

CASE REPORT

Acute cholecystitis and myocardial infarction: a case study with coronary involvement

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Key Clinical Message

Possible links between inflammatory stimuli and atherothrombotic disease in the context of gallbladder pathology are not well understood. Our case demonstrates that clinical suspicion of cardiac disease after a diagnosis of acute cholecystitis should remain high in light of the dire consequences of a missed diagnosis.

Keywords

Acute cholecystitis, cardiac catheterization, inflammation, myocardial infarction.

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Case Report

We present the case of an 83-year-old female with a past medical history of non-insulin dependent diabetes mellitus (NIDDM), hypertension, hyperlipidemia, gastro-esophageal reflux disease (GERD), and chronic kidney disease (CKD), who presented to the emergency room at Mount Sinai Hospital, New York complaining of worsening, intermittent, sharp, right upper quadrant pain (RUQ) for approximately 3 months accompanied by nausea and occasional constipation. The patient gave a 6/10 pain score. She denied diarrhea, blood in the stool, dysuria, syncope, chest pain, shortness of breath, palpitations, or leg swelling. She reported no bowel movement in the past 2 days. Her exercise tolerance was one block, limited by worsening dyspepsia.

On physical examination, her blood pressure was 156/80, temperature was 36°C, pulse was 93 beats per minute, and respiratory rate was 16 breaths per minute. Her abdomen was soft and bowel sounds were normal with no distention, tenderness, or mass with the rest of her physical exam being unremarkable. She was afebrile and

had leukocytosis with a white blood cell (WBC) count of 20,000. Liver function tests (LFTs) were normal, hemoglobin A1C was 9.4%, and C-reactive protein was 15.5 mg/L. Her initial electrocardiogram (EKG) showed a normal sinus rhythm with a left axis deviation and a non-specific ST and T wave abnormality (Fig. 1).

Abdominal imaging with an RUQ ultrasound revealed a 2-cm stone at the neck of the gallbladder associated with common bile duct dilatation, gallbladder wall thickening, and elicited sonographic Murphy's sign to suggest acute cholecystitis. An additional 1.3-cm stone was seen in the body of the gallbladder, which was slightly larger than one seen on an ultrasound from 2011. A CT scan showed distention of the gallbladder, with prominent mucosal enhancement. She was initiated on IV piperacillin and scheduled for laparoscopic cholecystectomy.

Prior to the planned operation on the next day, however, an EKG revealed 2–3 mm T-Wave Inversions (TWI) in inferior leads without chest pain (Fig. 2). Cardiac biomarkers were elevated with a troponin (reference range 0.01–0.05 ng/mL) and CK-MB (reference range 1.3–

