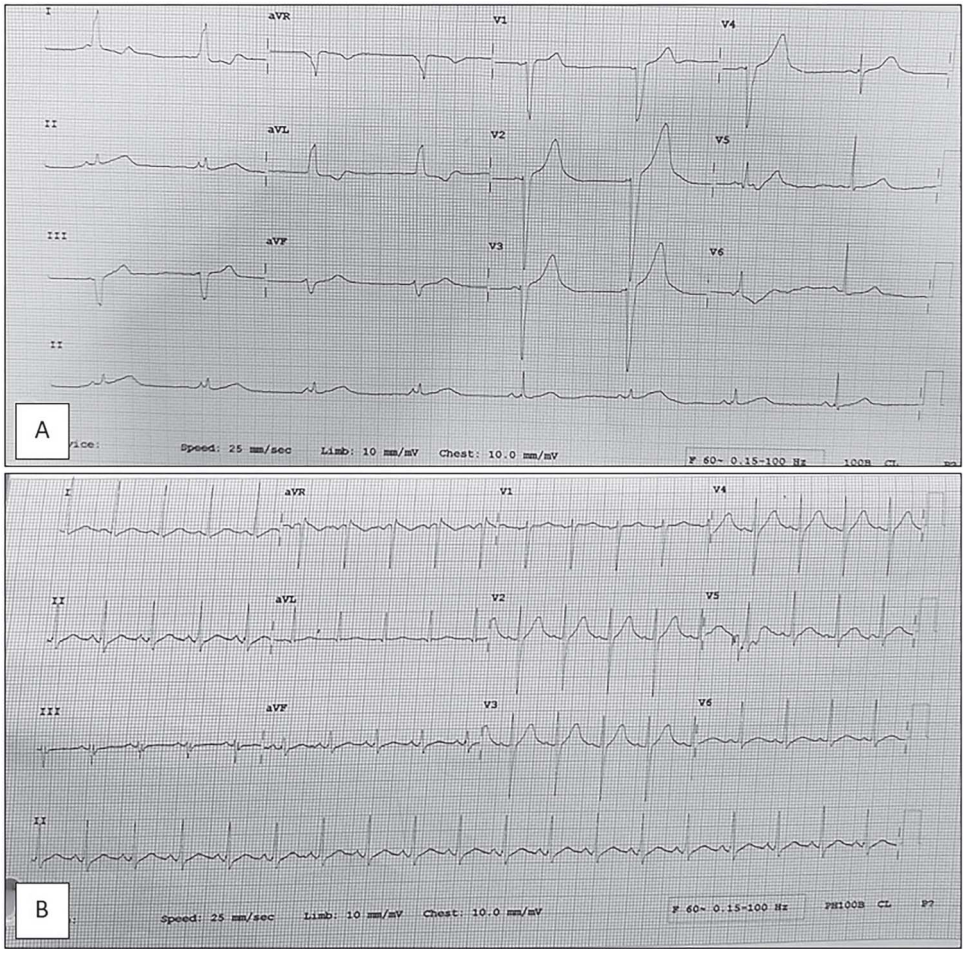

Figure 1. (**A**) Shows sinus bradycardia with a short PR interval, incomplete LBBB, left axis deviation and peaked T-waves in the precordial leads (V2 and V3). (**B**) Shows ECG at the time of discharge and it shows sinus tachycardia with the resolution of peaked T-waves, left axis deviation and incomplete LBBB.

