Saudi J Kidney Dis Transpl 2011;22(5):

Saudi Journal
of Kidney Diseases
and Transplantation

Case Report

ST Segment Elevation in a Patient Presenting with Renal Colic Pain

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ABSTRACT. ST segment elevation of more than 1 mm from the baseline is an electrocardiographic presentation of acute myocardial injury. This phenomenon is not confined only to ischemic injury, and various other etiological factors have been described in relation to ST segment elevation. We report the case of a young man who presented in the emergency department complaining of loin pain. He was subsequently found to have transient ST segment elevation, but a thorough evaluation showed no evidence of coronary artery disease. ST segment elevation is one of the best tools for diagnosing acute myocardial infarction, of which reperfusion therapy is the treatment of choice thus making it important to make the diagnosis as quickly as possible. It is equally important, however, not to misdiagnose acute myocardial infarction, as there are other causes of ST segment elevation. Therefore, we report this unique case of renal pain due to stone causing ST segment elevation.

Introduction

ST segment elevation is one of the tools used in conjunction with cardiac markers to diagnose acute myocardial infarction (AMI). ST segment elevation may result from an occlusive thrombus within the coronary artery, but other factors have been reported to produce this phenomenon on ECG tracings, including cardiac and non-cardiac causes. In case of myocardial infarction in an acute setting, early reperfusion or percutaneous angiography is crucial, and quick intervention is the most important factor in the quality of care. Hence, it is vitally important to recognize that myocardial infarction is not the only cause of ST segment elevation. Although there are numerous reports in the literature describing non-ischemic causes of ST segment elevation, to the best of our knowledge, this case due to renal pain secondary to renal stone is the first to be reported.[1]

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Case Report

A 34-year-old male with a history of renal stones presented in the emergency department of the King Khalid University Hospital, Riyadh,

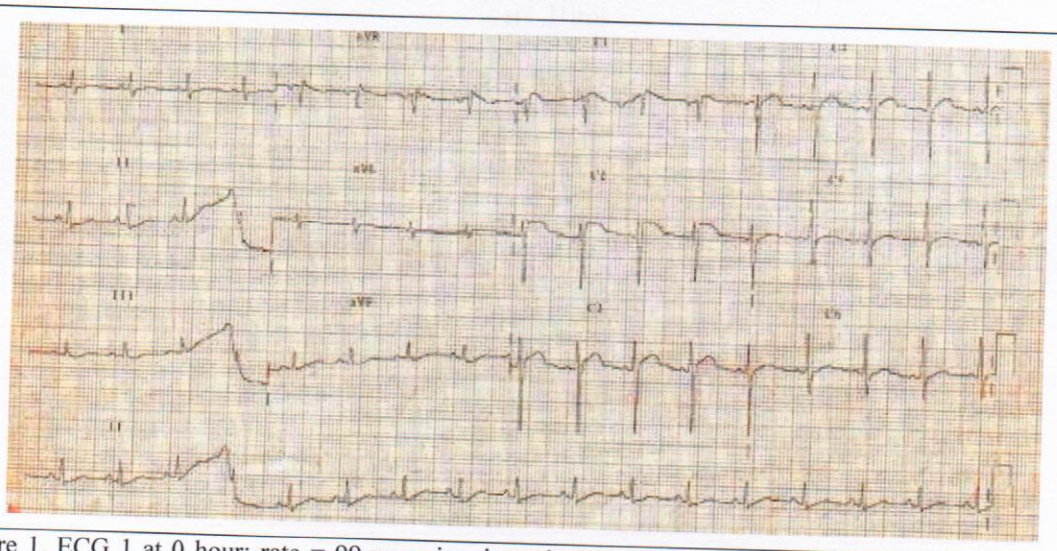

Figure 1. ECG 1 at 0 hour: rate = 99 per min, sinus rhythm, dynamic ST segment elevation in the pre-cordial leads V1–V3.

Saudi Arabia, because of severe left loin pain that was radiating to the left testis, and mild nausea that had begun two hours before presentation. He had no associated dyspnea, chest pain, or diaphoresis. There was no history of smoking, alcohol abuse, or use of illicit drugs, including cocaine, or family history of heart disease or any previous hospitalization for cardiac or any other problem. Two years earlier, he had experienced renal pain, which was similar in character to the current presentation and was relieved after pain management and spontaneous excretion of a stone.

On physical examination, the patient was alert and conscious. He had a pulse rate of 102/min, normal blood pressure of 125/85 mmHg, respiratory rate of 20 per min, temperature of 37.8°C, and oxygen saturation on pulse oximetry of 98% while breathing room air. No jugular venous distension was evident, and cardiovascular examination revealed normal audible S1 and S2 with no murmurs or pericardial rub. Respiratory examination revealed bilateral vesicular breathing with no adventitious sounds. The abdomen was scaphoid with mild right suprapubic and left loin tenderness with normal bowel sounds. Central nervous system examination was unremarkable.

