Received: 10 October 2021 | Revised: 13 November 2021 | Accepted: 22 December 2021

DOI: 10.1002/ccr3.5309

CASE REPORT

Clinical Case Reports Open Access WILEY

# Elevation of creatine kinase in acute pancreatitis: A case report

**Mehdi Sheibani**[1,2] | **Bahareh Hajibaratali**[3] | **Houra Yeganegi**[2]

[1]Cardiovascular Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran

[2]Clinical Research Development Center of Loghman Hakim Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran

[3]Department of Cardiology, Shahid Labbafi Nejad Medical Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran

**Correspondence**
Bahareh Hajibaratali, Shahid Beheshti University of Medical Sciences, Labbafi Nejad Hospital, Tehran, Iran.
Email: baharehbarati@yahoo.com

**Abstract**

Creatine kinase (CK/CK-MB) testing is an essential laboratory test approaching a patient with chest or epigastric pain. We report a 38-year-old man with acute pancreatitis and elevated CK/CK-MB level without myocardial involvement. Acute pancreatitis may be considered as a false-positive cause of CK/CK-MB test in patients presenting with chest pain.

**KEYWORDS**

CK-MB, creatine kinase, pancreatitis

## 1 | INTRODUCTION

Measuring creatine kinase and creatine kinase-myoglobin binding (CK and CK-MB) are an integral part of approaching a patient presenting with ischemic-related symptoms. In addition, CK is an enzyme found in high concentration in skeletal muscle and low concentration in the brain.[1] Therefore, myocardial damage, myopathies, some endocrine disorders with muscle involvement, and some medications may increase CK. However, acute pancreatitis is a rare cause of CK elevation. Elevated CK may be seen in a severe form of acute pancreatitis with the evidence of hypotension and myocardial involvement.[2] Recently, we observed a case of elevated serum CK/CK-MB in a patient with non-severe acute pancreatitis, which case is now presented.

## 2 | CASE PRESENTATION

A 38-year-old man presented to the emergency department with acute onset of epigastric and retrosternal pain that had awakened him in the middle of the night. The pain radiated through his back continuously and was associated with nausea and 3-h vomiting. The patient suffered from chest heaviness while he did not experience any dyspnea. The pain was not positional. Physical examination revealed hemodynamically stable vital signs. His blood pressure was 110/90, pulse rate was 110/min, and temperature was 37°C. Abdominal examination showed mild epigastric tenderness with no rebound. The abdomen was not distended. Cardiovascular and respiratory examinations were otherwise normal. His past medical history revealed he had alopecia areata and well-controlled hypothyroidism

with a recent TSH = 1.5. He was a nonalcoholic individual. Since he had retrosternal and epigastric pain, a 12-lead electrocardiogram (ECG) was obtained that revealed sinus tachycardia with nonspecific ST-T changes. The abnormal laboratory findings were CK 878 (normal 24–195 IU/L) and CK-MB fraction 121(normal 0–25 IU/L). Troponin I with 0.001(normal 0-ng/ml) and pro-BNP 22 (normal <125 pg/ml) were normal. Other laboratory tests are showed in Table 1. The patient was admitted to the chest pain unit due to high CK-MB and underwent cardiac monitoring. Serial ECG analysis did not show any evidence of myocardial infarction or active ischemia. We did not start treatment since all data were inconsistent with the acute coronary syndrome. After being thoroughly examined in the chest pain unit, he complained about localized, more severe epigastric pain. Therefore, amylase and lipase were tested with 236 (normal 40–140 U/L) and 42 (normal 24–151 U/L), respectively. His chest and abdominal radiograph were normal. An abdominal ultrasound showed hypoechoic extra-pancreatic inflammation, and computed tomography scan (CT scan) revealed interstitial edema without necrosis, hemorrhage, or abscess in the pancreas (Figure 1A,B). The patient was diagnosed with acute pancreatitis, and treatment was immediately started

