Article:
Pourfridoni, M., Khan, M.A.B., Khalil-Khan, A. et al. (2 more authors) (2022) Elevated troponin level and nonspecific ST-segment and T-wave changes in a suspected acute pancreatitis patient, post-SARS-Cov-2 infection: a case report. Clinical Case Reports, 10 (11). e6628. ISSN 2050-0904

https://doi.org/10.1002/ccr3.6628

Received: 14 September 2022 | Revised: 28 October 2022 | Accepted: 8 November 2022

DOI: 10.1002/ccr3.6628

Clinical Case Reports Open Access WILEY

CASE REPORT

Elevated troponin level and nonspecific ST-segment and T-wave changes in a suspected acute pancreatitis patient, post-SARS-Cov-2 infection: A case report

Mohammad Pourfridoni[1] | **Moien A. B. Khan**[2,3] | **Alam Khalil-Khan**[4] | **Afshin Mohammad Bagheri Rafsanjani**[5] | **Hedyeh Askarpour**[5]

[1]Student Research Committee, Jiroft University of Medical Sciences, Jiroft, Iran

[2]Health and Wellness Research Group, Department of Family Medicine, College of Medicine and Health Sciences, United Arab Emirates University, Al-Ain, United Arab Emirates

[3]Primary Care, NHS Northwest London, London, Sheffield, UK

[4]Department of Academic Unit of Primary Medical Care, The University of Sheffield, Sheffield, UK

[5]Clinical Research Development Center of Imam Khomeini Hospital, Jiroft University of Medical Sciences, Jiroft, Iran

Correspondence
Hedyeh Askarpour, Clinical Research Development Center of Imam Khomeini Hospital, Jiroft University of Medical Sciences, Jiroft, Iran.
Email: hedyehaskarpour94@gmail.com

Abstract

The case report describes a post-COVID-19 patient with severe right upper quadrant (RUQ) pain, moderate epigastric pain, high troponin levels, and nonspecific ST-segment and T-wave changes on electrocardiogram (ECG).

KEYWORDS

abdominal pain, acute pancreatitis, case report, COVID-19, electrocardiography, SARS-Cov-2, troponin

1 | INTRODUCTION

Acute pancreatitis (AP) is characterized by immediate necroinflammatory alterations of the pancreas. The condition is reported to be the most common gastrointestinal diagnosis, requiring prompt hospitalization, and ranks 21 in terms of overall prevalence. There is an equal affinity for AP among both sexes with an incidence ranging from 13 to 45 cases per 100,000.[1] As per the Revised Atlanta Criteria of 2012 (updated from 1992), two of three conditions must be met in order to diagnose AP. The following conditions may be present: Abdominal pain consistent with AP (e.g., epigastric abdominal pain radiating to the back), lipase or amylase levels that are greater than three times the upper limit of normal, and/or 3. CT, MRI, and ultrasound feature characteristic of AP.[2]

It can be challenging to make a clinical diagnosis of pancreatitis based on symptoms, such as chest pain, positive troponin, and nonspecific ST segments on the electrocardiogram (ECG). The primary cause of elevated

troponin levels without acute coronary syndrome (acute or chronic) is myocyte necrosis, which results from a mismatch between oxygen supply and demand. There are several factors that cause troponin to rise, including tachycardia, tachycardia infusion, and tachycardia release; strenuous activity; autonomic nervous system imbalance; and conditions directly affecting membrane permeability.[3,4]

In the COVIDPAN study, people with COVID-19 had more severe AP than those without.[5] There have been several reports of pancreatic involvement in COVID-19. Other viruses with a well-known ability to infect and inflame the pancreas support the hypothesis that COVID-19 may contribute to the rise in acute idiopathic pancreatitis diagnoses.[6]

In this report, we discuss an individual who presented with symptoms of AP 2 weeks after being infected with SARS-Cov-2 and who had an elevated troponin level along with non-specific ST-segment and T-wave changes within their ECG, as well as symptoms of AP.

2 | CASE PRESENTATION

A 62-year-old male patient, presented to the emergency department (ED) with severe abdominal pain in the right upper quadrant (RUQ) region, moderate epigastric pain, and chest pain. On entering the Emergency Department, the patient had 130/80 mmHg blood pressure, 80 pulse rate, 20 respiratory rate, 37°C air temperature, and 99% oxygen saturation.

