

T-wave depletion and bradycardia possibly secondary to acute pancreatitis: Review of the literature

Akut pankreatite bağı EKG'de T dalgası çökmesi ve bradikardi: Literatürün gözden geçirilmesi

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Acute pancreatitis has frequently been reported to be associated with transient electrocardiography changes mimicking myocardial infarction despite normal epicardial coronary arteries. Although the origin of these findings is poorly understood, suggested mechanisms have included electrolyte abnormalities, a vagally mediated reflex, coronary vasospasm, and myonecrosis because of the release of pancreatic proteolytic enzymes. We report a case of acute pancreatitis with new-onset electrocardiography changes and bradycardia despite no evidence of coronary artery disease. After resolution of inflammation in the pancreas, T-wave depletions in V1-V6 derivations in electrocardiography disappeared and the rhythm was sinus with 70/min. 201-Tl myocardial perfusion scintigraphy revealed no evidence of significant coronary artery disease.

Key words: Acute pancreatitis, electrocardiographic changes

INTRODUCTION

Acute pancreatitis has frequently been reported to be associated with transient electrocardiographic changes mimicking myocardial infarction despite normal epicardial coronary arteries (1, 2). Herein, we report T-wave inversion together with bradycardia secondary to acute pancreatitis in a patient without coronary artery disease (CAD).

CASE REPORT

A 47-year-old female patient was admitted to the Emergency Department of our hospital with epigastric pain and vomiting, which had started almost 12 hours before. Palpation of the abdomen revealed epigastric tenderness, whereas bowel sounds were hypoactive during auscultation. The rest of the physical examination was normal. She

Akut pankreatite eşlik eden geçici elektrokardiyografik değişiklikler daha önce literatürde pek çok kez bildirilmiştir. Altta yatan neden tam olarak anlaşılmasa da, elektrolit anormallikleri, vagal refleks, koroner vazospazm, ve pankreatik proteolitik enzimlerine bağlı miyonekroz üzerinde durulan mekanizmalardır. Biz burada akut pankreatitli bir hastada koroner arter hastalığı olmaksızın gelişen elektrokardiyografik değişiklikler ve bradikardiyi sunuyoruz. Tedavi sonrası pankreasta inflamasyonun gerilemesi ile elektrokardiyografide V1-V6 derivasyonlarındaki T dalga negatifliği düzeldi ve kalp hızı 70/dk, sinus ritmi oldu. Hastanın çekilen 201-Tl miyokard perfüzyon sintigrafisinde koroner arter hastalığı bulgusuna rastlanmadı.

Anahtar kelimeler: Akut pankreatit, elektrokardiyografik değişiklikler

was not using any drugs. She denied alcohol use and smoking. Her vital signs were as follows: blood pressure 135/86 mmHg, heart rate 42 beats/min and temperature 36°C. Her laboratory findings were: hematocrit 42.4%, white blood cells 8200/ml, glucose 98 mg/dl (reference range: 70-105 mg/dl), total bilirubin 3.94 mg/dl (reference range: 0.2-1.0 mg/dl), direct bilirubin 3.21 mg/dl (reference range: 0.1-0.5 mg/dl), aspartate aminotransferase 120 U/L (AST, reference range: 10-40 U/L), alanine aminotransferase 428 U/L (ALT, reference range: 10-35 U/L), calcium 9.4 mg/dl (reference range: 8.4-10.2 mg/dl), sodium 138 mmol/L, potassium 4.4 mmol/L, magnesium 2.2 mg/dl, chloride 102 mmol/L, amylase 3660 U/L (reference range: 0-125 U/L), lipase 2227 U/L (reference range: 3-60

U/L), and C-reactive protein 7.18 mg/L (CRP, reference range: 0-5 mg/L). Blood gas analysis showed: pH 7.39, pO₂ 89 mmHg, pCO₂ 40 mmHg, and HCO₃ concentration 24 mmol/L. Serum osmolarity was measured as 283 mOsm/kg. Serum lipid profile, renal function, thyroid function, complete blood count, calcium, and urinalysis were all normal.

The Ranson's score in this patient was zero on admission. Ultrasonography showed gallstones, and upper abdominal computed tomography (CT) revealed moderate pancreatic edema, compatible with acute pancreatic inflammation. Serial electrocardiograms (ECGs) demonstrated new-onset sinus bradycardia and negative T-wave in V₁-V₆ derivations (Figure 1) without any chest pain. The ECG six months before had been normal. Serum troponin T and CKMB levels were in normal range. Atropine was administered for bradycardia. Echocardiography revealed normal systolic functions; there was no evidence of significant CAD. 201-Tl myocardial perfusion scintigraphy revealed no evidence of significant CAD. She progressively improved after conservative and antibiotic therapy and her serum amylase and lipase levels returned to normal range five days after admission. The negative T-waves in V₁-V₆ derivations in ECG disappeared and her heart rate was 78 beats/min (Figure 2). A cholecystectomy was performed two weeks later.

DISCUSSION

Transient and permanent ECG changes in patients with conditions such as cholecystitis, pancreatitis, and pneumonia have been reported, mostly consisting of T-wave inversions, ST segment depression, and ST segment elevations (1, 2). Several attempts have been made to elucidate the mechanism for such ECG changes. Even though the nerve supply between the pancreas and the heart arises from different spinal cord lev-

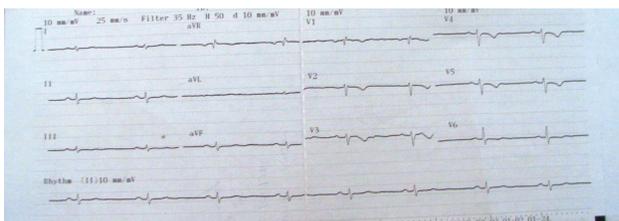


Figure 1. Sinus bradycardia (60 beats/min) and negative T-wave in V1-V6 derivations in ECG on the second day of hospitalization.

