

## CASE REPORT

## Acute Pancreatitis with Electrocardiographic Changes Mimicking Acute Coronary Syndrome: A Diagnostic Conundrum.

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### ABSTRAK

*Elektrokardiogram yang tidak normal boleh dikaitkan dengan radang pancreas akut. Tetapi malangnya punca-punca penyebab penyakit ini masih belum diketahui. Terdapat banyak laporan kes terdahulu menyatakan bahawa perubahan corak yang tidak normal pada ST dan T pada elektrokardiogram berkaitan dengan pankreas akut tetapi jarang sekali berlaku peningkatan enzim jantung. Kami membincangkan kes seorang wanita 70 tahun dengan masalah ini. Pesakit kami pada mulanya di rawat sebagai keradangan pancreas tetapi terdapat perubahan elektrokardiogram dan peningkatan enzim jantung. Selepas rawatan untuk pankreas akut diberikan, didapati elektrokardiogram pesakit kembali kepada normal. Ini membuktikan penyakit ini boleh menyebabkan perubahan biokimia dan elektrik yang menyerupai penyakit jantung. Oleh itu, pakar perubatan kecemasan perlu mengambil kira elektrokardiogram yang tidak normal adalah disebabkan oleh radang pancreas akut.*

*Kata kunci: diagnosis, keradangan pankreas, sindrom koronari akut*

### ABSTRACT

Electrocardiographic abnormalities can be associated with acute pancreatitis. However, data regarding the actual causative factor still remains elusive. Many previous cases were reported on non-specific ST and T wave abnormalities concurrent with acute pancreatitis but rarely with an increasing trend of cardiac markers. We describe the case of a 70-year-old female who presented with one such conundrum. Our patient had typical presentation of acute pancreatitis but had dynamic ECG changes with markedly increased cardiac markers. Subsequently

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after initiation of treatment for acute pancreatitis and observation for the course of several days, the ECG returned to the baseline as pre admission. This substantiates the fact that acute pancreatitis can mimic both biochemical and electrical manifestation of an acute coronary syndrome. Thus, Emergency Physicians should consider acute pancreatitis as a possible diagnosis in patients who present with abnormal electrocardiograms.

Keywords: acute pancreatitis, acute coronary syndrome, diagnosis

## INTRODUCTION

Acute pancreatitis is easily distinguishable from acute coronary syndrome and it rarely presents as a diagnostic dilemma. However, the problem arises when a patient is diagnosed with acute pancreatitis but also has overlapping features of coronary artery disease in the form of new onset and evolving electrocardiographic abnormalities (Buch et al. 1980). This is further complicated by raised cardiac markers without typical symptoms of chest pain, positive clinical features of cardiac disorders and hemodynamic instabilities (Khairy & Marsolais 2001).

A variety of ECG changes have been identified in association with acute pancreatitis such as bradycardia, arrhythmias, T-wave changes, ST-segment elevation (Khan et al. 2014) or depression and intraventricular conduction disturbances, but is rarely associated with corresponding cardiac enzyme derangements (Antonio et al. 2017).

In the case discussed, evolving ECG changes is not only prominent in contiguous leads but also presents with uprising cardiac markers, posing a difficult decision on whether treatment directed towards coronary artery disease should be initiated.