**TABLE 1** Patient's laboratory test results

| Laboratory | Result | Reference range |
|---|---|---|
| Hb | 17.9 | 13.5–18 mg/L |
| WBC | 11400 | 4–10.1 μl/$10^3$ |
| PLT | 204000 | 150,000-450,000/μl/$3^{\Delta}10$ |
| BUN | 18 | 7–20 mg/dl |
| Cr | 1.1 | 0.7–1.4mg/dl |
| Na | 138 | 134–148 mEq/L |
| K | 4.5 | 3.5–5.3 mEq/L |
| Blood glucose | 140 | <140 mg/dl |
| ALT | 46 | <37 IU/L |
| AST | 23 | <41 IU/L |
| Bilirubin (total) | 0.7 | 0.1–1.2 mg/dl |
| Bilirubin (direct) | 0.2 | <0.3 mg/dl |
| Ca | 8 | 8.6–10.3 mg/dl |
| Albumin | 3.8 | 3.4– 5.4 g/L |
| LDH | 424 | 140–280 U/L |
| LDL | 186 | <100 mg/dl |
| HDL | 30 | desirable >60 mg/dl |
| TG | 1074 | <150 mg/dl |
| CK | 878 | 24–195 IU/L |
| CK-MB | 121 | 0–25 IU/L |
| Troponin I | 0.001 | 0–0.01 ng/ml |
| Pro-BNP | 22 | <125 pg/ml |

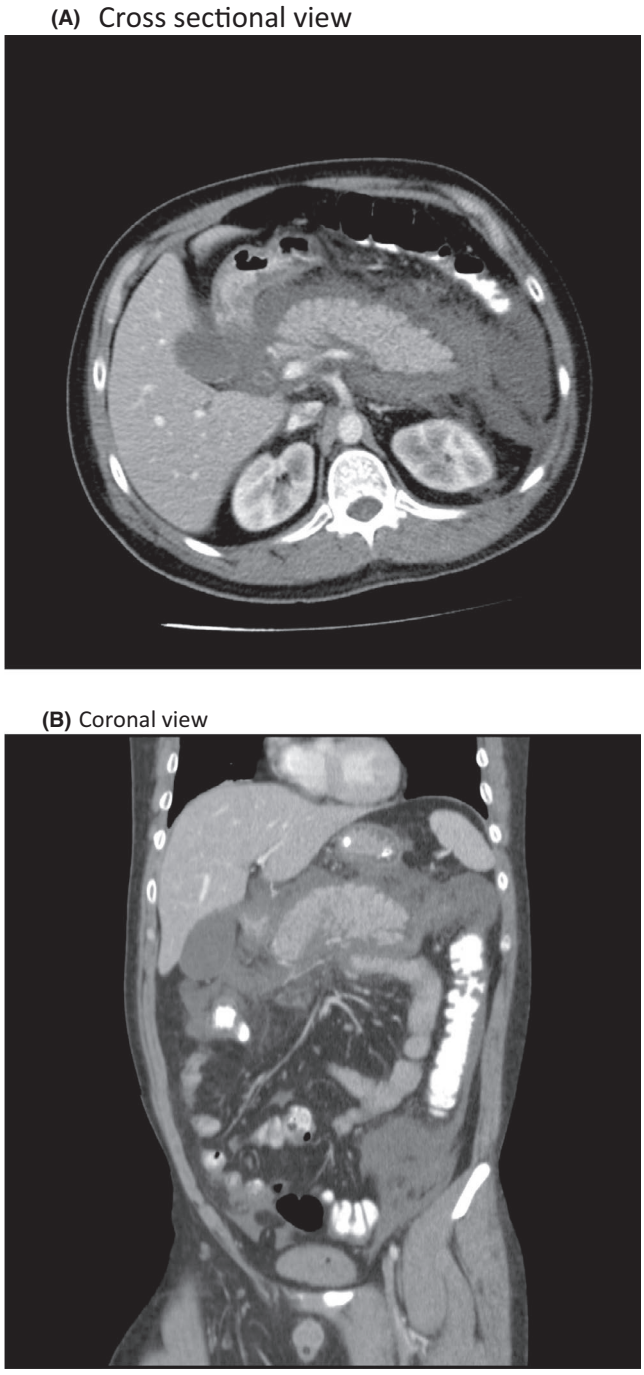


**FIGURE 1** Abdominal CT scan findings revealed interstitial edema without necrosis, hemorrhage, or abscess in the pancreas (a. cross-sectional view and b. coronal view)

with intravenous fluids, nutritional support, and supportive care. He was not allowed to eat or drink anything for 5 days. The abdominal pain persisted for 3 days. On the fourth hospital day, he had no more abdominal pain, and his amylase and lipase levels were 108 and 75 U/L, respectively. The CK/CK-MB activity peaked the day after admission, with CK = 1065 and CK-MB = 151 (almost 14% of total CK) but returned to normal after 5 days. He was discharged 12 days later.