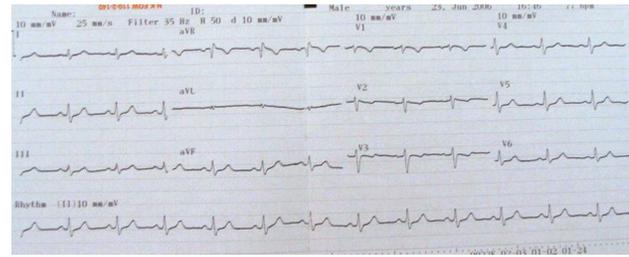


Figure 2. ECG after pancreatitis treatment (78 beats/min).

els, there are intermediate neurons that connect these branches and cause a common vagal response with subsequent changes in coronary hemodynamics. This so-called 'cardiobiliary reflex' has been inhibited by vagotomy or atropine administration (3, 4). The bradycardia in our patient might be a result of this 'reflex', as atropine administration was useful. In addition, the accelerated heart rate in the re-examination of our patient supports this theory.

Persistence of ECG changes along with wall abnormalities has been reported previously by Patel *et al.* (5), and these findings have been attributed to hypokalemia, hypomagnesemia, or both, but even after the correction of such imbalances, the ST segment pattern of ischemia did not change. In our case, there was no electrolyte imbalance during the hospitalization period to explain the ECG changes.

Electrocardiography changes mimicking acute myocardial infarction (AMI) in patients with acute pancreatitis have been reported in the literature (1, 5-8). In these cases, cardiac-specific enzymes remained within normal levels, suggesting that ST-segment elevation was not due to AMI. In one case, there were concomitant wall motion abnormalities in echocardiography examination (5). In some patients, the coronary angiogram was normal, thus totally excluding the coexistence of CAD (5-8). Undoubtedly, in such cases, there is a considerable diagnostic and therapeutic challenge for the clinician. In patients presenting with acute pancreatitis and ECG changes suggesting AMI, measurement of serum troponin T concentrations can aid in differentiating ECG changes driven by acute pancreatitis from those of true myocardial ischemia or infarction. In our case, we performed ECGs and cardiac-specific enzymes every 12 hours to exclude AMI. Echocardiography and 201-Tl myocardial perfusion scintigraphy were performed, and both revealed no evidence of CAD.

On the other hand, AMI in the course of acute

pancreatitis has been documented in three cases (9, 10). In one case, a 64-year-old man proved to have normal coronary arteries (10). The possibility of CAD in our case is relatively low since she was healthy without any cardiovascular disease or risk factor, and the 201-Tl myocardial perfusion scintigraphy revealed no evidence of significant obstructive CAD.

Various hypotheses have been formulated for the underlying mechanism of electrocardiography abnormalities and/or myocardial damage in acute pancreatitis (7-10). These include toxic effects of the pancreatic proteolytic enzymes on the myocardium, autonomic imbalance with vagal predominance, coronary artery spasm, metabolic and electrolyte abnormalities, prothrombotic derangements, hemodynamic instability, and systemic inflammatory response-induced cardiac

damage. The ECG changes and heart rate of our patient had normalized in the re-examination, supporting the effect of the autonomic imbalance with vagal predominance. The pericardial effusion detected in echocardiography supports the effect of the pancreatic proteolytic enzymes, hemodynamic instability, and systemic inflammatory response, but the exact cause of the ECG changes in our patient remained uncertain.

Several theories exist to explain the genesis of electrocardiography changes and/or myocardial damage in the presence of acute pancreatitis; the exact mechanism of such findings remains to be elucidated. We wish to alert physicians that acute pancreatitis can cause T-wave inversion and bradycardia even in patients with normal coronary arteries. However, these patients should be followed to exclude CAD.

REFERENCES

1. Cohen HM, Rotsztain A, Bowen JP, et al. Electrocardiographic changes in acute pancreatitis resembling acute myocardial infarction. *Am Heart J* 1971; 82: 672-7 Abstract.
2. Krasna MJ, Flancbaum L. Electrocardiographic changes in cardiac patients with acute gallbladder disease. *Am Surg* 1986; 52: 541-3.
3. Morrison LM. Role of gastrointestinal tract in production of cardiac symptoms. *JAMA* 1940; 114: 217-23 Abstract.
4. Kaufman JM, Lubera R. Preoperative use of atropine and electrocardiographic changes. *JAMA* 1967; 200: 198-200 Abstract.
5. Patel J, Mohaved A, Reeves W. Electrocardiographic and segmental wall motion abnormalities in pancreatitis mimicking myocardial infarction. *Clin Cardiol* 1994; 17: 505-9.
6. Cafri C, Basok A, Katz A, et al. Thrombolytic therapy in acute pancreatitis presenting as acute myocardial infarction. *Int J Cardiol* 1995; 49: 279-81.
7. Hung SC, Chiang CE, Chen JD, et al. Pseudo-myocardial infarction. *Circulation* 2000; 101: 2989-90.
8. Khairy P, Marsolais P. Pancreatitis with electrocardiographic changes mimicking acute myocardial infarction. *Can J Cardiol* 2001; 15: 522-6.
9. Wagner AM, Santalo M. A correct decision? *Lancet* 2002; 359: 157.
10. Albrecht CA, Laws FA. ST segment elevation pattern of acute myocardial infarction induced by acute pancreatitis. *Cardiol Rev* 2003; 11: 147-51.