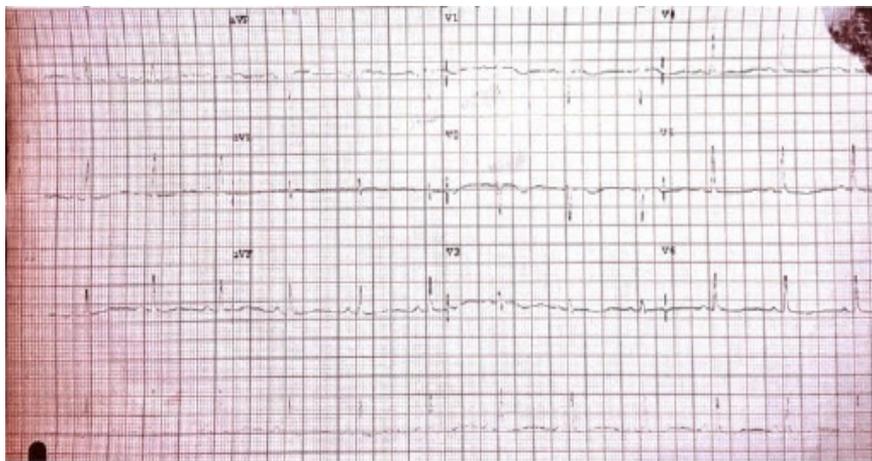


Figure 1a : ECG on arrival demonstrating repolarization changes

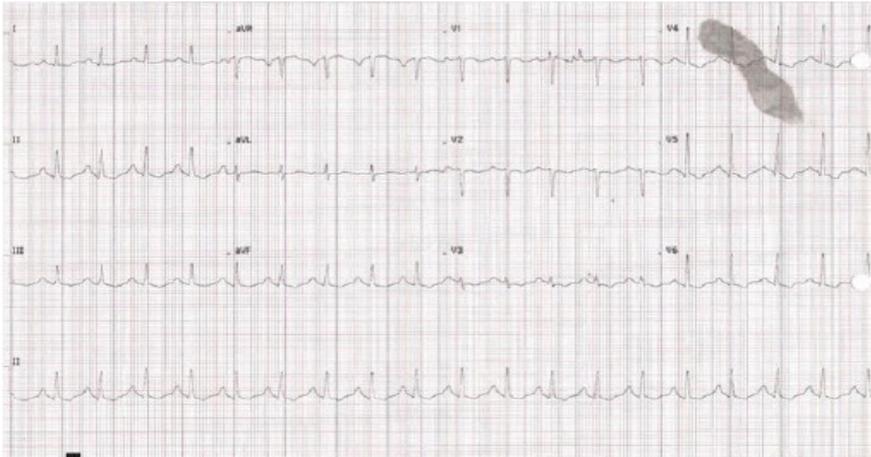


Figure 1b: repeat x 1: ECG demonstrating evolving changes over the T waves at the limb and precordial leads

### CASE REPORT

A 70-year-old female presented to the Emergency Department after experiencing non-exertional epigastric pain with progressive radiation to the retrosternal region. The pain was sharp in nature and the onset of the symptoms started 6 hours prior to presentation and progressively worsened. This was associated with nausea and one episode of vomiting,

diaphoresis and palpitation but no reports of dyspnea or pre syncopal episodes. No relieving factors were reported. No change of pain intensity with bending forward. The patient has underlying Type 2 Diabetes Mellitus and hypertension. She was previously well with no complaint of chest pain, decrease effort tolerance, orthopnea or paroxysmal nocturnal dyspnea. Clinical examination revealed normal heart sounds with no added sounds,



Figure 1c: repeat x 2: ECG demonstrating evolving changes over the T waves at the limb and precordial leads



Figure 1d: ECG upon discharge

soft but severely tender abdomen over the epigastric region, with no signs of peritonitis. Pain had no radiation to the other abdominal quadrants. Hemodynamically stable but documented temperature at 39°C.

An ECG was done on arrival and revealed: Sinus rhythm, HR: 75/min, Flat T wave in I, II, III, aVF, aVL, biphasic T waves in V1–V3 (Figure 1a). A repeated ECG >1 hour later revealed evolving changes : sinus tachycardia (HR: 110-114/min), with evolving widespread T wave inversion over the precordial leads from V2 – V6 and limb leads II, III and aVF (Figure 1b and 1c). Serum biochemistry demonstrated a serum amylase of 763. Cardiac biomarkers, were within the normal range and subsequently raised 6-fold from the initial figure (Table 1). Infective markers were also raised (CRP). A bedside ECHO was performed which revealed no regional wall motion abnormalities, and normal chamber sizes of the heart. An ultrasound of the abdomen was performed but the findings were inconclusive.

The case was referred to both the cardiologist and the surgical team. Due to the atypical nature of presentation, the patient was placed under observation with serial ECGs and cardiac markers. She was initially loaded with dual anti-platelet therapy in the Emergency Department but subsequently it was discontinued as the cause of the ECG changes and raised cardiac markers were attributed to the underlying acute pancreatitis. The patient was admitted under the surgical team where she was started on adequate hydration and pain relieve. During her stay in the surgical unit, her condition improved and the electrocardiographic changes (Figure 1d) recovered to the baseline as per on arrival. She was discharged with a prophylactic dose of aspirin and scheduled for a cardiology clinic review on a later date.