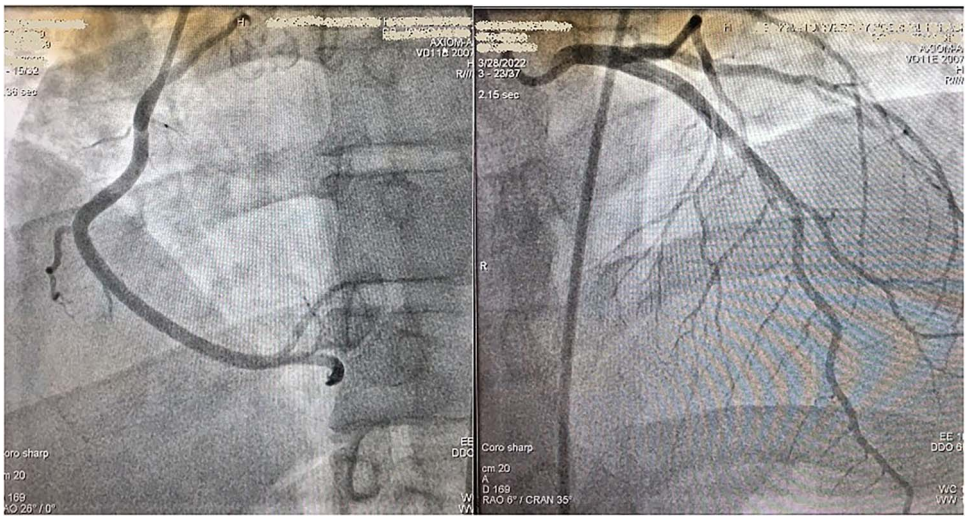

Figure 2. Coronary angiogram showing normal right coronary artery, left mainstem, left anterior descending artery and left circumflex artery.

laboratory workup showed raised lipase. CT abdomen was done, which showed features suggestive of NAP.

Course of hospitalization

After being diagnosed with NAP, the patient was shifted to GS service. He was initially kept in special care unit and managed with intravenous fluids, analgesics and antiemetics. The patient's intake and output were monitored.

A nasojejunal tube (NJT) was inserted because of intolerance to oral feeding, and NJT feed was started, which he tolerated. He was eventually mobilized out of bed. Incentive spirometry and chest physiotherapy were done. In the course of his illness, he

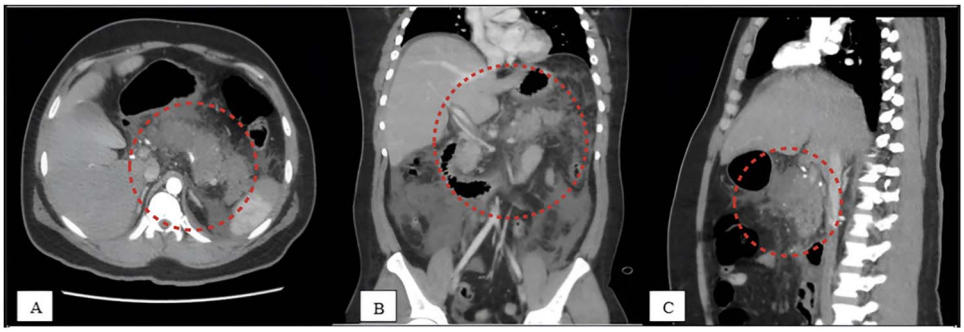

Figure 3. CT abdomen with intravenous contrast ((**A**) axial view, (**B**) coronal view and (**C**) sagittal view) shows a swollen pancreas with significant peripancreatic fat stranding extending to the root of the mesentery and a non-enhancing body of the pancreas representing 30–40% necrosis—features suggestive of acute severe pancreatitis (dotted red circle).

Table 1. Summary of laboratory investigations.

Investigations	Result	Normal value
Hemoglobin	15.9 g/dl	12.3–16.6 g/dl
WBC	20.8 × $10E^9$/L	4.8–11.3 × $10E^9$/L
Neutrophils	92%	34.9–76.2%
Platelets	379 × $10E^9$/L	154–433 × $10E^9$/L
Creatinine	1.2 mg/dl	0.9–1.3 mg/dl
Serum sodium	141 mmol/L	136–145 mmol/L
Serum potassium	4.8 mmol/L	3.5–5.1 mmol/L
Serum bicarbonate	20.4 mmol/L	20–31 mmol/L
Serum chloride	102 mmol/L	98–107 mmol/L
Serum ionized calcium	3.86 mg/dl	4.64–5.28 mg/dl
Serum magnesium	1.9 mg/dl	1.6–2.6 mg/dl
Total bilirubin	0.9 mg/dl	0.1–1.2 mg/dl
AST	30 IU/L	<35 IU/L in male
ALT	59 IU/L	>45 IU/L in male
GGT	185 IU/L	<55 IU/L in male
LDH	374 IU/L	120–246 IU/L
Lipase	2613 U/L	6–51 U/L
Troponin	<2 ng/L	0–57 ng/L male 0–37 ng/L in female

developed a fever spike for which blood cultures were sent, and an intravenous antibiotic (imipenem) was started. He remained hemodynamically stable therefore discharged and advised for follow-up.

