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Case and Review

Acute Pancreatitis Masquerading as Inferior Wall Myocardial Infarction: A Review

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Keywords

Pancreatitis · Cardiac catheterization · ST elevation · Myocardial infarction · Coronary angiography · Cerebral vascular accident · Stroke

Abstract

A rare presentation of acute pancreatitis is with electrocardiographic (ECG) changes that mimic myocardial ischemia. We present a report of a patient that presented with hemodynamic instability and new ECG changes of ST segment elevations in contiguous leads II, III, and aVF mimicking an inferior wall myocardial infarction. Emergent coronary angiography showed no significant coronary obstruction, but it was followed by a left-sided hemiplegia with radiographic evidence of diffuse embolic stroke. The patient was later found to have an underlying diagnosis of pancreatitis. Additional history that later became available indicated a history of severe acute pancreatitis treated elsewhere a few months prior to the current admission. We present the first comprehensive review of the literature comprising 36 total cases with pancreatitis masquerading as acute myocardial infarction, with inferior wall STEMI pattern being the most frequent. We present this case to highlight the diagnostic dilemma posed by this masquerade of a high acuity myocardial infarction and to highlight alternative diagnoses to be considered in such clinical circumstances.

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Introduction

The adage, “Appearances may be deceptive,” is exemplified by the propensity of some acute abdominal conditions to mimic acute myocardial ischemia. Acute pancreatitis takes this one step further by producing ST segment elevation in an electrocardiogram (ECG), thus mimicking ST elevation myocardial infarction (STEMI). ECG detects acute myocardial infarction with 81% sensitivity and 69% and specificity [1]. Although ECGs manifest T-wave and ST segment abnormalities in a quarter of cases of acute pancreatitis [2], one mimicking a STEMI is distinctly rare.

We recently cared for a man with pancreatitis, whose clinical history and objective data supported a high suspicion of a STEMI; emergent cardiac catheterization and coronary angiography revealed unremarkable coronary arteries. His subsequent course indicated that he had acute pancreatitis that spuriously manifested STEMI. To ascertain the prevalence of this phenomenon, we conducted a PubMed search for key words of acute pancreatitis, electrocardiogram changes, mimicry, ST elevations, catheterization, myocardial infarction, and thrombolytic agents. We included cases with an underlying diagnosis of acute pancreatitis that presented, wherever possible, with 12-lead ECG changes suggestive of acute myocardial infarction with resolution of the ECG changes during admission, or exclusion of acute coronary syndrome either by coronary angiogram or autopsy. We omitted reports with concomitant myocardial infarction complicated by acute pancreatitis. Our yield of 36 studies is shown in [Table 1](#), [Table 2](#), and [Table 3](#).

Case Report

A 77-year-old, non-English-speaking Hmong man presented for acute chest and back pain with vomiting. His daughter-in-law provided much of the history. The patient came to her door around midnight, complaining of chest, back pain, and nausea with vomiting. He had coronary artery disease treated with coronary bypass (CABG, with LIMA-LAD, and SVG-LCX grafts), hyperlipidemia, essential hypertension, type 2 diabetes mellitus, ascending aortic aneurysm, left ventricular (LV) mural thrombus (on warfarin), stage 3 chronic kidney disease, and asthma. She contacted paramedics, who noted tachycardia, BP 60/40 mm Hg, and SpO₂ of 80% on room air. Initial ECG was concerning for inferior wall STEMI. He arrived in the ED with pulse of 108, BP 83/50, and requiring 15 L of oxygen on a non-rebreathing mask. Basic CBC and BMP were unremarkable with a normal troponin I level of 0.02 ng/mL; his INR was therapeutic at 2.2. A 12-lead ECG showed ST elevations in leads II, III, and aVF with T wave inversions in aVL and anteroseptal leads ([Fig. 1](#), confirmed by a cardiologist), which was new from previous ECGs. He was taken to emergent right femoral heart catheterization and coronary angiography within 30 min of arrival and was administered 5 mg of i.v. vitamin K. No new coronary artery disease was found; his grafts were patent and his LV ejection fraction (LVEF) was estimated at 60%. The cardiologist concluded that there was no STEMI and that his presentation and hypotension were from a non-cardiac etiology.

The medicine team first evaluated him in the ICU post coronary angiography catheterization. He was sedated and lethargic. His left side was flaccid; on pain stimulation, he only moved his right side. With his daughter translating, the team noted that he could follow one-step

commands with his right hand, but not with his left. The left nasolabial fold was effaced and a left facial droop was now evident; these findings prompted a stroke alert. Clinically, the right upper abdomen and epigastrium were tender, prompting serum lipase determination and an ultrasound exam. A priority head CT without contrast showed no hemorrhage. Follow-up CT angiogram showed decreased vascularity through the right middle cerebral artery (MCA) superior distribution and right cortical posterior communicating artery (PCA). An extensive, irregular, non-calcified plaque along the aortic arch extending into the brachiocephalic trunk, left common carotid artery, and left subclavian artery were highly concerning for an embolic source (Fig. 2a). Follow-up brain MR imaging without contrast showed extensive areas of acute ischemia of cerebral and cerebellar hemispheres (right greater than left) with predominant right MCA and bilateral PCA distributions (Fig. 2b). The neurologists recommended aspirin and statin therapy only. His diffuse cerebrovascular accident precluded tissue plasminogen activator, neuro-intervention, or therapeutic anticoagulation.