## DISCUSSION

This case substantiates the fact that acute pancreatitis can mimic

Table 1: Progression of biochemical markers

	Day 1	Day 2	Day 3	Reference values
CK	114	77	47	(29 - 168) u/L
Troponin T	<10.0	65.2	42.6	<15.6 pg/mL
Amylase	763	672		(25 – 125) u/L
LDH	911			(125 – 220) u/L
Random urine amylase		1436		< 460 u/L
Bilirubin	19.2			(3.4 – 20.5) umol/L
ALT	53			(0 – 55) u/L
ALP	97			(40 – 150) u/L
AST	833			<45 u/L
WBC	10.8			(4.1 – 11.4) x 10 <sup>9</sup> /L
CRP		12.42		<0.5 mg/dL
Sodium	131	136	137	(36 – 145) mmol/L
Potassium	3.0	3.1	3.4	(3.5 – 5.1) mmol/L

both biochemical and electrical manifestation of an acute coronary syndrome. There are reports of cases documented in the past whereby patients with acute pancreatitis present with nonspecific repolarization abnormalities (Patel et al. 1994). However, evolving ST – T wave abnormalities coupled with progressive increase in cardiac markers are rare and indicates a possibility of either a concurrent cardiac disorder in acute pancreatitis or features manifested secondary to cellular level disturbances. The exact cause of these changes remain largely unknown even though its co relations has been established in many different case reports (Shamma'a et al. 1962).

Drummond described the first case of acute pancreatitis associated with ECG changes suggestive of myocardial ischemia in 1934 (Yegneswaran et al. 2011). Subsequently, numerous case series and reports have documented nonspecific ST and T wave changes in patients with acute pancreatitis (Munir & Shamma'a 1962). There are

many different schools of thought on conditions that precipitate myocardial ischemia in inflammatory conditions such as myocarditis, bacterial shock, cholecystitis, pneumonitis, and as mentioned acute pancreatitis. These include autonomic imbalance with vagal predominance, toxic effects of the pancreatic proteolytic enzymes on the myocardium, metabolic and electrolyte abnormalities, coronary artery spasm, systemic inflammatory response-induced cardiac damage, pro thrombotic derangements and hemodynamic instability.

Electrocardiographic changes that are usually seen ranges from T wave inversion, ST segment depression and in rare cases even ST segment elevation (Rubio-Tapia et al. 2005). A subject of controversy remains if all these changes can manifest together with elevated cardiac markers in the absence of a structural and mechanical dysfunction of the heart. Our case which was similar to case reports discussed previously lacks definitive proof of investigation, i.e.:

Percutaneous Coronary Intervention.

Several different possibilities have been proposed in the past pertaining to ECG changes in acute pancreatitis. One such hypotheses are the possibility of electrolyte (Yaylaci et al. 2015) and metabolic abnormalities such as hyponatremia, hypokalemia, hypocalcemia, hypomagnesemia, hypophosphatemia and even hypoglycaemia. However, our patient did not present with any such marked abnormalities which rules out the possibility stated above.

Another largely debated notion is that pancreatic proteolytic enzymes like Trypsin 1 may result in direct myocyte cellular damage resulting in changes in cellular permeability, and possible tissue necrosis and electrical changes. This was demonstrated by Aaron et al. (1953) who noted ECG changes after intravenous injection of proteolytic enzymes (Khairy & Marsolais et al. 2001) that subsequently resolved after a duration of 2 weeks. Proteolytic enzymes, lipases or phospholipolytic enzymes are argued to cause electrical changes in the form of sublethal damage (Patel et al. 2015) rather than myocardial damage, thus all ECG changes should be transient and normalises to baseline and with no histological evidence that demonstrates myocardial necrosis.

Many cases also reported alternate mechanisms involving coronary vasospasm (Khan et al. 2014). This reaction subsequently exacerbates the pre-existing coronary artery disease resulting from increased platelet adhesiveness or pancreatic enzyme induced coagulopathy. A study by

Phadke et al. (2013) reported a case of a patient with typical presentation of acute pancreatitis concurrent with features of myocardial infarction. Coronary angiogram later revealed a thrombus in the left anterior descending artery. Another causative factor is vasospasm induced by the pancreatitis, resulting from endothelial dysfunction of the coronary arteries. This could not be ruled out in our case as no invasive diagnostic procedures were carried out to identify coronary artery obstruction.

Cardiobiliary reflex has also been postulated as a possible causative factor. This is due to cardiac damage by auxillary coronary blood flow or effect to the myocardium itself. Another phenomena which is largely debated is the vagal mediated reflex. This reflex has also been cited as the cause of T wave abnormalities in acute cholecystitis, pancreatitis, gastrointestinal haemorrhage and intracranial bleeds.

## CONCLUSION

Despite the findings of evolving ECG changes and elevated cardiac markers, further complementary non-invasive and invasive studies are required to establish a more definitive diagnosis. The case report clearly demonstrated that acute pancreatitis can present with electrocardiographic and biochemical changes that mimic coronary artery disease in the absence of an underlying cardiac pathology or a mechanical or structural dysfunction. The etiology of these cardiac - related changes are still largely unknown and remains

speculative. Further studies and case series are warranted in order to establish the postulated pathophysiological probabilities as stated above.

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