Because the nature of pain was similar to that of the past episode and, on the basis of the physical examination findings, it was decided to start pain management in the form of an intravenous stat dose of tramadol 100 mg and an intravenous normal saline bolus followed by infusion. While treatment was ongoing, the patient's suprapubic pain increased in intensity and he complained of testicular pain on the right side, for which he was given 10 mg of intravenous morphine.

Despite treatment, his pain did not subside and he started to have chest discomfort. An ECG was obtained (Figure 1), which showed ST segment elevation in the pre-cordial leads V1–V3. Therefore, he was immediately transferred to a resuscitation area and connected to the cardiac monitor and a cardiac troponin I test was immediately ordered. During this observation and about six hours after presentation, he had the urge to urinate and passed a large volume of urine with a blood-clotted stone measuring 1.0 × 0.5 cm. After passing the stone, his pain subsided. Because of the ECG findings of ST segment elevation, he remained in the resuscitation area for 24 hours.

Subsequently, all his ECG tracings (Figures 2 and 3) were normal and serial cardiac troponin was 0.01 ng/mL (normal <0.06 ng/mL) at 0 hours and 0.0 ng/mL at 8 and 24 hours. Midstream urine was tested first at six hours, which showed 300 red blood cells/HPF (1–2/HPF), 3+

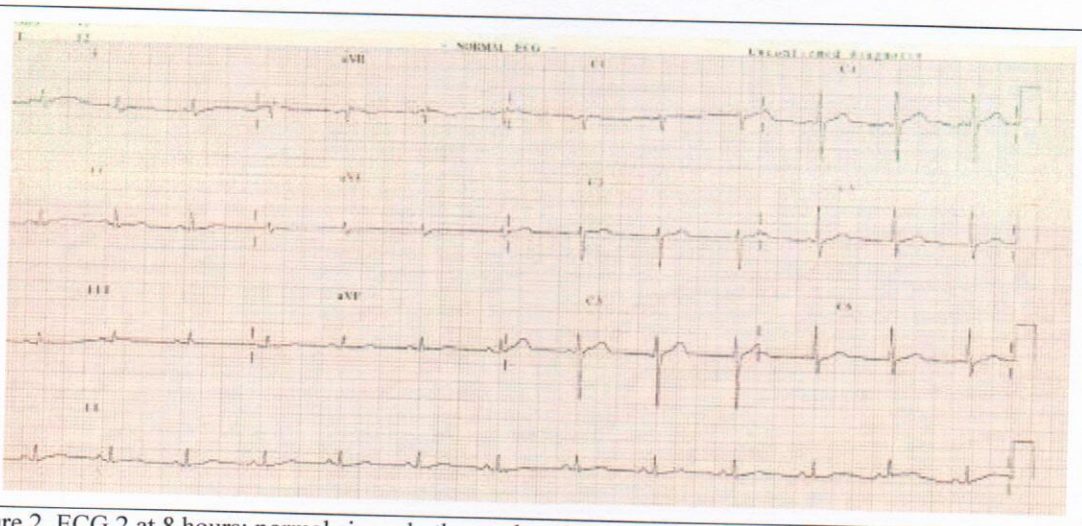

Figure 2. ECG 2 at 8 hours: normal sinus rhythm and no ST segment changes.

hemoglobin and 3+ blood; a second test at 24 hours showed 3 RBCs/HPF. A complete blood count showed white blood cells (WBCs) of 4.50 $\times 10^9$/L (4.0–11.000 $\times 10^9$/L), RBCs 5.76 $\times 10^{12}$/L (4.5–6. 5$\times$ 0^{12}/L), hemoglobin 16 g/dL (13–18 g/dL), platelets 186 $\times 10^9$/L (150–350 $\times 10^9$/L), blood urea 3.8 mmol/L (2.5–6.6 mmol/L), serum creatinine 67 µmol/L (60–120 µmol/L), sodium 144 mmol/L (135–145 mmol/ L), potassium 4.1 mmol/L (3.6–5.1 mmol/L), and chloride 105 mmol/L (95–107 mmol/L).

Echocardiography was also done in the emergency department, and it showed normal left ventricular dimensions and a contraction ejection fraction of 60% without focal wall motion abnormality. Valves appeared normal, and there was no pericardial effusion.

The patient was subsequently seen in the cardiac clinic and found to be stable. He was advised follow-up and to report to the emergency department if he experienced chest pain again. The patient was lost to follow-up following discharge.