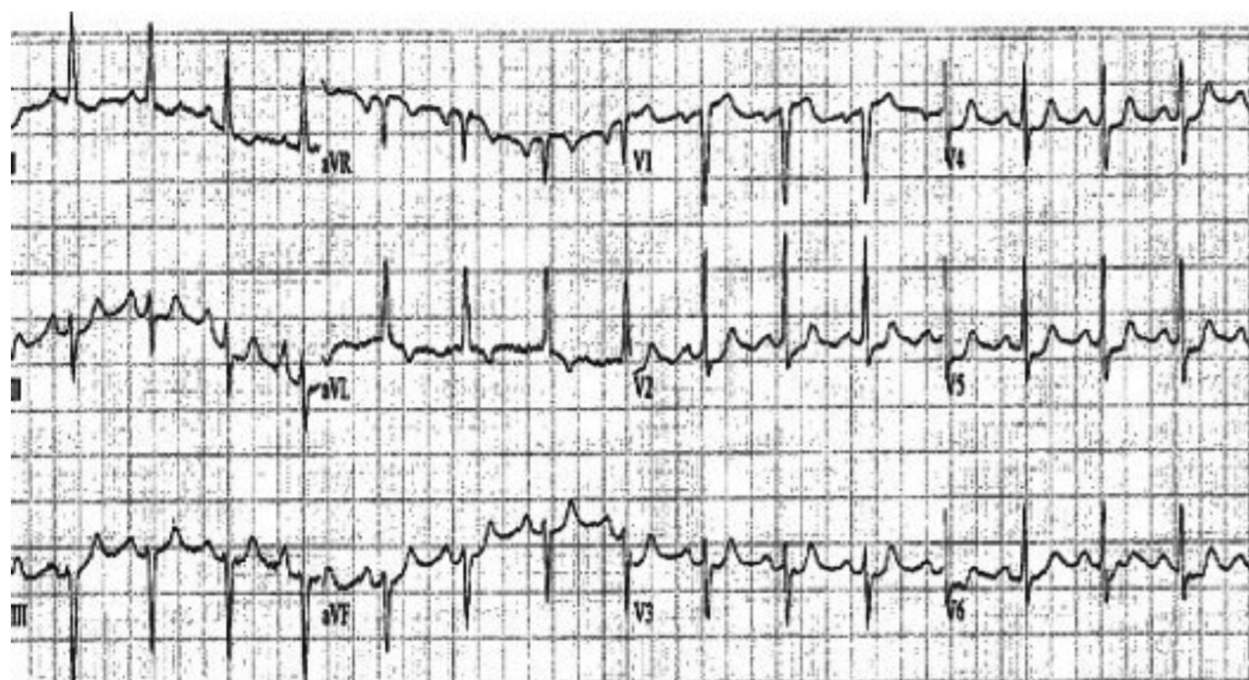


Figure 1. Electrocardiogram (ECG) taken at baseline, upon hospital admission.

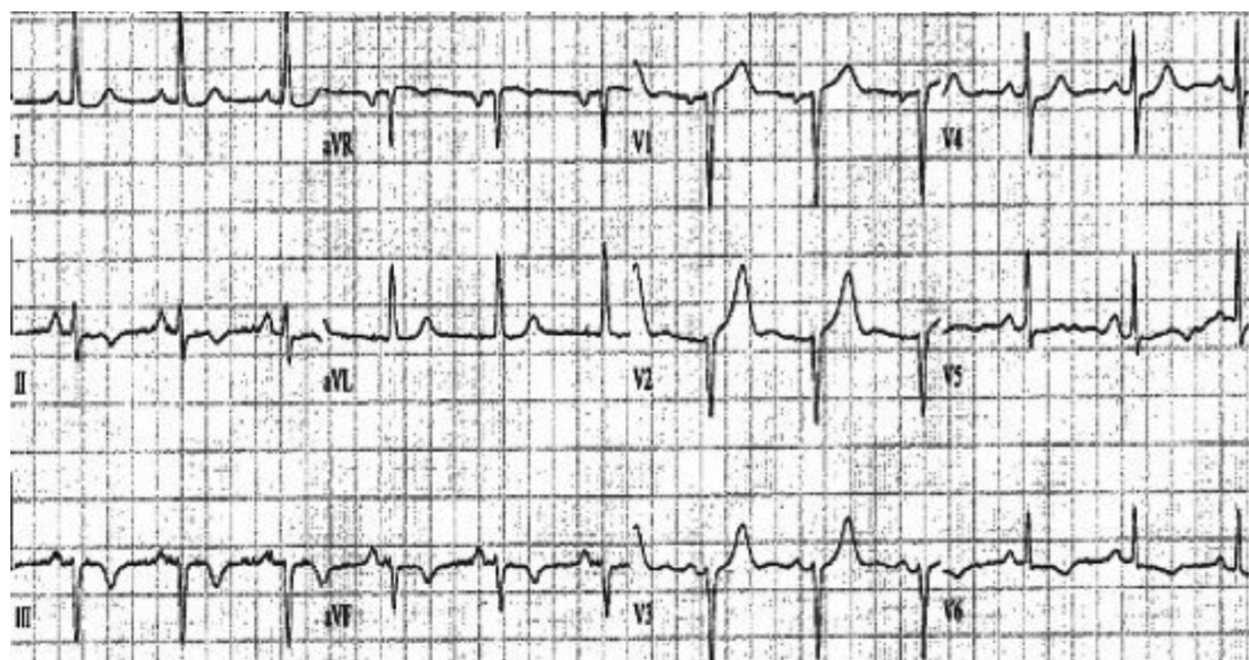


Figure 2. Electrocardiogram (ECG) taken prior to planned cholecystectomy.