A consultation with an internal medicine specialist was requested for the patient after admission to the general surgical ward. As soon as the patient became infected with COVID-19, he reported mild pain in the RUQ area, but within 3 days, this became severe, leaving him unable to conduct daily activities. Moreover, the patient reported that his pain worsened when eating, and the pain did not radiate.

Despite adjusting his position, there was no difference in the intensity of the patient's pain. As the patient's chest pain did not resemble typical cardiac pain, it was considered not to be specific chest pain. It was accompanied by nausea and vomiting. He did not have a history of drinking or abusing alcohol, nor did he have a history of trauma. The patient had no underlying disease, and her mother and sisters had cholecystectomy surgery.

2.1 | Investigations

On physical examination, Jugular Venous Pulse was raised but no obvious edema was noted. During abdominal percussion, there was no tympanic percussion, and the liver span was 14.5 cm at the midclavicular line. His ECG showed bradyarrhythmia and inverted T wave in lead V1- and ST-segment elevation in V2, V3, V4, V5, and V6 (Figure 1). The chest and abdomen, X-rays did not demonstrate pneumonia (Figure 2), nor the presence of air common bile duct (CBD) (9 cm), and also gallbladder has sludge. The patient's echocardiography showed a trivial mitral regurgitation (MR) and reported the patient's

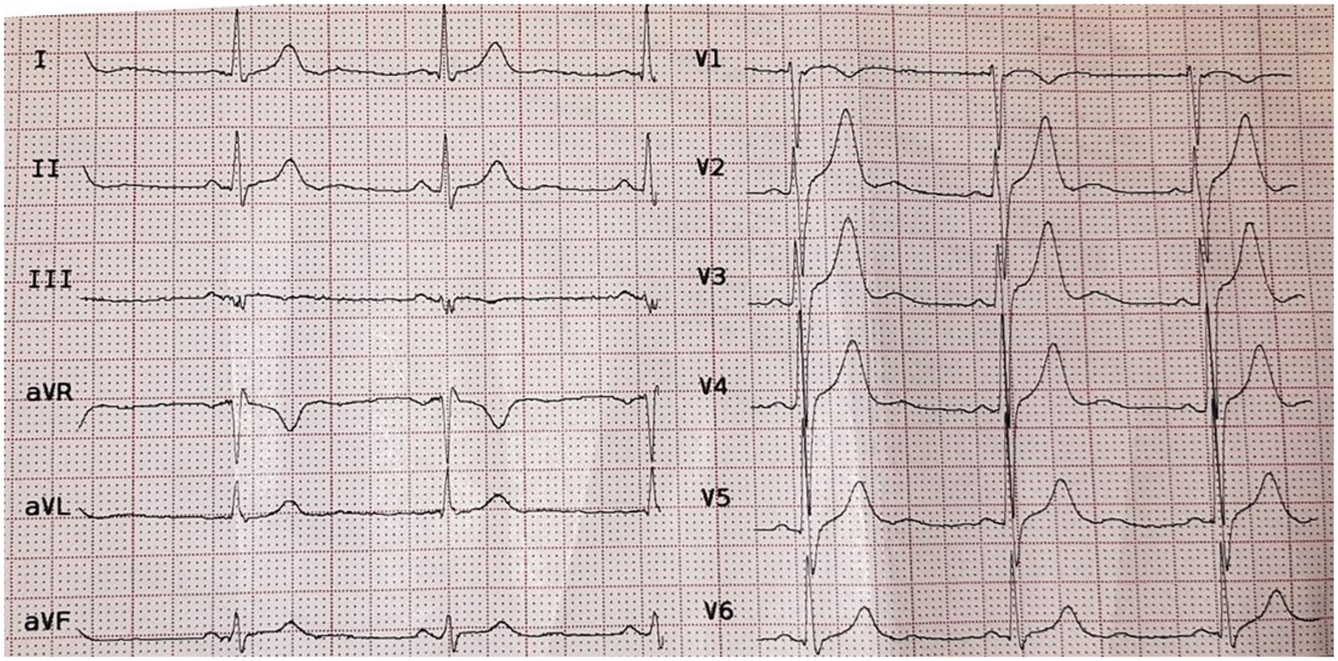

FIGURE 1 ECG: Bradyarrhythmia and inverted T wave in lead V1- and ST-segment elevation in leads V2, V3, V4, V5, and V6