Discussion

Acute myocardial infarction (AMI) resulting from an occlusive thrombus is recognized on an ECG tracing by ST segment elevation, and this electrophysiological phenomenon is very important in diagnosing AMI in conjunction with cardiac markers of injury, such as troponin.[2]

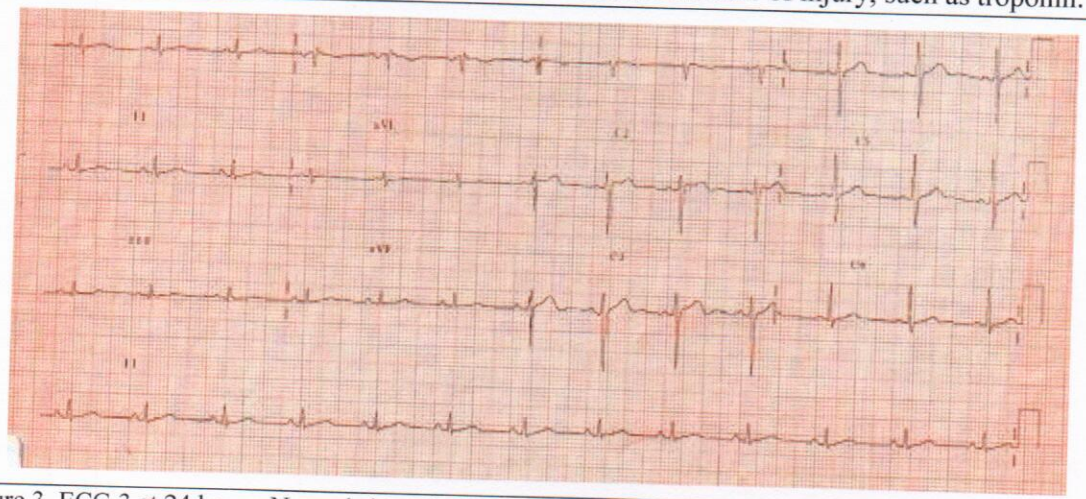

Figure 3. ECG 3 at 24 hours: Normal sinus rhythm and no ST segment changes.

Time is very limited when making the decision to perform primary angiography and/or thrombolysis in the case of AMI and, therefore, it is very important to diagnosis AMI correctly to avoid unnecessary interventions. The case reported here is an example of non-ischemic dynamic ST segment elevation. This electrophysiological reading can be due to direct or indirect cardiac phenomena and/or non-cardiac phenomena; in the literature, ST segment elevation has also been described as a normal variant.[1]

A normal male ECG pattern has been described as ST segment elevation of 1 mm or more in the pre-cordial leads V1-V4 (mainly in V2), with a normal female pattern being an elevation of 0.5–1 mm. These patterns can be labeled as normal provided there are no symptoms, the pattern is reproducible (no dynamic changes from baseline ECG tracing), the ST segment is concave, and there is a normal QT interval; in equivocal cases, echocardiography is useful to exclude regional wall motion abnormality.[3,4]

AMI presents as dynamic ST segment elevation with symptoms and positive cardiac markers. Other conditions may mimic AMI with ST segment elevation, including left bundle branch block,[5] pericarditis and myocarditis,[6] and hyperkalemia.[7] Left ventricular dysfunction or transient left ventricular apical ballooning is characterized by ECG changes, including ST segment elevation, with no evidence of coronary obstruction.[8,9] Transient ST segment elevation has been demonstrated in patients with variant angina and unstable angina.[10] In 1992, Brugada described eight patients with cardiac and ECG findings of right bundle branch block and ST segment elevation.[11] ST segment elevation appearing transiently in the Brugada-type ECG pattern may also be induced by cocaine.[12] Non-cardiac conditions that present with ST segment elevation include stroke, sub-arachnoid hemorrhage, and esophageal spasm.[1,13-16]

The mechanism of coronary arterial spasm and ST segment elevation with normal coronary arteries is poorly understood. One possibility is ischemia resulting from epicardial coronary arterial spasm. Increased sympathetic tone from mental stress can cause vasoconstriction in patients without coronary disease.[17] An alternative mechanism is microvascular spasm. Abnormal coronary flow in the absence of obstructive disease has been reported in patients with stress-related myocardial dysfunction.[18] Another possible mechanism of catecholamine-mediated myocardial stunning is direct myocyte injury. Elevated catecholamine levels decrease the viability of myocytes through cyclic AMP-mediated calcium overload[19] by interfering with sodium and calcium channel transport leading to change in the morphological pattern of myocytes.[20-22] Our patient did not have regional wall motion abnormality or elevated troponin. Therefore, it is unlikely that this patient had stress cardiomyopathy, but one of these mechanisms may represent the transient cause of coronary spasm in this patient.

We extensively searched the literature for cases similar to the one reported here, but we did not find any case of ST segment elevation due to renal stone. However, kidney-related pathologies have been described, including one report that mentioned transient ST segment elevation in critically ill patients with urosepsis and acute rejection of renal transplant. Similarly, some weak evidence of a high level of coupling factor 6 (CF6) has been found as one of the causes of myocardial ischemia in patients with end-stage renal disease. The authors propose the underlying mechanism as either stress-induced sympathetic tone or elevated catecholamine receptor density in such patients.[23,24] These mechanisms may theoretically explain the ST segment elevation in our case.

ST segment elevation is an important tool for diagnosing ongoing myocardial ischemia. However, in this era of thrombolysis where door-to-needle time is crucial and the targeted time for thrombolysis is 20 min, it is increasingly important, especially in an emergency setting, to consider conditions other than AMI that may be responsible for such an electrophysiological phenomenon to avoid unnecessary thrombolysis or primary angioplasty.

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