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CASE REPORT

An Atypical Presentation of Acute Cholecystitis with Left Sided Chest Pain and ST Elevation – A Case Report

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Background: ST elevation combined with typical chest pain is an indication for acute coronary vascularization and is usually associated with acute myocardial infarction. Herein, we present an unusual case of ST elevation.

Case Presentation: A 57-year-old male patient presented to the emergency department with chest pain radiating to both arms and the back. Typical clinical presentation and inferolateral ST elevations were suggestive of ST elevation myocardial infarction. Interestingly, coronary occlusion was excluded by coronary angiography. Despite extensive diagnostic workup, no underlying diagnosis was made. Four days later, the patient returned and reported pain in the right upper abdomen. Clinical presentation, laboratory analysis, and imaging features led to a diagnosis of calculous acute cholecystitis. Laparoscopic cholecystectomy was performed, and the diagnosis was confirmed. Electrocardiographic changes and pain resolved completely.

Conclusion: Acute calculous cholecystitis is initiated by gallbladder distension due to biliary duct occlusion caused by gallstones. ST elevations in response to gallbladder distension have been demonstrated in animal models. We hypothesize that the ST elevations observed in this patient with chest pain were linked to stone-mediated distension of the gallbladder, leading to reflex coronary vasoconstriction.

Keywords: electrocardiography, ECG, STEMI, cope's sign, chest pain, case report

Background

Myocardial infarction affects more than 7 million people worldwide every year.[1] It is differentiated according to features of the 12-lead electrocardiogram (ECG) into non-ST-elevation myocardial infarction and ST-elevation myocardial infarction (STEMI). STEMI is defined by the characteristic symptoms of myocardial ischemia associated with diagnostic ST elevation on 12-lead ECG.[2] Both American and European Guidelines[3,4] recommend an immediate reperfusion strategy for STEMI patients.

The global prevalence of gallbladder and biliary diseases is 193 million.[5] Cholecystitis affects approximately 200'000 people yearly in the United States alone.[6] Typical presentations include acute onset of pain in the right upper quadrant of the abdomen and fever. However, uncharacteristic pain localization, such as chest pain, has been described.[7] Patients with acute cholecystitis can show ECG changes without cardiac causes. Previously observed changes include bradycardia, atrioventricular block, T-wave inversions[8–11] and ST elevations.[12–18] Here, we present the case of a patient with early stage of acute cholecystitis with diagnostic ST elevations in the inferolateral leads (defined as ≥1 mm in more than two contiguous leads[2,19]). This is an unusual case report because there were no clinical or laboratory signs of cholecystitis at the time of the ST elevation.

Case Presentation

A 57-year-old Caucasian male patient presented to the emergency department. He reported a sudden onset of chest pain radiating in both arms and the back starting three hours before. The pain intensity was reported to be 7 on a scale of 1–10.

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The patient's medical history included chronic venous insufficiency. He took no regular medication and had no known allergies. The recorded 12-lead ECG showed ST elevations of ≥ 1 mm in leads II, III, aVF, V4, and V5 (see Figure 1). The clinical presentation and diagnostic ST elevations were highly suggestive of STEMI. The patient was administered 300 mg acetylic acid per os and immediately transferred for diagnostic coronary angiography and probably percutaneous coronary intervention. Contrary to expectations, coronary occlusion was excluded. Coronary artery sclerosis without relevant stenosis was noted.

Further diagnostic workup in the emergency department followed. The patient still reported chest pain with an intensity of 7 on a scale of 1–10. Focused echocardiography revealed no relevant wall-motion disorders. Computed tomography showed a normal thoracoabdominal aorta and no pulmonary embolism. Cholecystolithiasis without any signs of cholecystitis was noted. Further investigation of the abdominal and thoracic organs displayed no explanation for the patient's symptoms. Normal values were recorded in laboratory analyses for inflammation parameters, cardiac markers, liver and cholestasis parameters (see Table 1, column "Day 1"). The pain significantly decreased with the administration of analgesic drugs and proton pump inhibitors. The "Big Five" (myocardial infarction, aortic dissection, pericardial tamponade, pneumothorax, and lung embolization) could be ruled out with high certainty. However, no clear diagnosis was found for the chest pain. After discussion with the patient, he was discharged in a shared decision-making process.

He presented for a follow-up consultation the following day. Clinical examination results were unremarkable. The pathological changes on the 12-lead ECG were still present, but subsided. Laboratory findings (see Table 1, column "Day 2") showed mild leukocytosis (8.2 G/l, reference range 2.6–7.8 G/l) and a CRP level of 6 mg/l (reference range <5 mg/l). On sonography, the gallbladder was full and cholecystolithiasis without thickening of the gallbladder wall was observed.