6.3 ng/mL) of 7.4 and 43.2 ng/mL, respectively, and she was therefore referred for diagnostic cardiac catheterization. The coronary angiogram revealed triple vessel coronary artery disease (CAD), with >60% stenosis in the left main (LM) artery, left anterior descending (LAD) artery,

total occlusion in the left circumflex (LCx) artery, and a high-grade culprit lesion in the right coronary artery (RCA) (Figs. 3 and 4). In light of this extensive coronary vasculopathy, the patient was referred for cardiothoracic consultation for coronary artery bypass grafting (CABG),



Figure 3. Coronary angiogram displaying the location of the high-grade culprit lesion in the right coronary artery (RCA).

but was ultimately deemed to be too frail. She underwent percutaneous coronary intervention (PCI) the following day for the RCA lesion with drug eluting stent (DES) placement. Her troponin and CK-MB levels began to trend downwards thereafter and her EKG showed a normal sinus rhythm with a nonspecific ST and T wave abnormality (Fig. 5). In terms of acute cholecystitis, the patient was asymptomatic and was discharged home on amoxicillin clavulanate for a 7-day course. Upon 30-day follow-up visit, the patient remained asymptomatic and reported no chest pain. Two years after complete revascularization, and having completed her 1-year course of dual antiplatelet therapy, the patient underwent elective laparoscopic cholecystectomy, despite being informed of the increased risk of the procedure, given the patient's recent cardiac history. She successfully underwent the procedure and reported no complaints at her 30-day follow-up visit (Table 1).

Discussion

Acute inflammatory diseases are known to be linked to accelerated atherosclerosis [1, 2]. Patients are at a greater risk for MI than the general population and possible risk factors for CAD have been identified, including abnormal levels of inflammatory markers, that is, C-reactive protein [3–5]. As atherosclerosis is considered a chronic inflammatory disorder of the arterial wall, links between inflammatory stimuli and atherothrombotic disease are naturally intuitive. There are limited data, however, examining similar associations in the context of gallbladder pathology.

Previous studies have noted electrocardiographic changes consistent with STEMI with or without cardiac

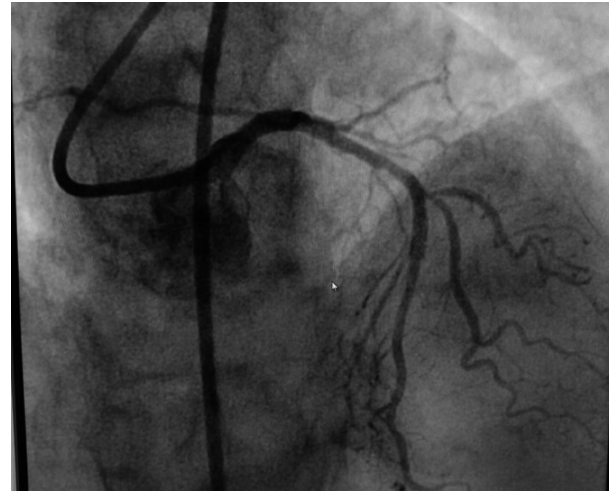


Figure 4. Coronary angiogram of the left main, the bifurcation of the left anterior descending artery (LAD) and its first diagonal branch (D1), and total occlusion of the left circumflex (LCx) artery.

biomarker elevation in patients admitted with acute cholecystitis [6–9]. Upon inspection of coronary arteries by coronary angiography, there were no signs to indicate CAD, ischemia, myocarditis, pericarditis, or coronary spasm. Postulated mechanisms for myocyte necrosis in these settings have included biliary-cardiac reflex, prolonged tachycardia or septic shock, but not atherosclerosis.

In contrast, our case is unique in that the patient had concomitant extensive CAD complicated by acute MI coinciding with gallbladder wall inflammation. We suspect that this case of gall bladder inflammation with subsequent MI might reflect acute biliary inflammation predisposing to arterial thrombosis. This hypothesis is supported by the well-established association observed between local inflammatory processes and acute MI.