Our lab now reported a serum lipase of 3,097 U/L. A lipid panel revealed triglycerides of 300, total cholesterol 218, HDL 35, and LDL 123. An ultrasound of abdomen showed neither biliary dilation nor gallstones; overlapping bowel gas precluded visualization of the pancreas. Nascent contrast use and history of CKD precluded a CT of abdomen and pelvis. Transthoracic echocardiogram showed a LVEF of 42% along with regional wall hypokinesis of the apical wall without basal hyperkinesis. The previous LV thrombus was unchanged despite being on warfarin.

As his clinical course unfolded, we also learned that he had been at another local hospital 3 months previously for gallstone-induced pancreatitis; this was documented by a peak serum lipase of 59,153 U/L, alkaline phosphatase of 220 U/L, abdominal ultrasound showing mild gallbladder distension with biliary sludge present, and fluid accumulation and stranding around the body and tail of the pancreas on CT angiogram (adjacent bowel artifacts obscured any gallstones). No evidence of necrosis or abscess was noted on the radiologist assessment. Elective cholecystectomy was offered, but the patient declined. Lipase declined upon discharge to 616 U/L.

We administered i.v. maintenance fluids with adequate urine output of at least 100 mL/h; Progressive respiratory failure and endotracheal intubation and mechanical ventilation followed believed to be from anoxic cerebral and brainstem injury. No surgical consult was considered due to the patient's poor prognosis. His hospital course was complicated by fever; however, blood and urine cultures remained sterile. *Serratia marcescens* and methicillin-sensitive *Staphylococcus aureus* were isolated from his endotracheal tube aspirates, thus leading to a short-course antibiotic therapy, which was later abandoned, with the fever being attributed to central dysregulation from his diffuse stroke. Despite withholding sedation, the patient was unable to regain consciousness. His family was apprised of his poor prognosis. They elected not to pursue further life support and the patient was terminally extubated. He was transferred to hospice for comfort care and passed away 13 days after his initial admission.

Review

To our knowledge, we present the first comprehensive review of the literature on pancreatitis mimicking myocardial infarction on ECG. Although ischemic ECG findings have been known to present in pancreatitis from the 1930s, these generally lacked 12-lead ECG data and standardized nomenclature [3, 4]. Since Bauerlein and Stobbe in 1954 [5], 36 instances of such mimicry have been noted, as shown in Table 1, Table 2, and Table 3 [5–38] (clinical course, diagnostic testing, and patient outcome was not specified in one report and was omitted in the prevalence values for the respective categories). An inferior wall infarction pattern appears to be the most frequent (44.4%, or 16/36 cases). Twelve of 36 (33.3%) involved an anterior wall pattern. In 4 cases, a new-onset left bundle branch block was found and in another 4, a pattern of Takotsubo cardiomyopathy, duly confirmed by echocardiogram. Chest pain was reported as a presenting symptom in 36.1% (13/36) of cases. Troponins were reported in 24 cases and considered elevated in 41.6% (10/24). Nineteen patients (54.3%, or 19/35) underwent coronary angiography during admission. Given the concern of myocardial infarction, 11.4% (4/35) of cases were administered thrombolytics. There were 7 known deaths (20% or 7/35, one report did not specify), mostly from severe necrotizing pancreatitis. In patients with clinical improvement, half (14/28) of the observed ECG changes resolved during admission or upon follow-up.

Discussion

In a patient with pancreatitis presenting with ECG features that show ST elevation, the clinical decision-making involves risks of both action and inaction. The risk of inaction is that one misses the diagnosis, and therefore proper treatment, of an acute myocardial infarction. Accepting the diagnosis of STEMI sets in motion the process of percutaneous coronary intervention (PCI) that encompasses coronary angiography and the possibility of angioplasty and stent placement if possible, with the latter necessitating therapy to prevent stent thrombosis, often for at least a year. Several risks of PCI include death, stroke, infection, and hematoma, and if certain stents are used, the necessity to avoid elective surgery for at least a year. While the relative contraindications to PCI (Fig. 3) [39] are all potentially reversible, the sheer acuity of a STEMI and the implicit need to undertake PCI that it portends trumped the needs and even the potential benefits of getting a licensed interpreter just to get a better history. Although the patient's daughter was present, her English and medical experience was limited. Her initial impressions were that her father's presentation was similar to his NSTEMI presentation a year prior rather than resembling his episode of previous gallstone pancreatitis. The door-to-balloon time in PCI is generally accepted as below 90 min, some even prefer a shorter period to enhance outcomes. Even if one were to avoid PCI and were to resort to thrombolytics such as tissue plasminogen activator instead, the resulting conversion of an acute inflammatory pancreatitis into a hemorrhagic pancreatitis with potential consequences [22, 29, 34, 40, 41] may not constitute absolute contraindication to treating STEMI. In essence, the lethality of a STEMI demands action.