DISCUSSION

The ECG changes in AP were first described by Drummond in 1934, which include Sinus tachycardia, atrial fibrillation, LBBB, right bundle branch block and left anterior hemiblock [3]. The ECG changes are transient and revert to baseline once the AP recovers [4]. The new-onset LBBB with acute epigastric pain, shortness of breath and diaphoresis make the diagnosis difficult for AP. The pathogenesis of ECG changes is not clearly understood. There are a few mechanisms proposed to explain the ECG changes in AP like metabolic disturbances such as hypoglycemia, hypokalemia, hyperkalemia, hypomagnesemia and direct injury to the myocardium or pericardium, cardio-biliary reflex, hemodynamic instability, hypocalcemia and coronary artery vasospasm [3] as shown in Fig. 4.

The patients with AP commonly present with acute severe epigastric pain and left upper quadrant abdominal pain reaching the maximum intensity within minutes. The pain persists for hours and may be partially relieved by sitting up or bending forward. The pain is commonly associated with nausea and vomiting. In the case of severe AP, causing inflammation of the diaphragm, the patient might present with associated dyspnea [5]. Contrary to AP, the patients with ACS present with acute onset left-sided chest pain, radiating to the left arm, shoulder and jaw, associated with nausea, vomiting and diaphoresis. The pain in ACS typically aggravates with exertion and relieves with rest. The clinical history and physical examination might help to differentiate between the AP and ACS. However, it is difficult to differentiate between the two in certain cases based on only clinical history and examination [6].

The literature review shows multiple case reports and review articles on ECG changes in AP; however, coronary angiogram was done in a few cases [7–10]. There are cases reported in the literature in which AP and ACS were found simultaneously [11–13]. Table 2 summarizes AP's clinical presentations with cardiac symptoms and outcomes [9, 14]. Overall, 19 cases of AP with signs of acute myocardial infarction (AMI) were reviewed. Among them, 53% were male and 47% were female patients; 15% presented with chest pain and 85% presented with abdominal pain; 16% had LBBB on ECG and 84% had ST elevation; 47% had ST elevation resolved at the time of discharge and 16% had unresolved ECG changes at the time of discharge; 21% had elevated troponin and 38% had negative troponins and 41% had either no troponins checked or were unspecified; 58% went for a coronary angiogram and 90% were discharged with improvement in clinical condition, however, 10% died.

Modified Sgarbossa criteria might help to pick up AMI in the setting of AP with a new onset LBBB as it has a sensitivity of 80% and specificity of more than 90%. Therefore, it is crucial to interpret the ECG in the clinical context. The Modified Sgarbossa was negative in our case; however, the patient was considered to have ACS until proven otherwise because the sensitivity of Modified Sgarbossa is around 80%, which technically means that it can miss 20% of the cases with AMI [15, 16]. Retrospectively, an analysis of the ECGs was done which showed that when the patient was in bradycardia it showed LBBB, which resolved once the heart rate improved as shown in (Fig. 1B). The first case of bradycardia-induced LBBB was presented by Dressler [17]. This is one of the rare cases of bradycardia-induced LBBB conduction

Table 2. Summary of clinical presentations of AP with cardiac symptoms and outcomes.