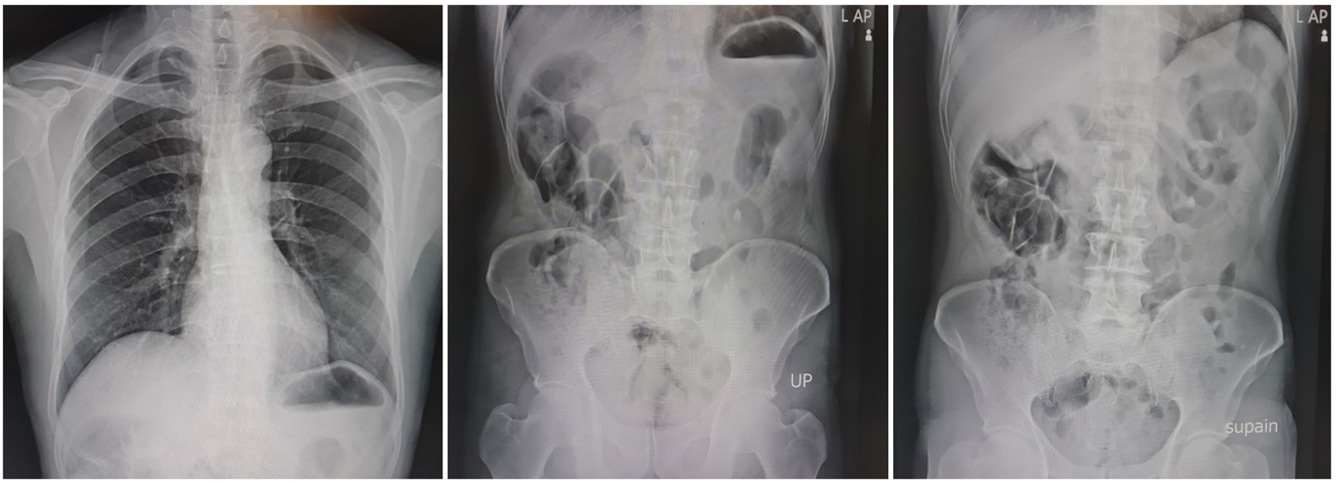

FIGURE 2 Chest X-ray, erect position (left): normal chest X-ray, no signs of pneumonia, air-under diaphragm, or pleural effusion. Abdominal X-ray erect position (middle): normal bowel gas pattern. No evidence of bowel wall thickening. Abdominal X-ray supine position (right): normal bowel gas pattern. No evidence of bowel wall thickening

ejection fraction (EF) to be 55%. His laboratory reports showed a highly elevated high sensitivity (hs)-troponin (2 times), amylase, lipase, AST, ALT, and a slight increase in blood sugar (BS), bilirubin total, bilirubin direct, alkaline phosphatase, and urea (Table 1).

2.2 | Differential diagnosis

There was severe abdominal pain in the right upper quadrant (RUQ) region, mild epigastric pain, and chest pain when the patient presented to the ED. The patient had COVID-19 14 days prior to the onset of these new symptoms, but no fever or dyspnea was observed in the ED, and no evidence of lung infection was found in the chest X-ray. He had an abnormal ECG, as well as two separate readings (0 and 6 h) of highly elevated hs-troponin, raising the likelihood of acute coronary syndrome diagnosis. The echocardiography shows trivial MR. ST-segment changes in ECG are non-specific (they do not have coved-type or reciprocal changes), thus myocardial injury is excluded. Meanwhile, the ultrasound abdomen showed dilated CBD, and the lab results showed high levels of bilirubin, AST, ALT, amylase, and lipase. Hence, biliary pancreatitis was considered the most probable diagnosis based on the evidence.