Three days after the initial presentation, the patient complained of aggravated pain in the right upper abdominal quadrant. Murphy's sign was positive on clinical examination. Sonography revealed cholecystolithiasis and thickened gallbladder wall with no bile duct dilatation. Laboratory analysis revealed signs of inflammation (see Table 1, column "Day 4"). Cholecystitis was suspected and the patient was admitted to the hospital for diagnostic laparoscopy and cholecystectomy. An inflamed gallbladder was described intraoperatively, and acute ulcerative and phlegmonous cholecystitis was histologically confirmed. The postoperative course was uncomplicated, and the pathological ECG tracings completely resolved (Supplementary Figure 1).

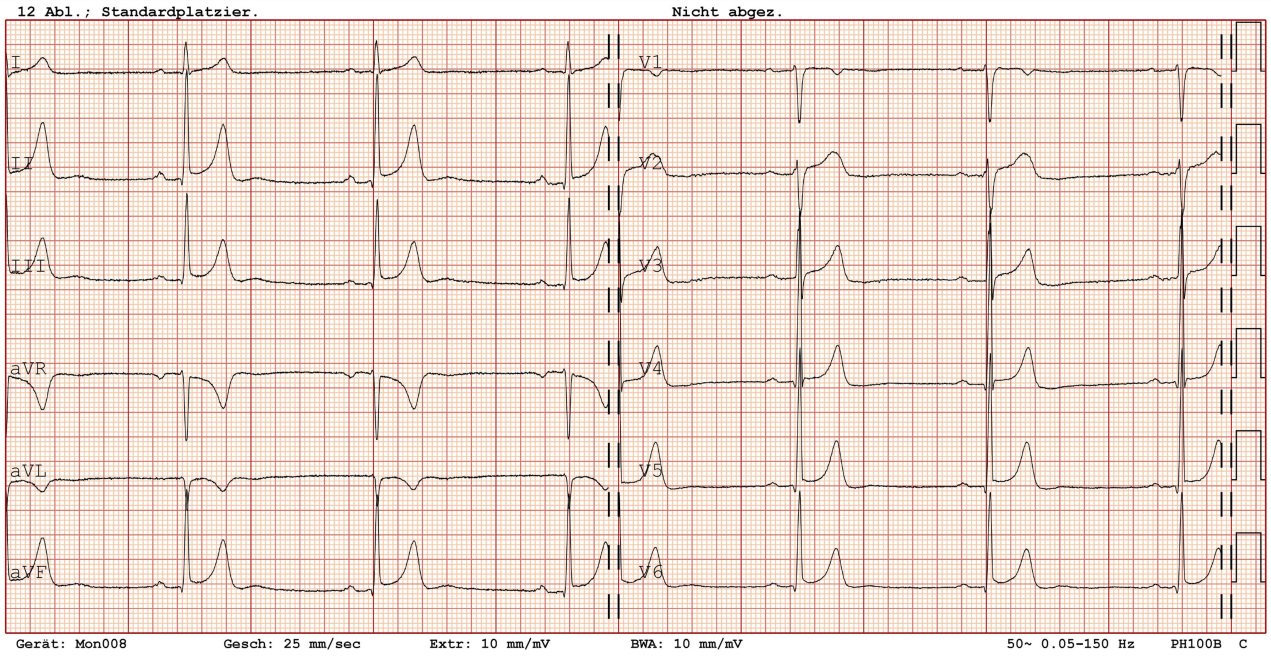

Figure 1 12-lead Electrocardiogram at time of presentation.

Table 1 Laboratory Results from the Three Presentations in the Emergency Department with Reference Ranges

	Reference	Day 1	Day 2	Day 4
Hemoglobin (g/l)	127–163	137	136	128
Hematocrit (%)	0.37–0.46	0.39	0.41	0.39
White-cell count (Giga/L)	2.6–7.8	6.7	8.2	14
Platelet Count (Giga/L)	130–330	210	197	201
Neutrophils (Giga/L)	0.9–4.5	4		10.94
C-Reactive Protein (mg/L)	<5	<5	6	103
Sodium (mmol/L)	136–145	139	139	138
Potassium (mmol/L)	3.4–4.5	3.9	4.2	4.2
Urea (mmol/L)	2.76–8.1	6.8		7.3
Creatinine (µmol/L)	59–104	89	91	87
Glucose (mmol/L)	4.11–6.05	5.7	5.6	5.1
Calcium (mmol/L)	2.15–2.50	2.21		2.18
Albumin (g/l)	35–52	39	42	40
Lipase (U/l)	13–60	39	44	28
Alanine Aminotransferase (U/l)	10–50	22	10	40
Aspartate Aminotransferase (U/l)	10–50	24	25	42
Alkaline Phosphatase (U/l)	40–129	88	83	98
Gamma GT (U/l)	8–61	20	15	24
Total Bilirubin (µmol/L)	<21	4		8
Troponin T hs (ng/l)	<14	6	32	24
After 1 hour		6	33	

The signs and symptoms of this patient at initial presentation were highly suggestive of ST-elevation myocardial infarction. Initially, no clinical signs or laboratory findings associated with cholecystitis were found. Pain localization in the chest with radiation into both arms and ST-segment elevations on the 12-lead ECG were misleading and prompted the algorithm for acute coronary syndrome.