Study	Year	Age (years)	Gender	Symptoms	ECG changes	Cardiac evaluation	Lab findings	Outcomes
Khan *et al.*	2022	30	F	Severe abdominal pain radiating to back with nausea and vomiting	ST elevation in anterolateral leads	Coronary Angiogram—Normal	Troponin T—5 ng/ml Amylase—1524	Discharged: Takotsubo Cardiomyopathy, S/p sphincterotomy for CBD calculus retraction. ECG changes resolved.
Hajimorad *et al.*	2021	25	M	Severe epigastric pain and left Upper Quadrant pain, nausea and vomiting	ST elevation in V3–V6, I, II and aVF	Coronary Angiogram—Thrombotic lesion in proximal LAD artery and total thrombotic occlusion of mid-LAD artery	Troponin normal Lipase—853 U/l Amylase—1003 U/L	Discharged: Resolution of ST elevation before discharge
Egashira *et al.*	2020	31	F	Upper abdominal pain and fever	ST elevation in V3–V6	Coronary angiogram—normal	Troponin elevated Amylase—370 U/L	Discharged: Fulminant Type 1 DM with Pancreatitis and Myocarditis, ECG changes normalized before discharge
Yu *et al.*	2019	77	M	Chest and back pain, nausea and vomiting	Inferior ST elevation (II, III, aVF)	Coronary Angiogram—Normal	Troponin I—0.02 ng/ml Lipase—3097 U/L	Deceased: Diffuse embolic cerebrovascular accident
Panaylotides *et al.*	2013	76	F	Abdominal discomfort, nausea and vomiting	New onset LBBB	Echo	Negative Troponin Lipase unspecified	Discharged: ERCP without significant findings, resolution of LBBB
Barto *et al.*	2013	60	M	Chest and upper abdomen pain	Inferior ST elevation (II, III, aVF)	Unspecified	Lipase elevated	Unspecified
Meuleman *et al.*	2011	51	M	Upper abdominal pain	ST elevation in Anterior leads (V3–V6)	Coronary Angiogram—Normal	Troponin Negative	Discharged: Resolution of ST elevation
Ullah *et al.*	2010	65	M	Lower central chest and upper abdominal pain	New LBBB with first-degree heart block	None	Troponin Negative Amylase—1210 U/L	Discharged: 1st-degree AV block and LBBB resolved
Cheezum *et al.*	2010	76	F	Nausea, vomiting	ST elevation in the Lateral leads to unspecified	Coronary Angiogram—Normal	Troponin—T—0.67 ng/ml Lipase—>4000 U/L	Discharged: Stress-induced cardiomyopathy
Clementy *et al.*	2010	78	F	Abdominal pain	ST elevation in infero-anterolateral (II, III, aVF, V2–V6	Coronary Angiogram—Normal	Troponin—I—6.6 ng/ml Lipase—3873 U/L	Discharged: Resolution of ST elevation without Q-waves.
Ro *et al.*	2004	43	F	Epigastric pain, nausea and vomiting	Precordial TWI, prolonged QT interval	Echo	Troponin —I—0.73 ng/ml Lipase—763 U/L	Discharged: Resolution of ECG and troponin on 3-week follow-up
Yu *et al.*	2003	71	M	Left upper quadrant pain, nausea, vomiting and diaphoresis	New, evolving LBBB	Coronary Angiogram—Normal	Lipase—2469 U/L Troponin T—Negative	Discharged: SMV Thrombosis, Pancreatic Pseudocyst formation
Wagner *et al.*	2002	56	M	Epigastric pain, nausea and diaphoresis	Anterolateral ST elevation (V2–V4, I, aVL)	None	Unspecified	Discharged: tPA was given 4 h after symptom onset and ST elevation resolved
Khairy *et al.*	2001	64	F	Burning epigastric pain radiating to the chest and back, nausea and vomiting	Anterior ST elevation (V2-V4, TWI aVL, V5-V6)	Coronary Angiogram—Normal	Lipase—5750 U/L Troponins unspecified	Discharged: Persistent ST elevations with diffuse TWI
Patel *et al.*	1994	57	F	Abdominal pain radiating to back, nausea and vomiting	Anterolateral ST elevation (V3–V6, TWI V3 and V4)	Coronary Angiogram—Normal	Amylase—103 U/L Lipase—94 U/L	Discharged: The US shows a mottled pancreatic body and tail. Resolution of ST elevation by Day 5. New TWI in inferior leads
Burge *et al.*	1993	79	F	Severe retrosternal chest pain, nausea, diaphoresis	Complete LBBB	None	Troponins Negative Amylase—>5000 U/L	Discharged: Cholelithiasis, s/p cholecystectomy, ECG changes unchanged on discharge
Cohen *et al.*	1971	41	M	Epigastric pain, nausea and vomiting	Anterolateral ST elevation (V2–V5, TWI I, II, III, aVF, V2–V6)	Coronary Angiogram	Amylase—463 U/L	Discharged: Resolution of ST elevations, and precordial TWI
Fulton *et al.*	1963	61	M	Upper abdominal pain, nausea and vomiting	Anterior ST elevation (V2—V4)	Autopsy	Amylase—370 U/L	Deceased: Severe necrotizing pancreatitis
Shamma's *et al.*	1962	43	M	Epigastric pain with bilious vomiting	ST elevation in Inferior leads (III, aVF, TWI, I, III, aVF	None	Amylase—2279 U/L	Discharged: Resolution of amylase and leukocytosis within 1 week