Our patient unfortunately developed a stroke following PCI via a femoral artery approach. A radial artery approach might have subdued this risk, as demonstrated by the CARAFE and RIVAL studies that produced improved patient comfort, decreased cost, enhanced comfort

during procedure, and lower rate of acute myocardial infarction, stroke, and death within 30 days of procedure via this technique [42, 43]. Disturbing the aortic atheromatous plaques was the most likely reason in our patient, although disruption of the irregular non-calcified plaque and its extension into the brachiocephalic trunk is also possible. It is unknown if a radial artery approach would have disrupted the latter any less.

Pathogenesis of Electrocardiogram Changes

Besides acute pancreatitis, many other non-ACS conditions have been known to evoke ST elevations in contiguous ECG leads to mimic myocardial infarction (Fig. 4) [44, 45]. Gu et al. [44] found that of 820 (2.3%) consecutive patients with suspected STEMI who were referred for primary PCI over an 18-month period, 19 (2.3%) had a final diagnosis other than myocardial infarction. Notably, only 7 of these had a non-cardiac condition, and none of them had pancreatitis as the underlying condition [44]. Rubio-Tapia et al. [46] reported that acute pancreatitis evokes changes in ECG from baseline in 55% of confirmed cases, the most common changes being nonspecific repolarization, sinus tachycardia, and left anterior hemiblock. These polarization changes could be related to electrolyte abnormalities that frequently complicate pancreatitis – lower phosphorus (especially in those with associated sinus tachycardia), hypocalcemia (QT prolongation), hypokalemia (vomiting), hyperkalemia (with onset of acute renal failure), hypomagnesemia (which can cause coronary vasospasm and stunning of the myocardium to resemble ischemic-appearing ECG changes) [46]. Both extremes of potassium can induce ECG changes, with U waves, and flattened T waves with hypokalemia along with risk of ventricular tachycardia. Hyperkalemia results in peaked T waves and QRS prolongation. No correlation was noted in the levels of amylase and lipase between pancreatitis patients with normal and abnormal ECG findings [46].

Severe cases of pancreatitis can lead to hypoperfusion of organs and promote a sympathetic response that can stress the heart and unmask underlying coronary ischemia. Furthermore, a Takotsubo stress cardiomyopathy can also occur during critical illness that can lead to cardiac depression and ECG changes from catecholamine excess and sympathetic stimulation. By definition, a stress cardiomyopathy is a completely reversible LV dysfunction not investigated by coronary artery disease. Cheezum, Rajani, and others report pancreatitis-induced classic Takotsubo findings with apical akinesis with hyperbasal contraction and normal coronary arteries [11, 13, 19, 21]. Triggers can range from emotional distress and systemic illness with an estimated incidence of approximately 27 and 38%, respectively [47]. Coronary vasospasm can also induce ECG changes associated with changes in autonomic activity [48] that can also hypothetically occur during acute pancreatitis.

Interrelated to pancreatitis, there have been several reported cases of ischemic mimicry occurring in cholecystitis patients [10, 49–51]. Morrison and Swalm [52] proposed a cardiobiliary reflex that results in a vagal nerve-mediated attenuation of inotropy stemming from pain stimulus in the gall bladder. Sustained vagal nerve stimulation of 31–45 hrs. has been found to induce histological myocardial injury that can be attenuated by infusion of atropine in dogs [53]. In humans, it has also been found that increased pancreatic enzyme secretion is inducible by vagal stimulation [54]. Sorić et al. [55] caution both calculous and acalculous cholecystitis should be considered in a patient that has unexpected sinus arrest and bradycardia with abdominal pain.

The proposed pathogenesis of pancreatitis involves intra-acinar activation of proteolytic enzymes that result from accumulation of active trypsin in the pancreas. Lysosomal enzymes destabilize vacuoles which rupture and release trypsin that inappropriately activates dormant zymogens resulting in autodigestion of the pancreatic tissue before appropriate release into the duodenum [56]. Injections of exogenous proteolytic enzymes of trypsin, ficin, and papain into rabbits result in focal necrosis of cardiac and skeletal muscle on histology [57]. In a mouse model, harvested organs were pretreated with physiological concentrations of digestive pancreatic enzymes *in vitro*. Heart, liver, brain, spleen, intestine, diaphragm, kidney, and lung tissues produced significantly increased cellular activation and inflammatory signaling with neutrophil pseudopod formation when exposed to trypsin. Chymotrypsin digests of cardiac tissue exclusively also increased levels of pseudopod formation. Lipase-treated heart, kidney, and lung tissue demonstrated significant increase in cell death in comparison to controls [58]. Pleural and pericardial effusions have also arisen transiently during cases of acute pancreatitis [59] and it has been postulated that lymphatic drainage via the thoracic duct may provide an avenue for digestive enzymes to injure the myocardium [60, 61]. However, a localized transdiaphragmatic epicardial cardiac inflammation from pancreatitis has been more favored [15, 17] as isolated lymph from pericardial fluid obtained during an acute pancreatitis episode lacked detectable lactate dehydrogenase and amylase [62]. The majority of reports (Table 1, Table 2, Table 3) appear to have a propensity for mimicking an inferior wall STEMI with elevations in leads II, III, and aVF. Transdiaphragmatic passage of inflammation may anatomically explain why a large proportion of pancreatitis cases mimic inferior wall ischemia due to the posterior, retroperitoneal location of the pancreas.

