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Acute Cholecystitis Presenting as Acute Coronary Syndrome with ST-Segment Depression

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ABSTRACT

Background: Acute cholecystitis may mimic acute coronary syndrome (ACS), both symptomatically and on electrocardiography (ECG), which can lead to cardiac interventions. While ST-segment elevation in such scenarios has been reported, ST-segment depression is a rarer presentation.

Case Summary: A 55-year-old woman with a history of Hypothyroidism presented to the emergency department with central chest pain, epigastric discomfort, dyspnea, and nausea since few days. ECG showed ST depression in infero-anterior leads (II, III, aVF, V1-V6). Cardiac biomarkers (CPK-MB=2.5 ng/mL, Troponin I=0.01 ng/mL) were normal suggestive of Unstable Angina. Coronary angiography revealed only minor coronary artery disease. Abdominal ultrasound confirmed acute calculous cholecystitis. The patient was managed conservatively and planned for cholecystectomy. Patient had gradual resolution of symptoms and dynamic ECG changes (T inversion in infero-anterior leads). This case report has been so far the first case publication from Nepal but very few case reports have been published internationally.

Conclusion: This case demonstrates that acute cholecystitis can present with ST-segment depression mimicking ACS. Clinicians should maintain a broad differential diagnosis in patients with chest pain, especially when cardiac workup is inconclusive and gastrointestinal history is relevant.

Keywords: Acute cholecystitis; ACS; electrocardiography; Nepal

INTRODUCTION

Myocardial infarction affects over 7 million people globally each year.[1] It is diagnosed by a combination of clinical features, elevated cardiac biomarkers, and characteristic ECG changes.[2] ST-elevation myocardial infarction (STEMI) demands urgent reperfusion therapy. However, non-cardiac causes, such as gastrointestinal disorders, can occasionally mimic these findings.[3] Gallbladder disease is prevalent globally, with cholecystitis affecting approximately 200,000 individuals annually in the United States alone.[4] Though right upper quadrant pain is typical, cases with chest pain and even ischemic ECG changes have been reported.[5] We present a case of acute calculous cholecystitis presenting with ST-segment depression, closely resembling ACS.

CASE REPORT

A 55-year-old woman, known Hypothyroidism presented to the emergency department on 2025/06/09 with several days of central chest pain (non-radiating), epigastric discomfort, nausea, vomiting, and dyspnea. There was no sweating, syncope, limb weakness, or dizziness.

Vital Signs at Presentation

- Pulse: 74 bpm, BP: 130/90 mmHg, RR: 20/min, Temperature: 97.3°F, SpO_2: 94% on room air

Investigations

- ECG: ST depression in leads II, III, aVF, V1–V6 as shown below in Fig 1 and 2.
- Troponin I: 0.01 ng/mL CPK-MB: 2.5 ng/mL
- Echo: Mild Mitral Regurgitation, Normal LV systolic function (EF-62%)
- Chest X-ray-Normal
- Serology tests:
  1. HBsAG: Non-Reactive
  2. HIV-1 and II Ab: Non-Reactive
  3. RPR (VDRL) test: Non-Reactive
  4. HCV (Hepatitis C Antibody): Non-Reactive

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Other relevant biochemistry, hematology and radiology investigations were done as shown below in Table 1.

Table 1. Laboratory informations of patients.

Parameters	Value	Reference Range
Biochemistry		
Urea	15.2 mg/dL	14-45
Creatinine	0.5 mg/dL	0.4-1.4
Sodium (Na+)	140 mmol/L	135-145
Potassium (K+)	4 mmol/L	3.5-5.2
Haematology		
Haemoglobin	10.7 g/dL	11-17
TLC	5300 /cu. mm	4000-11000
RBC	3.9 million/cu.mm	3.5-5.5
Neutrophils	53%	40-75
Lymphocytes	40%	20-45
Eosinophils	5%	1-6
Monocytes	2%	2-10
Platelets	136000 /cu.mm	150000-400000
MCV	83.5 fL	76-96
MCH	27.4 pg	27-32
MCHC	32.9 g/dL	31-34
RDW CV	12%	11.5-14.5
Thyroid Function Test (TFT) & Amylase		
T3	3.79 pg/mL	2.0-4.4
T4	1.06 ng/dL	0.8-1.8
TSH	1.45 µU/mL	0.4-4.0
Amylase	54.4 U/L	30-110

Radiology findings

- Liver: Mild fatty liver
- Gallbladder: 9 mm calculus, edematous wall (up to 4 mm)
- No abnormalities in pancreas, spleen, kidneys, uterus, adnexa

Coronary Angiography (CAG) performed on 2025/06/09 revealed Normal coronaries except minor Ostio-proximal plaque in D1 as shown below in figure 3.

Symptoms were worsened by fatty meals and improved with PPIs, supporting a gastrointestinal etiology. The patient was admitted to the CCU after CAG, treated medically, and discharged with supportive medical therapy. Patient has undergone Surgery consultation and advised for Cholecystectomy.

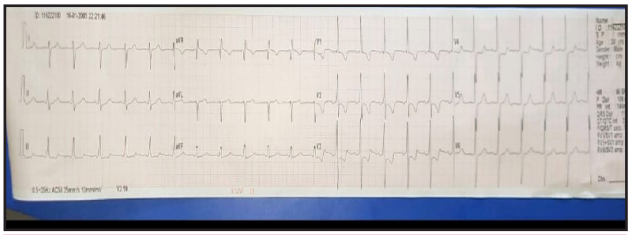

Figure 1. ECG showing ST depression in II, III, AVF and V1-V6.