M, male; F, female; Echo, echocardiograph; tPA, tissue plasminogen activator; TWI, T-wave inversion.

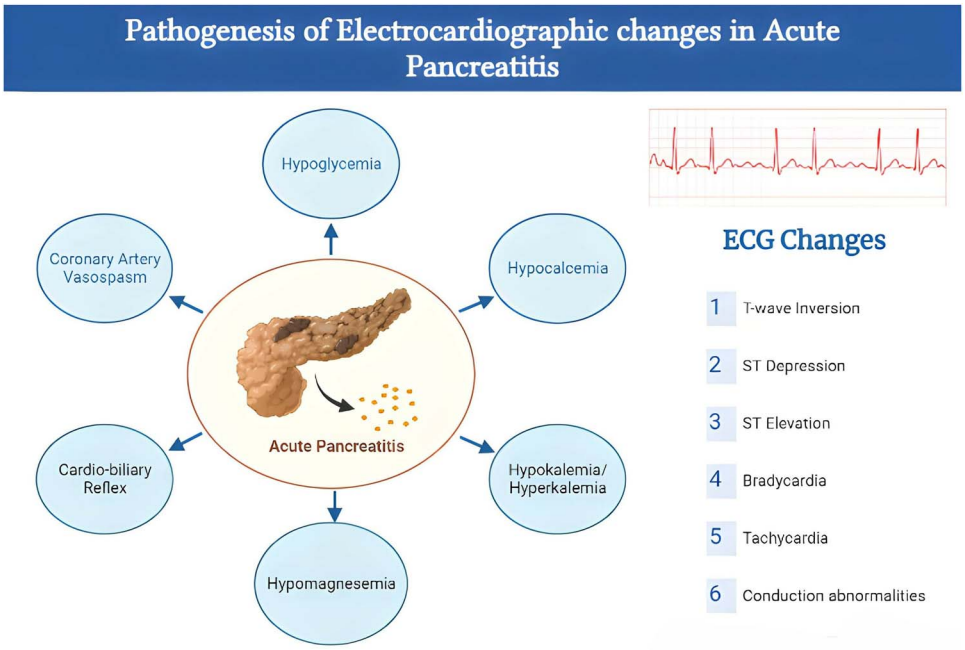

Figure 4. Shows pathogenesis of the ECG changes in AP.

pattern associated with AP masquerading ACS. This case also reinforces the significance of Modified Sgarbossa criteria for the diagnosis of ACS in the presence of LBBB.

ABBREVIATIONS

AP—acute pancreatitis.
NAP—necrotizing acute pancreatitis.
AMI—acute myocardial ischemia.
Cath Lab—catheterization laboratory.
LBBB—left bundle branch block.
ECG—electrocardiogram.
CT—computed tomography.
GS—general surgery.
CCU—cardiac care unit.
ACS—acute coronary syndrome.
NJT—nasojejunal tube.
WBC—white blood count.
AST—aspartate transaminase.
ALT—alanine transaminase.
GGT—gamma-glutamyl transferase.

ACKNOWLEDGEMENTS

None.

CONFLICT OF INTEREST STATEMENT

There are no conflicts of interest to declare.

FUNDING

None.

ETHICAL APPROVAL

Standards concerning publication ethics were observed, and the study was approved by the hospital's ethical review committee.

CONSENT

The patient gave consent to the publication.

GUARANTOR

I, Dr Nirdosh Kumar, accept official responsibility for the overall integrity of the manuscript and attest that all statements in the manuscript are true to my knowledge.

