CASE REPORT JSLS

Acute Cholecystits Leading to Ischemic ECG Changes in a Patient with No Underlying Cardiac Disease

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ABSTRACT

Although chest pain with ST-segment elevation is often indicative of cardiac ischemia, it has also been described with surgical conditions such as acute cholecystitis. We report the case of a 34-year-old Caucasian female who was referred with symptoms consistent with acute cholecystitis. An electrocardiogram (ECG) showed unexpected changes with inferolateral ST-segment elevation indicative of an inferolateral myocardial infarct. Further investigations and analysis of the results along with the clinical picture meant an acute cardiac event was excluded.

Gallstones were seen on ultrasound and an inflamed gallbladder, consistent with acute cholecystitis, was confirmed at laparoscopic cholecystectomy. This led to the resolution of her symptoms and a return to the isoelectric baseline of the ST segments on the ECG. Five previous cases of cholecystitis induced ECG changes have been described in the literature. This case describes the youngest patient with no previous cardiac disease. We review the literature and suggest the pathophysiological mechanism to explain these findings. When the initial diagnostic interventions for chest pain with ST-segment elevation do not yield the expected results, an alternative diagnosis such as cholecystitis should be considered.

Key Words: Acute cholecystitis, ECG changes, ST elevation

INTRODUCTION

A variety of noncardiac conditions have been reported to mimic ischemic heart disease both clinically and with ECG changes. Some of these include cholecystitis,[1–10] pancreatitis,[11–14] and pneumonitits.[15] Usually these conditions lead to diffuse ECG changes, such as nonspecific T-wave inversions or ST-segment depressions.[6–10] Although chest pain with ST-segment elevation frequently indicates cardiac ischemia, it has also been reported with gastric distension,[16] acute cholecystitis,[1–3] pericarditits,[4] neoplastic invasion of the myocardium,[4] acute cor pulmonale,[4] and hypothermia.[4] Awareness of these differentials is crucial to ensuring appropriate diagnostic investigations, and early confirmation of the alternative diagnoses. It will also avoid the complications of incorrect cardiac management such as thrombolysis and even attempted rescue angioplasty, thereby leading to a reduction in morbidity and mortality. In addition to improvement in clinical care, there are also the cost implications in failure to recognize these causes, which often are across speciality from medicine to surgery.

CASE REPORT

A 34-year-old Caucasian female with a body mass index of 39 was referred to the surgical team by a general practitioner as having suspected acute cholecystitis. She gave a history of acute onset of upper abdominal pain. It was of sudden onset with a one-day duration. She was awakened with severe, cramping, colicky abdominal pain that started on both sides of the posterior thorax and migrated to the epigastric region. A pain score of 7/10 was given by the patient, and no exacerbating or relieving factors were identified. There was no periprandial correlation. The patient had associated symptoms of nausea and 6 episodes of bilious vomiting. There was no history of chest pain or shortness of breath.

Past medical history included asthma, eczema, and polycystic ovarian syndrome. Her medications included spironolactone, orlistat, and a salbutamol inhaler.

She was an ex-smoker with a 7-pack per year history prior to quitting 10 years earlier with no significant alcohol intake, and no other risk factors for coronary artery disease.

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On examination, the patient appeared to be in significant abdominal discomfort, although apyrexial. She was bradycardic at a regular 45 beats per minute and a blood pressure of 150/70. Respiratory rate was noted to be 16 breaths per minute with an oxygen saturation of 100% on room air. Cardiothoracic auscultation revealed a grade III pansystolic murmur and vesicular breath sounds throughout. Examination of the abdomen elicited tenderness in the epigastric and right upper quadrant region with localized peritonism.

Hematological and biochemical investigation revealed a neutrophilia of 12.6 x 10^9/L. (normal range, 1.8 to 7.5) with normal urea and electrolytes, liver function tests, amylase and C-reactive protein. Erect chest and plain supine abdominal radiographs were reported as normal. The urine dipstick was clear, and βHCG was negative.