Conclusion

Information available to the emergency department and STEMI teams posed no absolute contraindication for our patient to undergo coronary angiography. In retrospect, risk factors can be assessed with more clarity, but given the patient's extensive coronary history and risk factors of hyperlipidemia, diabetes, and known hypercoagulable state with LV thrombus on therapeutic warfarin, a clinical suspicion of ACS was high. Additionally, the patient's calculated Thrombolysis in Myocardial Infarction (TIMI) score estimated a 30-day mortality rate of 36%. The diagnostic dilemma arises when a patient presents with clinical signs of ACS to recall that findings of ST elevations on ECG do not have absolute specificity. In the current situation of our inability to exclude a very serious problem of myocardial infarction, it seems the logical course to follow would be one of PCI, with all its attendant risks rather than assume that the ECG changes are due to pancreatitis.

It is our hope that we highlight some of the challenges that emergency physicians and hospitalists face when triaging high-acuity patients. We would emphasize that forming differentials are imperative for a practicing clinician even in the most stressing situations. Other causes, specifically gastrointestinal pathologies, can mimic characteristics similar to myocardial infarction. Percutaneous intervention is the standard of care for myocardial infarction, yet it is an invasive procedure with its own inherent risks. Clinicians should remain cognizant of alternative diagnoses to appropriately weigh the risk and benefits of proceeding with a potential intervention.

Statement of Ethics

The authors declare no ethical conflict. No ethical approval was required for publication of this case report. The patient was deceased and exhaustive attempts were made to contact next of kin without success. Discussion of the intellectual benefit was discussed with the editors of this journal to proceed with publication.

Disclosure Statement

Elliot S. Yu, Joel J. Lange, and Apoorv Broor declare no conflict of interest other than employment (Dr. Yu: Medical College of Wisconsin Affiliated Hospitals, Milwaukee, WI, USA, and Dr. Broor: Ascension St. Joseph, Milwaukee, WI, USA); Dr. Kesavan Kutty is employed by the Medical College of Wisconsin and serves as consultant at Ascension St. Joseph, Milwaukee, WI, USA, and has stock holdings at Medtronic, Inc., of USD >10,000, which has not in any way influenced the manuscript.

Author Contributions

Elliot S. Yu was involved in clinical care and conceptualized the format and writing of the manuscript. Joel J. Lange was on the clinical team and conducted a literature review and formation of the supplementary table along with revision and editing. Apoorv Broor was involved in clinical care and revision and review of the manuscript. Kesavan Kutty oversaw and edited the manuscript and provided additional clinical input and experience.

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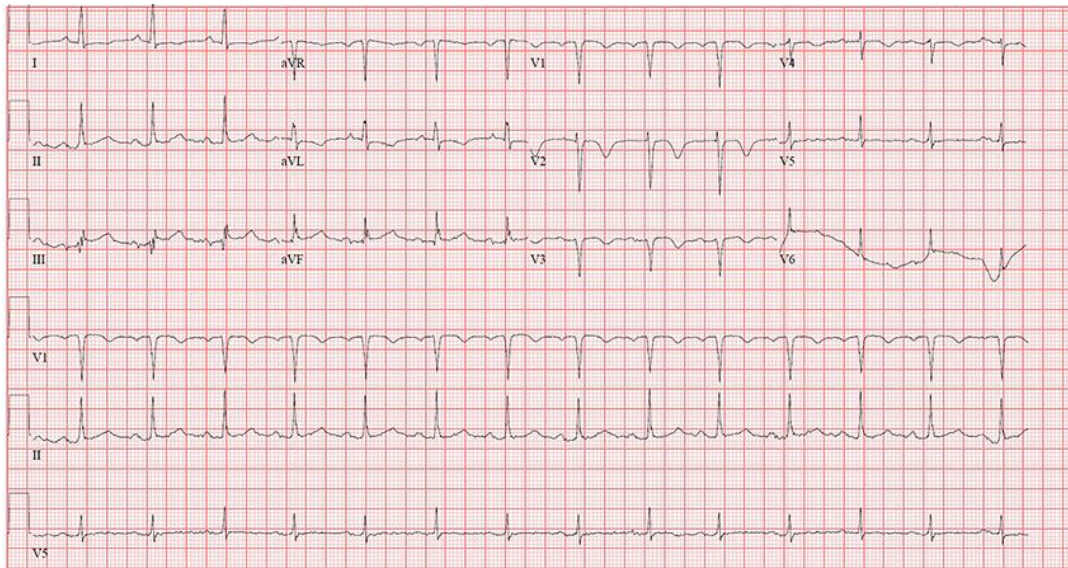


Fig. 1. ECG ST elevations in leads II, III, and aVF, T wave inversions in leads aVL and V1–V4.

