

# Case of Anterior Myocardial Infarction Presented with Urosepsis

**Emre Paçacı\*, Burak Cabbar Karakurt and Gülhan Yüksel**

Adana City Training and Research Hospital, Turkey

\*Corresponding author: Emre Paçacı, Adana City Training and Research Hospital, Adana, Turkey

## ARTICLE INFO

**Received:** 📅 October 28, 2023

**Published:** 📅 November 07, 2023

**Citation:** Emre Paçacı, Burak Cabbar Karakurt and Gülhan Yüksel. Efficacy and Safety of Nalbuphine for Epidural Labor Analgesia at High Altitude: An Observational Study. Biomed J Sci & Tech Res 53(4)-2023. BJSTR. MS.ID.008432.

## ABSTRACT

High cardiac enzymes occur often during sepsis. Sepsis complicated by ST elevation myocardial infarction has been reported rarely, and the exact mechanisms of myocardial injury remain unclear. This case describes an 85 years old male patient presenting with septic shock secondary to urosepsis and klebsiella pneumonia. The patient was treated in ER. In ECG, at V2-V5 leads, ST elevation as well as ST segment depression in DI and aVL are observed. The patient was taken to the emergency catheter laboratory. However, there were no findings in terms of obstructive coronary disease. When the patient, who was hypotensive, was taken to the intensive care unit, norepinephrine was given and re-evaluated, cardiogenic shock seemed less likely and septic shock was considered. ST segment elevations and elevated cardiac markers in the anterior leads were probably due to transmural ischemia secondary to increased oxygen demand. The patient was started on intravenous antibiotic therapy for the treatment of septic shock and was weaned from norepinephrine within 72 hours. Cardiac dysfunction in septic shock is well established, but the mechanisms are not yet clear.

## Introduction

Elevated cardiac enzymes, particularly troponin I, are commonly observed in acute coronary syndromes but may also occur in sepsis. The mechanism of high troponin in sepsis mainly includes hypoperfusion but the mechanisms are not clearly established. ECG changes, however, are less commonly observed in septic shock. ECG changes observed in sepsis include prolonged QTc interval, bundle branch blocks, non-specific ST changes and quite rarely ST segment elevations [1]. Additionally, the mechanism of the ST segment elevation observed in sepsis is not fully understood. In this case, we wanted to share an acute anterior myocardial infarction presenting with urosepsis.

## Presentation of the Case

An 85 years old male patient with a known history of diabetes mellitus and recurrent urinary tract infection was admitted to the emergency services with complaints of nausea, vomiting, and general deteriorating condition. Through information provided by the relatives of the patient, it was learned that his complaints started 10 days

ago and he applied to the infectious diseases polyclinic. Urine culture were taken from and fully automatic urinalysis were performed on the patient in the outpatient clinic, and empirical oral antibiotics were given. Although the patient's complaints did not regress, he did not go to the check-up and came to the emergency room when his symptoms increased and his state of consciousness deteriorated. In the emergency room, his fever was 38.5°C, heart rate was 133, blood pressure was 70/40, respiratory rate was 20 per minute, and room air saturation was 92%. In the ECG, at V2-V5 leads, ST elevation and ST segment depression in DI and aVL are observed (Figure 1). The patient was taken to the emergency catheter laboratory. However, there were no findings in terms of obstructive coronary disease. The hypotensive patient was started on norepinephrine in the intensive care unit. After klebsiella pneumoniae growth was observed in the urine culture taken 10 days ago and a physical examination, the patient was diagnosed with sepsis. Immediately, blood and urine cultures were taken from the patient and Infectious Diseases was contacted. Based on the previous urine culture, the patient was started on meropenem treatment. Ejection fraction 35% apical aneurysm mild mitral regurgitation and mild-moderate pericardial effusion that did not cause compression in

the heart were observed in the echocardiogram. Intravenous antibiotic therapy was continued due to septic shock caused in relation to the klebsiella pneumoniae bacteremia. Norepinephrine was discontinued

72 hours after the treatment and his general condition improved. The patient was discharged after 14 days of antibiotherapy (Figure 2).