TABLE 1 Laboratory investigation results, before surgery

Test	Result	Normal range
hs-Troponin 0 h	1547.6 pg/ml	≥100: positive
hs-Troponin 6 h	1342.8 pg/ml	≥100: positive
Amylase	2420 U/L	<100
Lipase	987 U/L	≤60
AST	800 IU/L	<37
ALT	760 U/L	<41
Alkaline phosphatase	361 U/L	80–306
BS (RANDOM)	203 mg/dl	–
Bilirubin total	3.27 mg/dl	0.1–1.2
Bilirubin direct	2.37 mg/dl	Up to 0.3
Urea	68 mg/dl	20–45

TABLE 2 Laboratory investigation results after surgery

Test	Result	Normal range
Amylase	40 U/L	<100
Lipase	44 U/L	=<60
AST	60 IU/L	<37
ALT	270 U/L	<41
Alkaline phosphatase	300 U/L	80–306
Bilirubin total	0.7 mg/dl	0.1–1.2
Bilirubin direct	0.19 mg/dl	Up to 0.3
Urea	31 mg/dl	20–45

2.3 | Treatment

The patient was admitted to the general surgical ward and received IV fluids and analgesics. As the patient had a raised JVP, trivial mitral regurgitation, and bradyarrhythmia, underlying cardiac disease was suspected. The IV fluids were administered to the patient cautiously, gradually, and in limited quantities, and carefully monitored for fluid balance. A cholecystectomy was performed on the patient without any post-operative complications. After being deemed medically fit for discharge, the patient was discharged home. Table 2 below summarizes laboratory results after surgery.

2.4 | Outcome and follow-up

The patient improved after acute management and post-surgery, and the pain gradually subsided. In addition to being discharged, he was instructed to attend outpatient clinics for general surgery and gastroenterology. Additionally, the patient was suggested to visit a cardiology outpatient clinic so that his cardiovascular health could be monitored further.

3 | DISCUSSION

The contractile system in skeletal and cardiac myocytes is composed of troponin, a protein found within the heart. Calcium ions and troponin proteins regulate and facilitate actin and myosin filament contact during muscle contraction. There are three subunits that make up cardiac troponin (cTn).[7] Cardiovascular troponin (cTn) assays are used to optimize the screening of patients with acute coronary syndromes (ACS).[8] The increase in troponin is a sign of myocardial cell injury, which can be caused by trauma, inflammation, ischemia, tachycardia, strenuous exercise, catecholamine infusions and releases, autonomic nervous system imbalances, and conditions that affect membrane permeability directly. Troponin levels may also be affected by chest contusion, toxins, systemic infections, diseases involving electricity infiltration, or renal failure.[9] Within a clinical setting, increased troponin levels have been observed even in the absence of acute myocardial injury.

Several animal studies have demonstrated that pancreatic enzymes can reach the bloodstream and damage the myocardium. In addition, AP can also cause permeability changes in myocardial cell membranes or coronary vasospasm. Cardiac infarction may result from pancreatitis-related microvascular dysfunction.[3]

Electrocardiogram changes and biomarkers of myocardial injury have been observed during AP in some studies.[10,11] The literature extensively documents transient electrocardiographic changes in individuals with acute pancreatitis. According to Bulava et al. study, T-wave inversion, ST-segment depression, and ST-segment elevation represent most of these changes.[12]

COVID-19 is associated with high mortality and morbidity rates, as well as several systemic inflammatory reactions. Patients with gastrointestinal problems progress more quickly. The symptoms of acute pancreatitis caused by COVID-19 are uncommon and can develop even after the viral infection has resolved.[13] Even after recovery, COVID-19 can cause changes in cardiac biomarkers and ECG. In some people, elevated troponin levels in the blood are accompanied by changes in the ECG.[14,15]

The case discussed in this study is notable because the patient developed AP symptoms after recovering from the COVID-19 infection. Additionally, the patient's cardiac biomarkers and non-specific ECG patterns have changed, possibly caused by pancreatitis or COVID-19 complications.

4 | CONCLUSIONS

Troponin release and ECG changes in AP and COVID-19 infections are not fully understood. According to the current case report, cardiac biomarkers and ECG may change following acute pancreatitis or COVID-19 infection. Through further research, these findings may be used to diagnose acute pancreatitis or post-COVID-19 gastrointestinal complications earlier. It should be noted that, if cardiac biomarkers or ECGs change, not all cases may be associated with pancreatitis or COVID-19. Following recovery from acute coronary artery disease, imaging or further treatment may be required. Future studies in this field are highly recommended as cardiac biomarkers and ECG may change in patients with specific underlying diseases or genetic variations.

