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Kasuistika | Case report

Unusual electrocardiographic changes during acute pancreatitis

Dante Antonelli, Ehud Rozner, Yoav Turgeman

Department of Cardiology, Ha'Emek Medical Center, 18100 Afula, Israel

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SOUHRN

Šestačtyřicetiletý muž pociťoval mírnou bolest v horní části břicha, jež se šířila do oblasti zad, a nevolnost; po týdnu si začal stěžovat i na diskomfort na hrudníku, který se šířil do oblasti krku; proto se dostavil na oddělení urgentního příjmu. Dvanáctisvodový elektrokardiogram prokázal velké hrotnaté vlny T ve svodech II-III-AVF a obrovské inverze vln T ve svodech aVL a V_1 až k V_6; o půl hodiny později diskomfort na hrudníku ustoupil a elektrokardiografické změny vymizely. Biochemické vyšetření séra prokázalo zvýšené hodnoty pankreatických enzymů, zatímco hodnoty elektrolytů, kreatinkinázy a troponinu T v séru zůstaly normální. Koronarografie prokázala nepostižené koronární tepny. Pacient byl následně léčen konzervativně.
I když elektrokardiografické abnormality již byly u nemocných s akutní pankreatitidou zaznamenány, široké, vysoké a hrotnaté vlny T, které jsme objevili na elektrokardiogramu našeho pacienta, dosud popsány nebyly.

Keywords:
Cardiobiliary reflex
Myocardial infarction
Myocardial ischemia
Pancreatitis

ABSTRACT

A 46-year-old man suffered from mild upper abdominal pain radiating to the back and nausea; after a week he began to complain also of chest discomfort radiating to the neck and presented to the emergency room. A 12-lead electrocardiogram showed large peaked T waves in leads II-III-AVF and giant T waves inversion in AVL, V_1 through V_6; half an hour later the chest discomfort and the electrocardiographic changes resolved. Serum biochemistry results showed elevated serum pancreatic enzymes; electrolytes, creatinine kinase and troponin T serum values remained normal. Coronary angiography showed normal coronary arteries. The patient was conservatively managed.
Electrocardiographic abnormalities were reported in patients with acute pancreatitis but broad, tall and peaked T waves, as found in our patient electrocardiogram, have not been yet reported.

Address: Dante Antonelli, MD, Department of Cardiology, Ha'Emek Medical Center, Afula 18101, Israel, e-mail: antonelli_dante@hotmail.com
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Introduction

Acute pancreatitis presents with abdominal pain and elevated pancreatic enzymes in serum. It is associated with variable involvement not only of pancreatic tissue but also of other organs. Alcohol abuse and gallstones are its two most common etiological factors [1].

Case report

A 46-year-old man suffered from mild upper abdominal pain radiating to the back and nausea. After a week he began to complain also of chest discomfort radiating to the neck and presented to the emergency room. He had no known cardiac disease or cardiovascular risk factors. The patient had no previous attacks of pancreatitis or cholecystitis and he was hospitalized for the first complaints; he had no history of immoderate alcohol consumption.

On physical examination the patient was afebrile, had a Body Mass Index of 30.1, blood pressure 141/90 mmHg, pulse 72 beats/min, oxygen saturation 94% on air, cardiac and pulmonary examination were unremarkable; abdominal palpation disclosed mild diffuse tenderness, worse in the epigastrium; bowel sounds were normal.

A 12-lead electrocardiogram (ECG), taken at his arrival to the emergency room, showed sinus rhythm 60 beats/min, large peaked T waves in leads II-III-AVF and giant T waves inversion in AVL, V_1 through V_6 (Fig. 1A); about half an hour later the chest discomfort resolved and a repeat ECG revealed resolution of the ischemic changes (Fig. 1B).

Serum biochemistry results are showed in Table 1. Transthoracic echocardiography, performed when the ECG changes resolved, found normal left ventricular function with no wall motion abnormalities and no pericardial effusion.

An upper abdomen ultrasound examination revealed multiple small gallstones in the gallbladder.