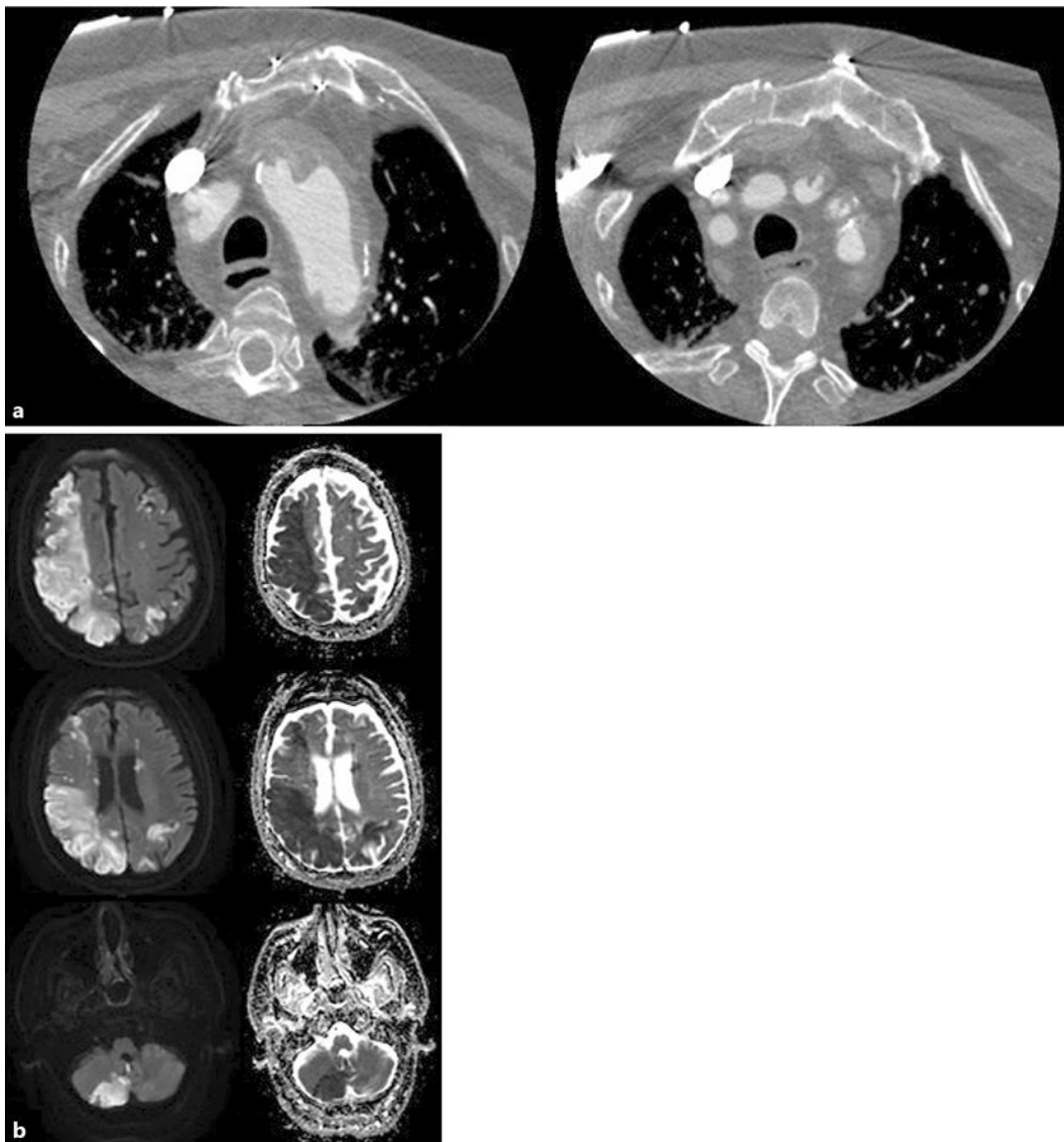


Fig. 2. **a** CTA head and neck with contrast. Diffuse irregular noncalcified mural plaque surrounding the aortic arch and great vessels of the aorta with ascending aortic aneurysm. **b** Brain MRI without contrast. Diffusion-weighted imaging (left), apparent diffusion coefficient (right). Extensive areas of acute ischemia involving more the right than left cerebral and cerebellar hemispheres with predominant right MCA and bilateral PCA distributions, most compatible with embolic etiology infarcts.