Discussion

We hypothesized that the initial distention of the gallbladder in our patient led to reflex coronary vasoconstriction and the observed ST elevations. The pathophysiology of calculous acute cholecystitis begins with (transient) obstruction of the cystic duct by gallstones. Initially, this leads to increased intraluminal pressure and distension of the gallbladder.[6] Edema, inflammation, and tissue necrosis begin to develop only 2–4 days later.[20] In addition, atypical pain localization such as chest pain has been described in acute cholecystitis.[9]

Our patient likely presented at the beginning of the cholecystitis development. At the time of the first presentation, we assumed that only distension of the gallbladder and the subsequent pain were present. Gallbladder inflammation and florid cholecystitis had only developed on day four. Inflammatory markers and imaging changes at that timepoint were consistent with the diagnosis of acute cholecystitis.

Three mechanisms have been described in literature explaining the pathophysiology of ECG changes in patients with cholecystitis. The first is a reflex mediated by the vagus nerve, mostly referred to as the cardiobiliary reflex. Stimulation of the gallbladder, such as mechanical stimulation during surgery[13] or dilation of the gallbladder,[21] can lead to activation of efferent cardiac vagal nerves. The resulting ECG observations included bradycardia and atrioventricular block.[13] However, vagus nerve activation alone does not explain the development of ST elevation.

Another possible mechanism is the anatomical proximity of the gallbladder to the heart. Most cases of cholecystitis and ST elevation report an ECG mimicking inferior or inferolateral STEMI.[12–16] Local inflammation of the gallbladder in cholecystitis can lead to irritation of the diaphragm and the inferior myocardial wall, therefore affecting the myocardial tissue due to the close proximity. This irritation may induce the changes observed within the 12-lead ECG. Interestingly, predominantly inferior ST elevations have been described in patients with pancreatitis.[22] In these patients, as in cholecystitis, the inflammatory processes of the pancreatic tissue are in anatomical proximity to the heart, supporting the above theory.

The third mechanism has been described in animal studies, where reflex coronary vasoconstriction with reduced coronary blood flow has been observed after experimental distension of the gallbladder.[21,23] In a study of anesthetized pigs, the decrease in coronary blood flow was linear with the grade of gallbladder distension.[21] This effect was absent after surgical removal of the vagus nerve. The authors suggested an afferent pathway of the reflex response within the vagus nerve and involvement of sympathetic mechanisms in the coronary arteries of the efferent pathways.

In the present case, no laboratory or clinical signs of cholecystitis were observed at the same time as the ST elevations. Subsequently, inflammation and cholecystitis developed. It is tempting to hypothesize that the bile duct was transiently occluded by gallstones at the time of initial presentation. The proposed mechanism for ST elevation in our case indicates reflex coronary vasoconstriction triggered by gallbladder dilation.

Of interest, the presence of ST elevations in combination with reciprocal ST-segment depressions would have been particularly suggestive of acute myocardial ischemia.[24] Furthermore, in patients with STEMI and reciprocal ST-segment depressions higher maximum troponin levels and higher mortality have been observed compared to patients without reciprocal ST-segment depressions.[25]

It is important to be aware of atypical symptoms of the early stages of cholecystitis and the uncommon causes of ST elevation. Our case report describes an unusual case of ST elevation in a patient without any laboratory or clinical findings of cholecystitis. Previously described ST elevations associated with cholecystitis have been reported during advanced stages of inflammation. We propose the theory that in our patient, the mere presence of gallbladder dilation led to reflex coronary vasoconstriction, leading to ST elevation and therefore mimicry of STEMI. We believe this case report to be a relevant contribution to other physicians who encounter similar clinical situations.

Abbreviations

CRP, C-reactive protein; ECG, electrocardiogram; STEMI, ST-elevation myocardial infarction.

Data Sharing Statement

All data generated or analyzed during this study are included in this published article and its supplementary information file.

Ethics Approval

No institutional ethics approval was required to publish the case details.

Informed Consent for Publication

Written and signed informed consent from the patient is available.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no competing interests.

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