## 3 | DISCUSSION AND CONCLUSION

Pain is the hallmark of acute pancreatitis, and it may mimic acute coronary syndrome (ACS). [3] In addition, serial CK /CK-MB testing is considered a reasonable approach to determine the probability of a cardiac cause. In the mentioned case, the patient was first admitted as a probable acute ischemia due to retrosternal and epigastric pain, elevated CK/CK-MB as well as nonspecific ST-T changes in ECG. Within a few hours as the patient's symptoms became more focused on epigastric pain and positive amylase and lipase levels were noted on the laboratories, the diagnosis of acute pancreatitis seemed more likely. There are several non-cardiac causes of CK elevation, such as exercise or heavy manual labor, which can increase the enzyme even up to 30 times the upper limit of normal. Statins can develop CK elevation up to 2–10 times upper limit of normal, endocrine disorders such as hypothyroidism and rarely hyperthyroidism, acute alcohol ingestion or alcohol myopathy, muscular dystrophy, polymyositis, stroke, trauma, convulsions, and prolonged immobilization as well as surgery, cardiac catheterization, or electric cardioversion are other possible causes.[4] However, none of the above causes were related to our patient. In this case, although CK/CK-MB was significantly elevated, other specific myocardial/cardiac markers such as Cardiac Troponin I and Pro-BNP were in the normal range. Therefore, myocardial injury unlikely caused the elevation of the CK/CK-MB. On the contrary, Zao et al[2]. reported elevated CK activity that may rarely occur in patients with acute pancreatitis peaked 1 day after admission. Their study also revealed the cardiac dysfunction can happen in the early stage of acute pancreatitis and is usually demonstrated by echocardiography (cardiac regional wall motion abnormalities and impaired diastolic function) and electrocardiography (T-wave flattering and ST-segment depression).[2] Although our patient had some nonspecific ST-T change in ECG, echocardiography showed no systolic or diastolic dysfunction and no regional wall motion abnormality. Randeva et al.[5] showed that the CK-MB was less than 5% of total CK activity, and in myocardial involvement increased over time that peaks 24 h later, while in extracardiac elevation of MB isoenzyme a plateau pattern may be more common. In the present case report, unexpectedly, the ratio of CK-MB to total CK was approximately 14% and it was increased and peaked 24 h after admission. Liu and Bigler[6] also reported a case of acute pancreatitis which all three forms of CK isoenzyme (CK-MM, CK-MB, CK-BB) were significantly increased without any cardiac, brain, or any organ involvement other than pancreas. Tsung[7] showed that CK-BB encompasses almost 93% of the total CK activity in the pancreas and pancreatic necrosis can increase this enzyme and subsequently total CK. Although CK-BB is not included in the routine laboratory tests these days, it might have been the main culprit that causes MB isoenzyme elevation since both assays are based on B subunit of CK.[1]

However, some studies reported almost 75% myocardial involvement may occur in acute pancreatitis.[3,8] To explain cardiac involvement in acute pancreatitis, large amounts of inflammatory cytokines such as tumor necrosis factor-alpha, interleukin-1 beta, and endotoxin may be involved.[9,10] Furthermore, reactive oxygen species (ROS) causing cardiomyocyte apoptosis via activating the mitogen-activated protein kinase (MAPK) pathway or nicotinamide adenine dinucleotide phosphate (NADPH) oxidase hyperactivity,[11] or interleukin-1 release should be considered.[12] Cardiac ischemic events caused by severe hypotension, coagulation abnormality, and coronary artery spasm (often induced by electrolyte alteration, such as hypomagnesemia) might play a secondary role.[13] All mentioned explanations are related to cardiac involvement and elevated cardiac biomarkers and symptoms compatible with myocardial injury. Zao et al.[2] reported that the level of CK-MB showed a predictive ability of severe acute pancreatitis occurrence while cardiac troponin and pro-BNP did not. Mainly CK-MB was considered an independent determinant of organ failure in acute pancreatitis, which may lead to severe acute pancreatitis. However, according to the 2012 revised Atlanta guideline,[14] our patient did not meet the criteria of severe acute pancreatitis. Despite Zao et al.,[2] the results in other study[13] did not show the ability of CK to predict severe acute pancreatitis and CK/CK-MB was considered as a myocyte as well as muscle involvement in the course of the disease.

In conclusion, acute pancreatitis may cause the elevation of CK/CK-MB without any evidence of myocardial involvement, whereas, rising CK/CK-MB may also be a predictor of severe acute pancreatitis.