- Acute Renal Failure
- Chronic renal failure secondary to diabetes
- Acute Gastrointestinal bleed
- Unexplained fever
- Untreated active infection
- Acute stroke
- Severe anemia
- Severe, uncontrolled hypertension
- Severe electrolyte imbalance
- Refusal for definitive therapy
- Digitalis intoxication
- Anaphylactic drug reaction to contrast media
- Severe peripheral vascular disease (Limited Vascular access)
- Decompensated congestive heart failure or pulmonary edema
- Severe coagulopathy
- Aortic valve endocarditis

Fig. 3. ACC/AHA guidelines for relative contraindications to coronary angiography.

- Coronary Aneurysm
- Pericarditis
- Myopericarditis
- Cardiomyopathy
- Brugada Syndrome
- Aortic Stenosis
- Subarachnoid Hemorrhage
- Pneumonia
- COPD
- Mediastinal Tumor
- Peritonitis
- Pancreatitis
- Cholecystitis

Fig. 4. Alternative pathologies that can mimic myocardial ischemia.

Table 1. Reports of myocardial ischemic mimicry in acute pancreatitis

Report, year	Age, sex	Symptoms	ST elevation leads	Lab values	Cardiac evaluation	Outcomes
Yu [present report], 2019 (C,D,H,L,R)	77 years, Asian man	Chest and back pain, nausea and vomiting	Inferior II, III, aVF	Negative initial troponin I: 0.02 ng/mL Lipase: 3,097 U/L	Coronary angiography, echocardiogram	(D), diffuse embolic cerebrovascular accident
Agrawal [6], 2018	60 years, Asian man	Epigastric pain with syncope	Inferior II, III, aVF, V3 (2 mm)	Negative initial troponin Lipase 25,304 U/L	Coronary angiography	(I), persistent V2–V3 ST elevations
Antonelli [7], 2017 (N)	46 years, man	Nausea, mild upper abdominal pain radiating to the back, chest discomfort radiating to the neck	Large peaked T waves II, III, aVF, large T wave inversions aVL, V1–V6	Negative serial troponin T: <14 ng/mL Lipase: 4,192 U/L Amylase: 2,311 U/L	Coronary angiography	(I), resolution of T wave changes half an hour after initial presentation; gallstones visualized on ultrasound with follow-up cholecystectomy
Villa [8], 2017 (N)	21 years, woman	Sternal chest pain radiating posteriorly, vomiting, diarrhea	ST depression: II, III, aVF, V3–V6	Negative high-sensitive troponin T Lipase: 1,116 U/L Amylase: 1,136 U/L	Echocardiogram	(I), regression of ST depressions the day after presentation
El-Khabiry [9], 2016 (D,L)	27 years, woman	Upper abdominal pain	Anterior V4–V6	Negative troponins, unspecified Lipase: 1,163 U/L	Echocardiogram	(I), resolution of ST elevations within 2 h
Sethi [10], 2016 (H,L)	56 years, woman	Midsternal chest pain, nausea and vomiting, diaphoresis	Inferior II, III, aVF	Elevated serial troponins 6.0, 1.2, 0.87 Lipase: 9,080 U/L	Coronary angiography	(I), resolution of inferior ST elevations
Sethi [10], 2016 (A)	28 years, man	Midsternal chest and epigastric pain radiating to axilla and back	Inferior II, III, aVF, V5–V6	Elevated serial troponins: 4.0, 1.96, 0.86 Lipase: 4,256 U/L	Coronary angiography	(I), resolution of ST elevations
Bruenjes [11], 2015 (A,H)	55 years, African American man	Nonradiating epigastric pain and left lower sternal border	ST depression and TWI: I, II, III, aVF, V1–V6	Elevated serial troponin I: 0.29 ng/mL, 0.658 Lipase: 773 U/L Amylase: 97 U/L	Coronary angiography, echocardiogram	(I), Takotsubo cardiomyopathy, normalization of ST-T segment depression with improving TWI on discharge
Khan [12], 2014	78 years, man	Nonexertional epigastric pain, nausea, diaphoresis	Inferior II, III, aVF	Negative serial troponin T: <0.01, <0.01, 0.02 µg/L; Lipase: >40,000 U/L	Coronary angiography	(D), necrotizing pancreatitis + acute respiratory distress syndrome + vasodilatory shock
Leubner [13], 2014 (D,H,L)	76 years, woman	Epigastric pain, nausea and vomiting	Anteroseptal, leads unspecified	Elevated troponin peak: 9.94 ng/mL, elevated troponin on presentation	Coronary angiography, echocardiogram	(I), anteroseptal hypokinesia consistent with Takotsubo cardiomyopathy, s/p laparoscopic cholecystectomy, ERCP and sphincterotomy
Barto [14], 2013 [†] (D,H,L)	60 years, man	Chest and upper abdominal pain	Inferior II, III, aVF	Unspecified troponin Lipase elevated, unspecified	Unspecified	Unspecified
Panayiotides [15], 2013	76 years, Caucasian woman	Abdominal discomfort, nausea and vomiting	New-onset left bundle branch block, 24 h after admission	Negative troponin T, unspecified Lipase unspecified	Echocardiogram	(I), s/p ERCP without significant findings, resolution of LBBB on 3-week follow-up