However, the patient's ECG **(Figure 1)** revealed a sinus bradycardia with 1-mm ST segment elevation in leads V5, V6, and aVF, and 2-mm ST-segment elevation in leads II and III. In view of the unexpected ECG findings. which did not correlate with the clinical signs and symptoms, the patient was initially managed conservatively by the medical team on the Acute Coronary Syndrome (ACS) protocol for anticoagulation with high-dose low molecular weight heparin, aspirin, and clopidogrel, whilst a 12-hour troponin was awaited. A repeat ECG after 30 minutes showed some resolution, but 30 minutes after that showed persistent ST-elevation in the lateral leads. In view of the atypical nature of the pain and delayed presentation since the onset of the pain at the hospital, thrombolysis was not indicated. Troponin I levels obtained both on admission and 12 hours later were noted to be negative (<0.02ng/mL), which made an acute coronary event unlikely. An echocardiogram showed a mildly enlarged left atrium and left ventricle, but with a good ejection fraction and no regional wall motion abnormalities.

An ultrasound scan organized by the surgical team to rule out gallbladder pathology, revealed only a thin-walled gallbladder that contained multiple small gallstones, with a common bile duct of 5mm. The pancreas, liver, and kidneys were reported to be normal. In view of continuing episodes of pain, the decision was made to proceed with an emergency laparoscopic cholecystectomy. At operation, a thick-walled inflamed gallbladder was found with multiple gallstones within it. No other abnormality was found, and there were no complications with the surgery. Histology of the specimen confirmed a hemorrhagic mucosa with multiple stones present within the gallbladder, and the wall had features of florid acute cholecystitis. A postoperative ECG was done, in which the abnormalities from admission had resolved **(Figure 2)**.

DISCUSSION

We describe the case of a 34-year-old woman with epigastric and right upper quadrant abdominal pain with clinical features suggestive of acute cholecystitis but associated ST-segment elevation in the distribution of the circumflex and right coronary arteries on ECG. Initally, this prompted treatment under the ACS protocol with therapeutic anticoagulation using low-molecular weight heparin. All subsequent

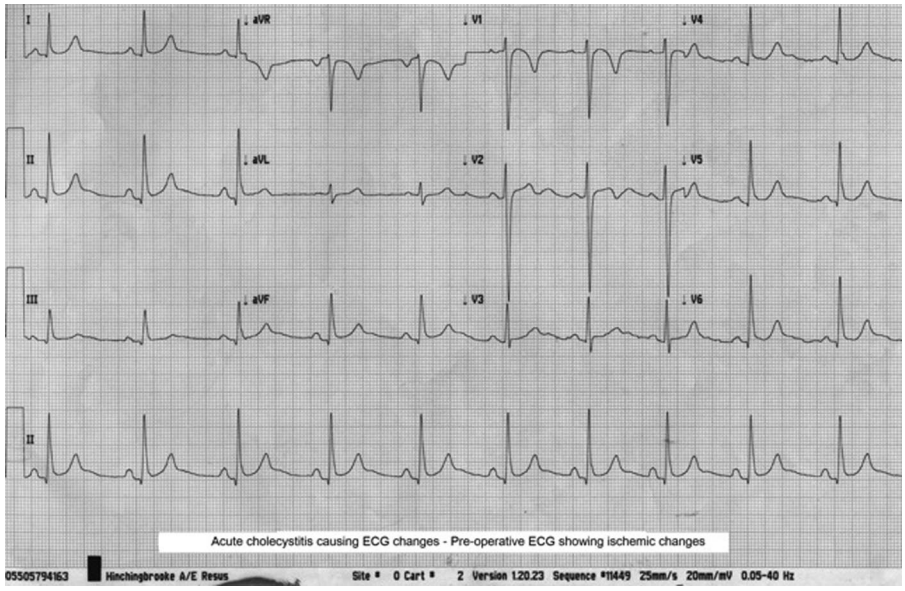

Figure 1. Acute cholecystitis causing ECG changes – Postoperative ECG showing ischemic changes.