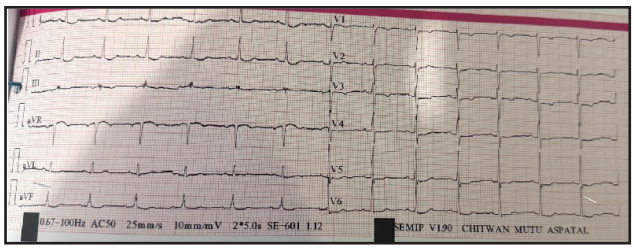

Figure 2. ECG showing gradual resolution of ST changes with T inversion in inferior and anterior leads.

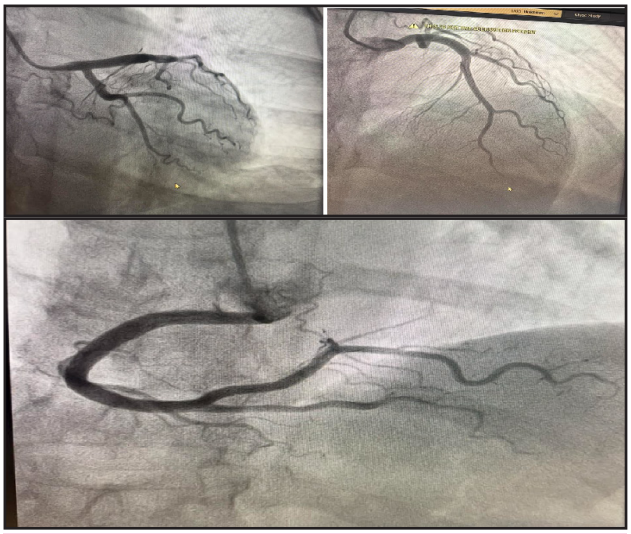

Figure 3. CAG shows normal coronaries except Ostioproximal D1 with minor plaques.

DISCUSSION

This case aligns with prior literature demonstrating Acute Cholecystitis can simulate ACS. However, unlike many previous reports, which highlighted ST-elevation as the mimicker[6], this patient presented with ST-depression: a subtler and possibly more misleading finding.

In comparison to a published case by [HM et al.][7], where ST-elevation preceded any clinical or imaging signs of cholecystitis, our patient had clear ultrasound findings at the time of ECG changes. Studies by Sethi P et al.[8], and Nasir GM et al.[9], also reported

ST segment elevation with Normal Coronaries on Angiography.

The mechanism behind ECG abnormalities in cholecystitis includes[10]:

1. Cardio-biliary reflex - mediated via the vagus nerve, causing coronary vasospasm.
2. Diaphragmatic irritation - due to anatomical proximity, affecting the inferior myocardium.
3. Reflex coronary vasoconstriction - documented in animal models after gallbladder distension.

In our patient, ST-depression in inferolateral leads may reflect diaphragmatic or visceral pain-mediated autonomic influences without direct myocardial ischemia.

Unlike the earlier case that showed ST-elevation with normal ultrasound initially and progression to cholecystitis over days, our case had concurrent ultrasound evidence and ECG changes supporting the hypothesis that the inflammatory process itself, not just distension, can trigger cardiac mimicry.

CONCLUSIONS

This case highlights an atypical presentation of acute cholecystitis mimicking ACS through ST-depression on ECG. Given the prevalence of both cardiovascular and biliary diseases, clinicians must remain vigilant for non-cardiac causes of chest pain, particularly in patients with known gallstone disease and inconclusive cardiac findings. Early recognition can prevent unnecessary invasive procedures and lead to timely cholecystectomy.

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REFERENCE

1. Reed GW, Rossi JE, Cannon CP. Acute myocardial infarction. Lancet. 2017;389(10065):197–210. [PubMed]
2. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction (2018). Eur Heart J. 2019;40(3):237–269. [PubMed]
3. Gulati M, Levy PD, Mukherjee D, et al. 2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR guideline for the evaluation and diagnosis of chest pain: a report of the American College of Cardiology/American Heart Association joint committee on clinical practice guidelines. Circulation. 2021;144(22):E368–E454. [DOI]
4. Abbafati C, Abbas KM, Abbasi M, et al. Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020;396(10258):1204–1222. doi: [DOI]
5. Patel N, Ariyarathenam A, Davies W, Harris A. Acute cholecystits leading to ischemic ECG changes in a patient with no underlying cardiac disease. JSLS. 2011;15(1):105–108.[DOI]
6. D'Alessandro AD, Smith AT. ST elevation in a patient with abdominal pain. Ann Emerg Med. 2019;73(6):624–626. [DOI] [PubMed] [Google Scholar]
7. Hodge GB, Messer AL, Hill H, Durham M. Effect of the distention of the biliary tract on the electrocardiogram. Arch Surg. 1947;55:710. [DOI] [PubMed] [Google Scholar]
8. Sethi P, Murtaza G, Sharma A, Paul T. ST segment elevation with normal coronaries. Case Rep Med. 2016;2016:1–4. doi: 10.1155/2016/3132654 [DOI] [PMC free article] [PubMed] [Google Scholar]
9. Nasir JM, Durning SJ, Sweet JM, Cation LJ. Chest pain and ST segment elevation attributable to cholecystitis: a case report and review of the literature. Mil Med. 2006;171(12):1255–1258. [DOI] [PubMed] [Google Scholar]
10. Lanza GA, Careri G, Crea F. Mechanisms of coronary artery spasm. Circulation. 2011;124(16):1774–1782. [DOI] [PubMed] [Google Scholar]

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