(D), deceased; (I), discharged after clinical improvement. Reported risk factors key: A, alcohol use; C, coronary artery disease; D, diabetes; H, hypertension; L, hyperlipidemia; N, no specified cardiac risk factors; R, renal disease; S, smoking use. * Potassium levels reported within normal limits (range 3.61–4.68 mmol/L) during admission. [†] Clinical course, diagnostic testing, and patient outcome was not specified in this report and was omitted in the prevalence values for the respective categories. [‡] Amylase levels originally reported as Somogyi units; these values were converted to metric units.

Table 2. Reports of myocardial ischemic mimicry in acute pancreatitis (continued)

Report, year	Age, sex	Symptoms	ST elevation leads	Lab values	Cardiac evaluation	Outcomes
Meuleman [16], 2011 (A)	51 years, man	Upper abdominal pain	Anterior V3–V6	Negative troponins, unspecified	Coronary angiogram, echocardiogram	(I), 4-day resolution of ST elevations in V4–V6, V2–V3 persistent elevations
Ullah [17], 2010	65 years, man	Lower central chest and upper abdominal pain	New LBBB with 1st degree heart block	Negative serial troponin, unspecified Amylase: 1,210 U/L	None	(I), 1st degree AV block and LBBB resolved 15 min after presentation
Oleszewski [18], 2010	39 years, man	Abdominal pain, nausea and vomiting	Inferior II, III, aVF	Negative serial troponins, unspecified Amylase and lipase elevated, unspecified	Echocardiogram	(I), ST elevations resolved over 60 min
Cheezum [19], 2010	76 years, woman	Nausea and vomiting, tachypnea a day after admission	Lateral, leads unspecified	Elevated initial troponin T: 0.67 ng/mL Lipase: >4,000 U/L	Coronary angiography, left ventriculography	(I), stress induced cardiomyopathy (Takotsubo) with severe apical hypokinesis and hyperdynamic basal contraction, s/p MRCP and ERCP
Clementy [20], 2010 (A)	78 years, woman	Abdominal pain	Infero-antero-lateral II, III, aVF, V2–V6	Elevated troponin I: 6.6 ng/mL Lipase: 3,873 IU	Echocardiogram, cardiac MRI, delayed coronary angiography 1 month later	(I), 8 days later ST elevations resolved without Q waves, but newly developed TWI II, III, aVF, V2–V6
Rajani [21], 2010	72 years, woman	Acute abdominal pain	Diffuse TWI in leads V4–V6, II, III, aVF	Elevated troponin T: 0.32 µg/L Amylase: 1,237 U/L	Coronary angiography, left ventriculography	(I), left ventricular apical akinesis consistent with Takotsubo cardiomyopathy
Low [22], 2009 (D,H)	48 years, man	Crushing chest pain, diaphoresis, dyspnea	Inferior II, III, aVF	Unspecified troponin Lipase: 2,427 U/L Amylase: 1,427 U/L	Myocardial perfusion scan	(I), t-PA given, presented 1 h after symptom onset
Tejada [23], 2008 (A,S)	56 years, man	Nausea and vomiting, diaphoresis	Inferior II, III, aVF, V5–V6	Negative troponin T: <0.01 ng/mL Lipase: 1,845 U/L Amylase: 1,178 U/L	Coronary angiography	(I), resolution of ST elevations on 20-month follow-up
Makaryus [24], 2008 (H,L)	59 years, man	Midsternal chest pressure	Inferior II, aVF, Q waves in III	Negative troponins: <0.3 ng/mL Lipase: 24 U/L Amylase: 3,648 U/L	Coronary angiography	(I), pancreatitis confirmed on imaging
Korantzopoulos [25], 2005	59 years, man	Epigastric pain, nausea and vomiting	Anterior V2–V6	Elevated troponin T: 0.73 ng/mL Amylase: 749 U/L	None	(D), hemorrhagic pancreatitis leading to respiratory arrest
Ro [26], 2004 (A,H)	43 years, Caucasian woman	Nausea and vomiting, epigastric pain radiating to b/l flanks	Precordial TWI, prolonged QT interval	Elevated peak troponin T: 0.25 ng/mL Lipase: 763 U/L	Echocardiogram, myocardial contrast echocardiography	(I), resolution of ECG and troponins on 3-week follow-up
Albrecht [27], 2003	64 years, man	Recurrent abdominal pain	Infero-antero-lateral I, II, III, aVF, V2–V6	Elevated troponin T: 1.87 Lipase: 261 U/L	Coronary angiography	(D), acute respiratory distress and lactic acidosis

(D), deceased; (I), discharged after clinical improvement. Reported risk factors key: A, alcohol use; C, coronary artery disease; D, diabetes; H, hypertension; L, hyperlipidemia; N, no specified cardiac risk factors; R, renal disease; S, smoking use. * Potassium levels reported within normal limits (range 3.61–4.68 mmol/L) during admission. † Clinical course, diagnostic testing, and patient outcome was not specified in this report and was omitted in the prevalence values for the respective categories. ‡ Amylase levels originally reported as Somogyi units; these values were converted to metric units.