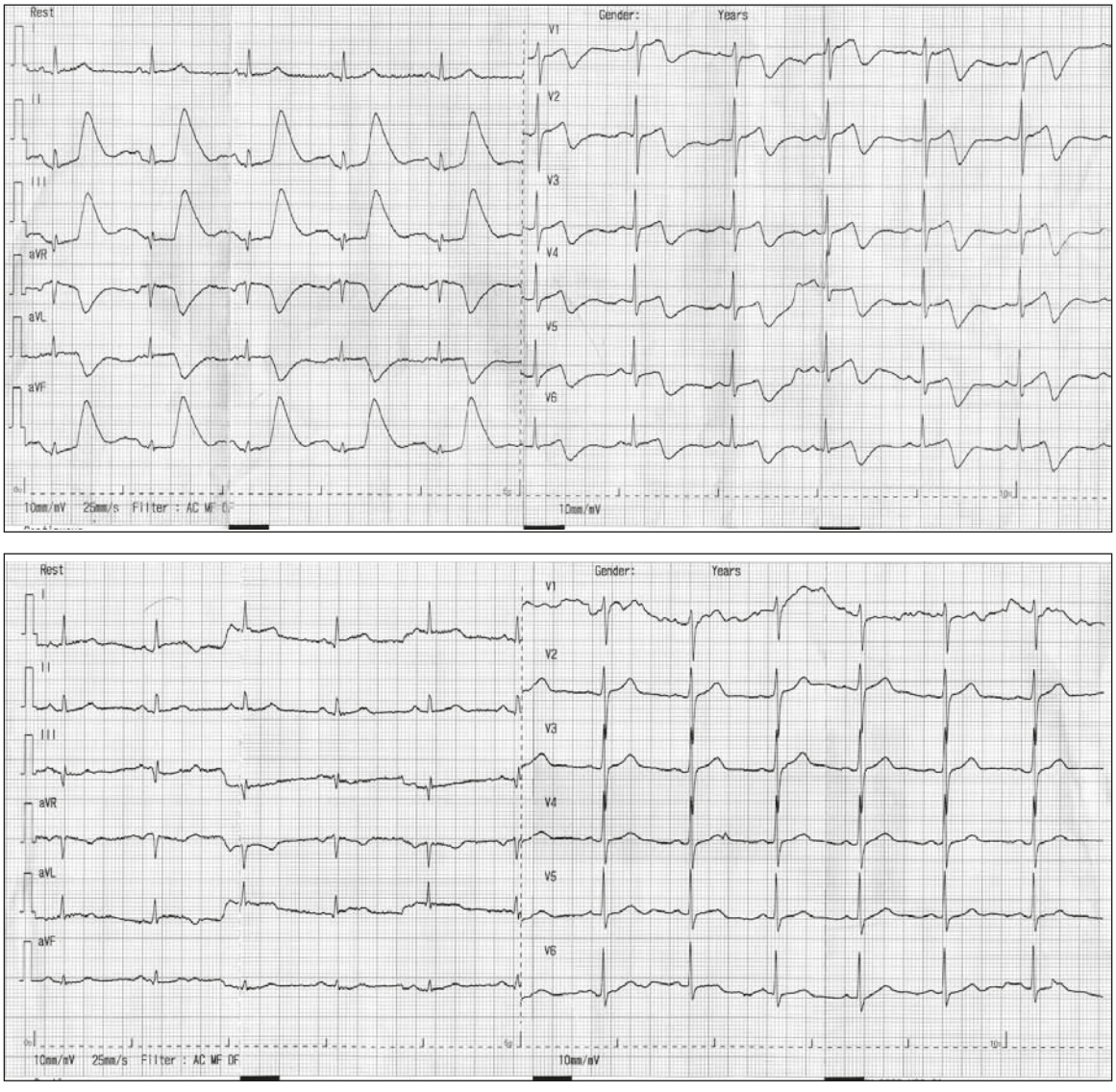

Fig. 1 – (A) Electrocardiogram on admission showing sinus rhythm 60 beats/min, large peaked T waves in leads II-III--AVF and giant T waves inversion in AVL, V_1 through V_6. (B) Electrocardiogram obtained half an hour after the initial one revealing resolution of the ischemic changes.