Conclusion

Although previous case reports have ruled out ischemia as a cause of abnormally high levels of troponin in patients with acute cholecystitis, suspicion should remain high because of the significance of a potential misdiagnosis and especially if the patient has a high-risk cardiovascular profile, as was in our case. This calls for periodic assessment of EKG changes, trends in cardiac enzymes, and cardiac symptoms. In our case, in light of new-onset EKG changes, concomitant with elevated cardiac biomarkers, we decided to perform diagnostic angiography and the patient was diagnosed with an NSTEMI with a high-grade coronary lesion. Thus, while ischemic signs and laboratory workup may be positive in highly acute inflammatory disease without any real, cardiac

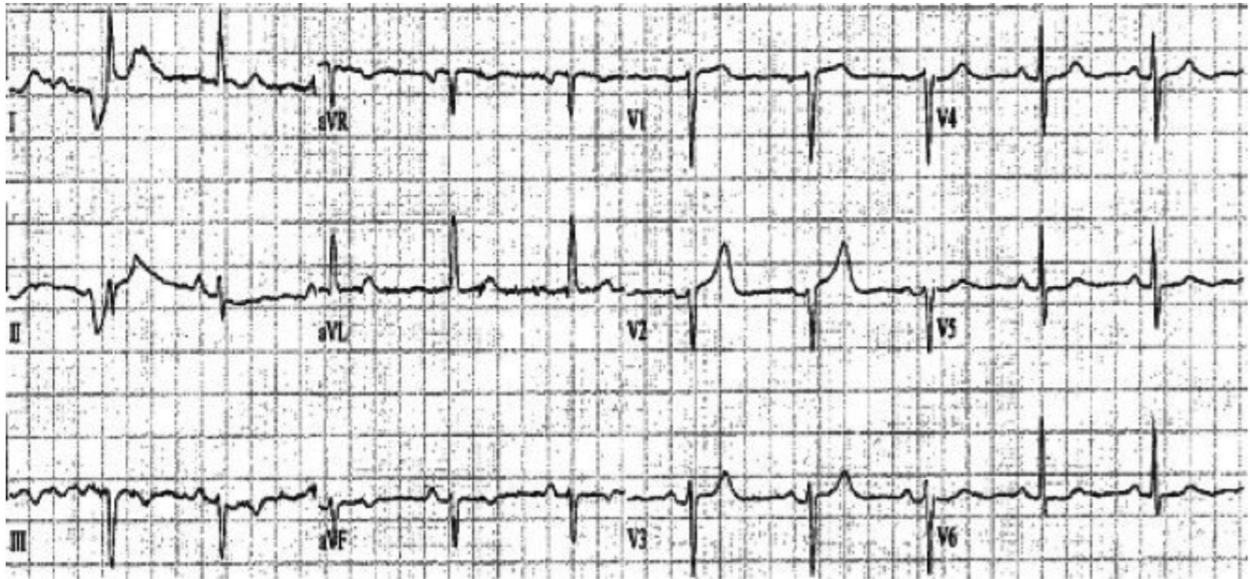


Figure 5. Electrocardiogram (ECG) taken post percutaneous coronary intervention (PCI).

Table 1. White Blood Cell differential counts of subject throughout hospital admission.

Cell population	Upon admission	Preoperative	Discharge
Leukocyte	19.3 × 10/L (reference range: 5–10 × 10/L)	13.9 × 10/L	8.0 × 10/L
Lymphocyte	64.8% (reference range: 20–40%)	46.2%	31.9%
Neutrophil	85.1% (reference range: 50–70%)	74.3%	61.8%
Basophil	2.7% (reference range: 0–1%)	2.1%	0.8%
Eosinophil	4.5% (reference range: 0–3%)	3.3%	2.3%
Monocyte	20.2% (reference range: 2–6%)	17.9%	11.6%

abnormality, the clinical suspicion of a major adverse cardiac event should remain high in light of the detrimental consequences of a missed diagnosis. We thereby consider this case to be noteworthy, particularly in light of the fact that all other reports of such nature in the medical literature are outdated and bereft of such findings.

Conflict of Interest

None declared.

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