REFERENCES

1. Li CL, Jiang M, Pan CQ, Li J, Xu LG. The global, regional, and national burden of acute pancreatitis in 204 countries and territories, 1990–2019. *BMC Gastroenterol* 2021;**21**:1–12.
2. Xiao AY, Tan MLY, Wu LM, Asrani VM, Windsor JA, Yadav D *et al.* Global incidence and mortality of pancreatic diseases: a systematic review, meta-analysis, and meta-regression of population-based cohort studies. *Lancet Gastroenterol Hepatol* 2016;**1**:45–55.
3. Yegneswaran B, Kostis JB, Pitchumoni CS. Cardiovascular manifestations of acute pancreatitis. *J Crit Care* 2011;**26**:225.e11–8.
4. Patel J, Movahed A, Reeves WC. Electrocardiographic and segmental wall motion abnormalities in pancreatitis mimicking myocardial infarction. *Clin Cardiol* 1994;**17**:505–9.
5. *Clinical manifestations and diagnosis of acute pancreatitis - UpToDate*. https://www.uptodate.com/contents/clinical-manifestations-and-diagnosis-of-acute-pancreatitis?search=acute%20pancreatitis&source=search_result&selectedTitle=2~150&usage_type=default&display_rank=2 (20 August 2022, date last accessed).
6. *Diagnosis of Acute Myocardial Infarction - UpToDate*. https://www.uptodate.com/contents/diagnosis-of-acute-myocardial-infarction?search=acute%20myocardial%20infarction&source=search_result&selectedTitle=2~150&usage_type=default&display_rank=2 (20 August 2022, date last accessed).

7. Meuleman VG, Schinkel AFL, Vos J. Electrocardiographic abnormalities caused by acute pancreatitis. *Neth Hear J* 2011;**19**:137.
8. Antonelli D, Rozner E, Turgeman Y. Unusual electrocardiographic changes during acute pancreatitis. *Cor Vasa* 2017;**59**:e446–9.
9. Yu ES, Lange JJ, Broor A, Kutty K. Acute pancreatitis masquerading as inferior wall myocardial infarction: a review. *Case Rep Gastroenterol* 2019;**13**:321–35.
10. Long Y, Tang M, Wang J, Liu H, Jian Z, Li G *et al.* Case report: abnormal ECG in a patient with acute pancreatitis. *Front Cardiovasc Med* 2021;**8**:2029.
11. Kumar AV, Mohan Reddy G, Anirudh Kumar A. Acute pancreatitis complicated by acute myocardial infarction – a rare association. *Indian Heart J* 2013;**65**:474.
12. Zubin S, Ravindra Pradeep S, Divya S. Acute pancreatitis with uncommon presentation of myocardial infarction. *Ann Clin Gastroenterol Hepatol* 2020;**4**:034–5.
13. *Acute Pancreatitis-Induced Frank Myocardial Infarction: An E... : Official Journal of the American College of Gastroenterology | ACG*. https://journals.lww.com/ajg/Fulltext/2017/10001/Acute_Pancreatitis_Induced_Frank_Myocardial.2820.aspx (11 June 2022, date last accessed).
14. Khan U, Petrechko O, Sagheer S, Majeed H, Zaidi SH, Wasty N *et al.* Acute pancreatitis and myocardial infarction: a narrative review. *Cardiology* 2023;**148**:1–11.
15. Seibolt L, Maestas C, Lazkani M, Fatima U, Loli A, Chesser M. Rate-related left bundle branch block and cardiac memory in a patient with bradycardia: case report and literature review. *Clin Cardiol* 2018;**41**:1097.
16. *Electrocardiographic diagnosis of myocardial infarction in the presence of bundle branch block or a paced rhythm - UpToDate*. https://www.uptodate.com/contents/electrocardiographic-diagnosis-of-myocardial-infarction-in-the-presence-of-bundle-branch-block-or-a-paced-rhythm?search=sgarbossa%20criteria&source=search_result&selectedTitle=1~2&usage_type=default&display_rank=1#H9 (20 August 2022, date last accessed).
17. Myburgh DP, van Gelder AL. Bradycardia-dependent bundle-branch block. *Circulation* 1968;**47**:1933–5.