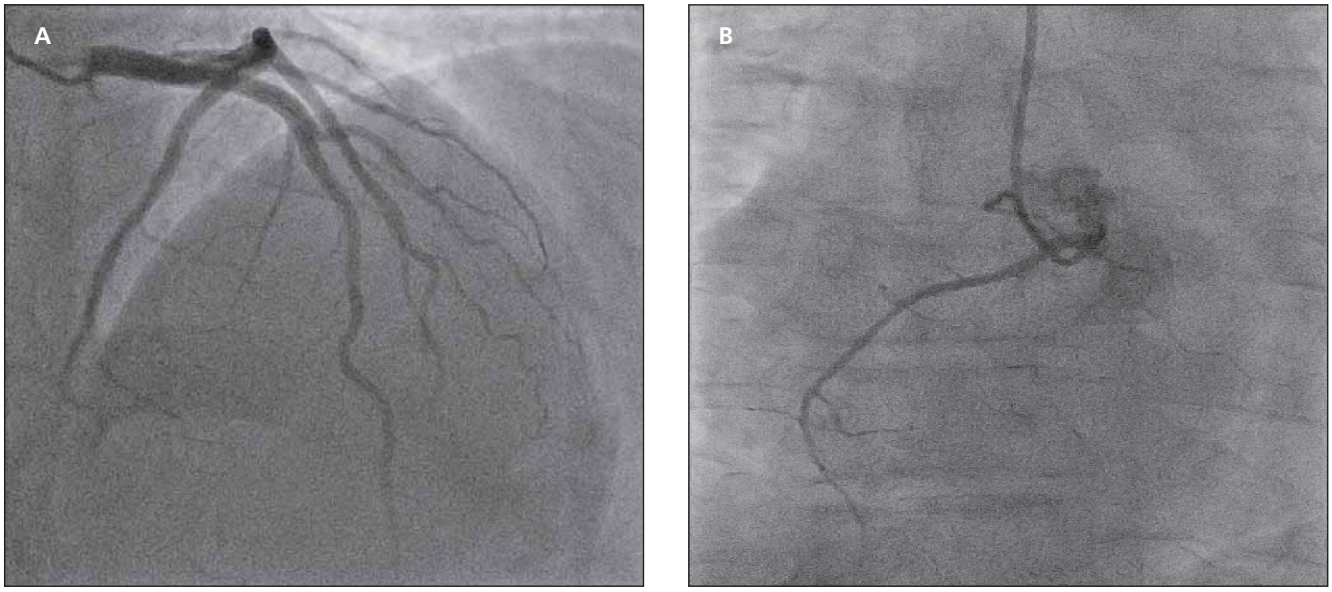

Fig. 2 – (A) Left coronary angiogram: normal left coronary artery. (B) Right coronary angiogram: non dominant right coronary artery.

Coronary angiography performed on the following day showed normal coronary arteries (Figs. 2A and 2B).

The patient was conservatively managed. He no further complained of chest discomfort; the abdominal symptoms improved and his ECG remained normal for the course of hospital stay. During outpatients clinic follow-up gradually his chemistries returned to normal levels. The patient underwent laparoscopic cholecystectomy 6 months after hospital discharge. One-year follow-up was uneventful regarding cardiac symptoms and pancreatitis.

Discussion

The abdominal pain, pathological values of serum pancreatic enzymes and ultrasound evidence of gallstones are suggestive of acute pancreatitis. Chest pain and tall peaked T waves on ECG are suggestive of myocardial ischemia; however, in our patient cardiac catheterization excluded presence of coronary artery disease.

Electrocardiographic abnormalities were found in about 50% of patients with acute pancreatitis [1,2] and included arrhythmias, ST-T waves changes and intraventricular conduction disturbances. Non-specific changes of

Table 1 – Serum biochemistry

Serum biochemistry	Normal values	16. 9. 2014 0:14	17. 9. 2014 6:50	18. 9. 2014 10:20	19. 9. 2014 9:00
Glucose	70–100 mg/dL	**114**	**169**	92	–
Bilirubin total	0.3–1.2 mg/dL	**4.24**	**2.25**	**1.31**	–
Bilirubin direct	0–0.3 mg/dL	**3.03**	**0.94**	**0.42**	–
Sodium	135–145 mmol/L	135	137	138	139
Potassium	3.5–5.1 mmol/L	3.61	3.66	4.39	4.68
Chloride	98–106 mmol/L	102	101	103	102
Calcium	8.5–10.5 mg/dL	–	9.53	9.39	9.36
GOT (AST)	0–35 U/L	**154**	**73**	**66**	**53**
GPT (ALT)	0–45 U/L	**346**	**272**	**232**	**183**
Amylase	28–100 U/L	**2311**	**1306**	**387**	–
Lipase	21–67 U/L	**4192**	**1289**	–	–
Alkaline phosphatase	30–120 U/L	**273**	**341**	**336**	**303**
Creatinine kinase (CPK)	20–200 U/L	139	–	–	–
Troponin T	<14 ng/L	<14	<14	–	–

GOT – aspartate aminotransferase; GPT – glutamic transpeptidase.

repolarization were the most frequent; ST segment elevation was rarely reported in the absence of coronary artery disease [3–5], although cases of acute pancreatitis complicated by acute myocardial infarction were reported [6,7]. T waves abnormalities found during acute pancreatitis included decreased voltage and inversion of T waves [2]. Broad, tall and peaked T waves, as found in our patient ECG, have been not yet reported.

Many theories have been advanced to explain the pathogenesis of ECG changes during acute pancreatitis: electrolytes disturbances, direct injury to myocyte membrane due to cardiotoxic effect of proteolytic enzymes, exacerbation of underline cardiac disease, coagulopathy, cardiobiliary reflex, coronary artery spasm [1].

In our patient no electrolytes abnormalities or coagulopathies were present and our patient had normal coronary arteries and no previous history of chest pain.

Pancreatic enzymes have been suggested to cause a direct myocardial damage [8], however, no differences were found in enzymes serum values in patients with normal or abnormal ECG [2].

It has been suggested that a vagal mediated reflex trough intermediate neurons connecting the nervous rami directed to the heart and biliary tree takes part in this pathology [9]. Although the innervation to the heart and gallbladder arises from different level of the spinal cord this cardiobiliary reflex is responsible for the T waves changes in acute cholecystitis and it has been associated also in pancreatitis, gastrointestinal and intracranial bleeding [10].

Coronary vasospasm is a possible explanation of transient ST segment elevation and normal coronary artery during acute pancreatitis [5]. In our patient ST segment elevation was not documented, but also peaking and/or increase in amplitude of the T wave occur during an attack of coronary spasm [11]; the fact that ECG changes resolved in 30 minutes made coronary vasospasm the most plausible mechanism.

Conclusions

This case reminds us to consider acute pancreatitis in the differential diagnosis in patients with chest pain and ECG changes mimicking acute myocardial ischemia or infarction.

Conflict of interest
None declared.

Funding body
None.

Ethical statement
Authors state that the research was conducted according to ethical standards.

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