Table 3. Reports of myocardial ischemic mimicry in acute pancreatitis (continued)

Report, year	Age, sex	Symptoms	ST elevation leads	Lab values	Cardiac evaluation	Outcomes
Yu [28], 2003 (H)	71 years, man	Left upper quadrant abdominal pain, nausea and vomiting, diaphoresis	New, evolving left bundle branch block	Negative serial troponin T Lipase: 2,469 U/L Amylase: 1,023 U/L	Coronary angiography	(I), complicated by superior mesenteric vein thrombosis and pancreatic pseudocyst development; left bundle branch persisted on serial ECGs during admission
Wagner [29], 2002	56 years, man	Epigastric pain, nausea, diaphoresis	Anteriolateral V2–V4 (2 mm), I and avL	Unspecified	None	(I), t-Pa given 4 h after symptom onset, resolution of ST elevation within hours
Khairy [30], 2001 (L,H,R,S)	64 years, Caucasian woman	Burning epigastric pain radiating to chest and back, nausea and vomiting	Anterior V2–V4, TWI AvL, V5–V6	Unspecified troponins Lipase 5,750 U/L	Coronary angiography	(I), persistent anterior ST elevations with diffuse less pronounced TWI
Cafri [31], 1995 (N)	54 years, man	Epigastric pain, recurrent vomiting	Inferior II, III, aVF	Amylase: 1,490 U/L	Echocardiogram	(I), given streptokinase, ST elevations resolved within 12 h
Patel [32], 1994 (S,T)	57 years, African American woman	Abdominal pain radiating to back, nausea and vomiting	Anteriolateral V3–V6, TWI V3–V4	Amylase: 104 U/L Lipase: 94 U/L	Coronary angiography, echocardiogram	(I), mottled pancreatic body and tail on RUQ U/S, resolution of ST elevations in V3–V6 by day 5 with deepening TWI and new inversions in II, III, aVF
Burge [33], 1993	79 years, woman	Severe retrosternal chest pain, nausea, diaphoresis	Complete left bundle branch block	Negative troponins, unspecified Amylase: >5,000 U/L	None	(I), cholelithiasis s/p laparoscopic cholecystectomy, ECG changes unchanged on discharge
Main [34], 1990 (A)	47 years, man	Retrosternal chest pain	Inferiolateral, leads unspecified	Amylase: 960 U/L	Autopsy	(D), anistreplase administered, died 15 h later, peritoneal cavity contained 3.5 L of blood and retroperitoneal hematoma with pseudocyst contents
Cohen [35], 1971 [†] (A)	41 years, African-American man	Epigastric pain, nausea and vomiting	Anteriolateral V2–V5, TWI I, II, III, aVF, V2–V6	Amylase: 463 U/L	Coronary angiography	(I), resolution of ST elevations, and precordial T waves, persistent TWI in limb leads
Spritzer [36], 1969 [†] (A)	47 years, Caucasian man	Inebriation, nausea and vomiting, anterior chest pain radiating to medial left arm to elbow	Inferior II, III, aVF	Amylase: 276 U/L	Coronary angiography	(I), resolution of ST elevations within 36 h
Fulton [37], 1963 [†] (H)	61 years, Caucasian man	Upper abdominal pain, nausea and vomiting	Anterior V2–V4 (2–3 mm) 24 h after admission on vasopressors	Amylase: 370 U/L	Autopsy	(D), severe necrotizing pancreatitis
Shamma'a [38], 1962 [†]	43 years, man	Epigastric pain with bilious vomiting	Inferior III, aVF, TWI: L II, III, aVF	Amylase: 2,279 U/L	None	(I), resolution of amylase and leukocytosis within 1 week
Bauerlein [5], 1954 [†]	51 years, Caucasian woman	Severe abdominal pain, nausea and vomiting, rebound tenderness	Anterior ST elevation I-II, V2–V6 5 mm V3, ST depression V1, aVR	Amylase: 140 U/L	Autopsy	(D), severe pancreatic necrosis

(D), deceased; (I), discharged after clinical improvement. Reported risk factors key: A, alcohol use; C, coronary artery disease; D, diabetes; H, hypertension; L, hyperlipidemia; N, no specified cardiac risk factors; R, renal disease; S, smoking use. *Potassium levels reported within normal limits (range 3.61–4.68 mmol/L) during admission. [†]Clinical course, diagnostic testing, and patient outcome was not specified in this report and was omitted in the prevalence values for the respective categories. [‡]Amylase levels originally reported as Somogyi units; these values were converted to metric units.