AUTHOR CONTRIBUTIONS

Mohammad Pourfridoni: Conceptualized the study, wrote, revised, and edited the manuscript. Moien A. B. Khan: Revised, and edited the manuscript. Alam Khalil-Khan: Analyzed and interpreted the ECG and Chest X-ray and revised, and edited the manuscript. Afshin Mohammad Bagheri Rafsanjani: Analyzed and interpreted the ECG and Chest X-ray and revised, and edited the manuscript. Hedyeh Askarpour: Conceptualized and revised the manuscript. All authors were involved in editing and approving the manuscript.

ACKNOWLEDGMENTS

The authors are grateful to our patient who allowed his story to be shared, and also Dr. Shoeleh Khajoei, chair of the Department of Internal Medicine, Imam Khomeini Hospital, Jiroft, for her valuable support.

CONFLICT OF INTEREST

No conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICAL APPROVAL

This case report was carried out following the recommendations of the Jiroft University of Medical Sciences ethics committee.

CONSENT

Written informed consent was obtained from the patient for the publication of this case report in accordance with the journal's patient consent policy.

ORCID

Mohammad Pourfridoni https://orcid.org/0000-0002-0510-3194

REFERENCES

1. Garber A, Frakes C, Arora Z, Chahal P. Mechanisms and management of acute pancreatitis. *Gastroenterol Res Pract*. 2018;2018:6218798.
2. Quinlan JD. Acute pancreatitis. *Am Fam Physician*. 2014;90(9):632-639.
3. Zaki HA, Shaban EE, Shaban AE, Elmoheen A. High troponin-T in acute biliary pancreatitis: is it a real myocardial injury? *Cureus*. 2021;13:e18637.
4. Jeremias A, Gibson CM. Narrative review: alternative causes for elevated cardiac troponin levels when acute coronary syndromes are excluded. *Ann Intern Med*. 2005;142(9):786-791.
5. de-Madaria E, Capurso G. COVID-19 and acute pancreatitis: examining the causality. *Nat Rev Gastroenterol Hepatol [Internet]*. 2021;18(1):3-4. doi:10.1038/s41575-020-00389-y
6. Balthazar JA, Chehter EZ. Acute pancreatitis and COVID-19: a new target for infection? *Einstein (Sao Paulo)*. 2022;20:eRW6667.
7. Garg P, Morris P, Fazlanie AL, et al. Cardiac biomarkers of acute coronary syndrome: from history to high-sensitivity cardiac troponin. *Intern Emerg Med*. 2017;12(2):147-155.
8. Vasile VC, Jaffe AS. High-sensitivity cardiac troponin for the diagnosis of patients with acute coronary syndromes. *Curr Cardiol Rep*. 2017;19(10):92.
9. Aundhakar SC, Mahajan SK, Agarwal AO, Mhaskar DM. Acute pancreatitis associated with elevated troponin levels: whether to thrombolyse or not? *Ann Med Health Sci Res*. 2013;3(1a):50-52.
10. Elliot SY, Lange JJ, Broor A, Kutty K. Acute pancreatitis masquerading as inferior wall myocardial infarction: a review. *Case Rep Gastroenterol*. 2019;13(2):321-335.
11. Long Y, Tang M, Wang J, et al. Case report: abnormal ECG in a patient with acute pancreatitis. *Front Cardiovasc Med*. 2021;8:741253.
12. Bulava A, Skvarilová M, Marek O, Lukl J. Electrocardiographic changes in patients with acute pancreatitis. Case report and review of the literature. *Vnitr Lek*. 2001;47(6):407-410.
13. Almutairi F, Rabeie N, Awais A, et al. COVID-19 induced acute pancreatitis after resolution of the infection. *J Infect Public Health [Internet]*. 2022;15(3):282-284.
14. Rivera-Morales MD, Pell R, Rubero J, Ganti L. Acute myopericarditis in the post COVID-19 recovery phase. *Cureus*. 2020;12(10):e11247.
15. Nedeljkovic IP, Giga V, Ostojic M, et al. Focal myocarditis after mild COVID-19 infection in athletes. *Diagnostics*. 2021;11(8):1519.

How to cite this article: Pourfridoni M, Khan MAB, Khalil-Khan A, Mohammad Bagheri Rafsanjani A, Askarpour H. Elevated troponin level and nonspecific ST-segment and T-wave changes in a suspected acute pancreatitis patient, post-SARS-Cov-2 infection: A case report. *Clin Case Rep*. 2022;10:e06628. doi: 10.1002/ccr3.6628