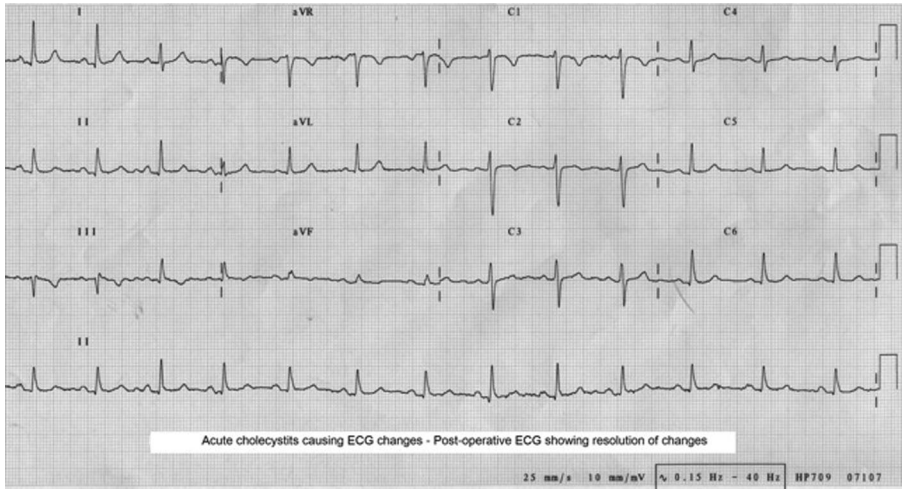

Figure 2. Acute cholecystitis causing ECG changes – Postoperative ECG showing resolution of ischemic changes.

cardiac investigations were unremarkable. A laparoscopic cholecystectomy for acute cholecystitis was carried out that led to a full resolution of the patient's ECG changes.

Many conditions can temporarily alter an electrocardiogram. Inflammation of the hepatobiliary system and pancreas, along with viral myocarditis has been noted to produce changes in electrocardiograms.[17] Acute inflammatory and ulcerative conditions involving the gallbladder or duodenum cause irritation and spasticity of surrounding structures. This can create reflex stimuli through autonomic pathways to restrict or alter the coronary blood supply, maybe such that existing minor deficiencies in the coronary circulation become manifest.[2] It is possible that acute upper abdominal disease can prematurely reveal subclinical changes in the coronary circulation. The alterations in the ECG could have been caused by temporary myocardial ischemia, since the changes disappeared. However, the exact pathophysiological mechanism underlying the ECG changes remain unclear.

Gallbladder distension has been shown to increase heart rate, arterial blood pressure,[18,19] and plasma rennin levels,[20] and studies in animals suggest that its effect on coronary blood flow may be a significant factor underlying these ECG changes.[21,22] Decreased coronary blood flow was demonstrated in dogs during distension of the common bile duct,[23] and in pigs, which showed a graded response to the magnitude of distension.[24] The reflexive coronary vasoconstriction was unaffected by atropine and propranolol, but was abolished with phentolamine,[20] indicating the reflex has an α-adrenergic sympathetic arc.

A diseased gallbladder has been associated with changes in the ECG similar to those of ischemic heart disease since 1878.[25–27] This prompted studies[28,29] examining the ECG effects of biliary distortion. In these studies, the ECG changes were usually diffuse, nonspecific, T-wave inversions or ST-segment depressions.

There have only been 5 previous reported cases of ST-segment elevation attributed to cholecystitis in the medical literature.[1–5] The patients' ages range from 46 to 64 years old. Four were men, and 2 had a history of coronary artery disease. Our patient is, therefore, the youngest to be reported with this phenomenon of ST-segment elevation. Due to the absence of any history of cardiac disease and negative cardiac enzymes, no invasive cardiac intervention was undertaken. Three of the previously reported patients underwent cardiac catheterization, all being normal,[1,2,4] and one underwent thrombolytic therapy.[3] Two of the patients who had angiograms, had resolution of ECG changes after cholecystectomy, one patient who was investigated with cardiac catheterization had resolution with antibiotic therapy,[1] and 2 had resolution without further treatment.[3,5]

CONCLUSION

ST-segment elevation is specific enough that this objective finding alone, in a setting with appropriate clinical suggestion of myocardial infarction, can justify reperfusion therapy.[30] However, we feel it is important for clinicians to be aware of the uncommon causes of ST-segment elevation, because the risk of reperfusion therapy, especially thrombolytic therapy, would not be balanced by any potential benefit. Furthermore, delays in diagnosing cholecystitis may lead to serious complications, including sepsis and pancreatitis.

When the initial diagnostic interventions for chest pain with ST-segment elevation do not yield the expected results (ie, normal cardiac enzyme results, and an atypical clinical picture), an alternative diagnosis such as cholecystitis should be considered.

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