### ACKNOWLEDGEMENT

The authors would like to express their appreciation from the Physicians, nurses, and staff of Loghman Hakim Hospital, Tehran, Iran.

### CONFLICT OF INTEREST

None declared.

### AUTHOR CONTRIBUTIONS

Mehdi Sheibani, Bahareh Hajibaratali, and Houra Yeganegi performed material preparation and data collection. Bahareh Hajibaratali wrote the first draft of the manuscript. Mehdi Sheibani and Houra Yeganegi revised the manuscript carefully. All authors contributed to this case report, and read and approved the final manuscript.

## CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

## DATA AVAILABILITY STATEMENT

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

## ORCID

*Bahareh Hajibaratali* https://orcid.org/0000-0002-2138-2007

## REFERENCES

1. Karachaliou I, Papadopoulou K, Karachalios G, Charalabopoulos A, Papalimneou V, Charalabopoulos K. An increase in creatine kinase secondary to acute pancreatitis: a case report. *Int J Clin Pract Suppl*. 2005;147:40-42. doi:10.1111/j.1368-504x.2005.00337.x. PMID: 15875618.
2. Zhao B, Sun S, Wang Y, et al. Cardiac indicator CK-MB might be a predictive marker for severity and organ failure development of acute pancreatitis. *Ann Transl Med*. 2021;9(5):368. doi:10.21037/atm-20-3095. PMID: 33842589; PMCID: PMC8033390.
3. Barassi A, Pezzilli R, Romanelli MC, Banfi G, Merlini G, d'Eril GM. Serum markers of myocardial damage in acute pancreatitis: a prospective time course study. *Pancreas*. 2015;44(4):678-680. doi:10.1097/MPA.0000000000000320
4. Moghadam-Kia S, Oddis CV, Aggarwal R. Approach to asymptomatic creatine kinase elevation. *Cleve Clin J Med*. 2016;83(1):37-42. doi:10.3949/ccjm.83a.14120. PMID: 26760521; PMCID: PMC4871266.
5. Randeva HS, Bolodeoku J, Mikhailidis DP, Winder AD, Press M. Elevated serum creatine kinase activity in a patient with acute pancreatitis. *Int J Clin Pract*. 1999;53(6):482-483. PMID: 10622080.
6. Liu TZ, Bigler WN. Unusual creatine kinase isoenzyme pattern association with a case of acute pancreatitis. *Clin Chem*. 1983;29:579-580.
7. Tsung SH. Creatine kinase isoenzyme patterns in human tissue obtained at surgery. *Clin Chem*. 1976;22:173-175.
8. Thandassery RB, Choudhary N, Bahl A, et al. Characterization of cardiac dysfunction by echocardiography in early severe acute pancreatitis. *Pancreas*. 2017;46:626-630.
9. Jambrik Z, Gyöngyösi M, Hegyi P, et al. Plasma levels of IL-6 correlate with hemodynamic abnormalities in acute pancreatitis in rabbits. *Intensive Care Med*. 2002;28:1810-1818.
10. Meyer A, Kubrusly MS, Salemi VM, et al. Severe acute pancreatitis: a possible role of intramyocardial cytokine production. *JOP*. 2014;15:237-242.
11. Wen Y, Liu R, Lin N, et al. NADPH oxidase hyperactivity contributes to cardiac dysfunction and apoptosis in rats with severe experimental pancreatitis through ROS-mediated MAPK signaling pathway. *Oxid Med Cell Longev*. 2019;2019:4578175.
12. Li L, Sun Z, Xu C, et al. Adenovirus-mediated overexpression of sST2 attenuates cardiac injury in the rat with severe acute pancreatitis. *Life Sci*. 2018;202:167-174.
13. Prasada R, Dhaka N, Bahl A, et al. Prevalence of cardiovascular dysfunction and its association with outcome in patients with acute pancreatitis. *Indian J Gastroenterol*. 2018;37:113-119.
14. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis–2012: revision of the Atlanta classification and definitions by international consensus. *Gut*. 2013;62(1):102-111. doi:10.1136/gutjnl-2012-302779. PMID: 23100216.

**How to cite this article:** Sheibani M, Hajibaratali B, Yeganegi H. Elevation of creatine kinase in acute pancreatitis: A case report. *Clin Case Rep*. 2022;10:e05309. doi:10.1002/ccr3.5309