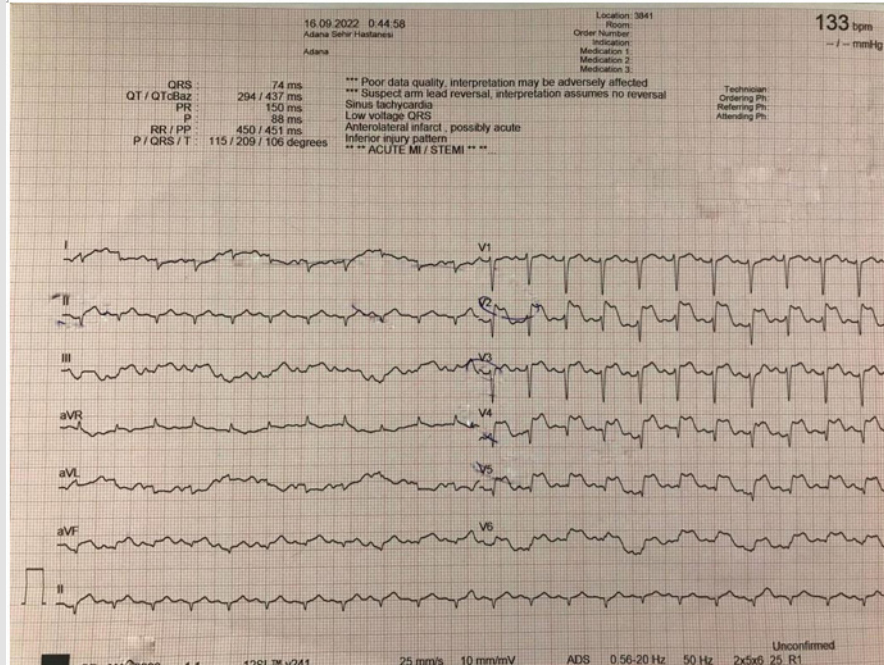


Figure 1,

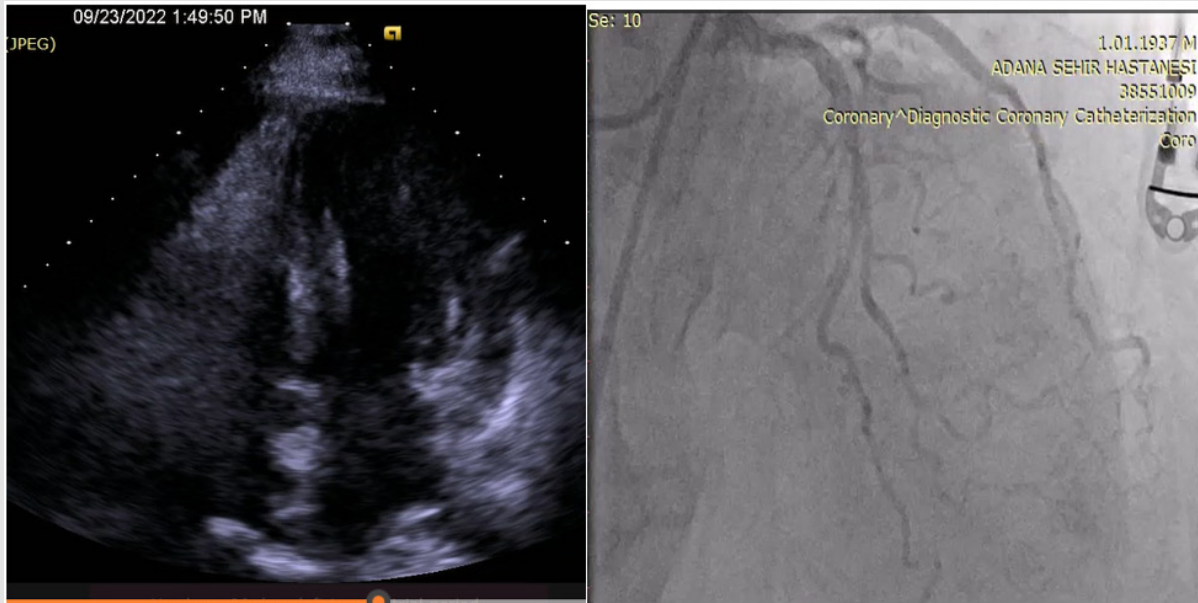


Figure 2,

## Discussion

Cardiogenic shock should be considered first in the differential diagnosis list in the presence of ST segment elevation, troponin elevation, and systemic hypotension. In this patient, in the early period echocardiogram performed for differential diagnosis, severe depressed left and/or right ventricular systolic function, high filling pressures and decreased stroke volume specific to cardiogenic shock were not observed, whereas an apical aneurysm and mild to moderate pericardial effusion were present. Echocardiographic studies show that 40-50% of patients with long-term septic shock develop myocardial depression [2]. The relationship between sepsis and acute myocardial infarction can be explained primarily in two ways. First, it may lead to myocardial hypoperfusion or type II MI due to the hemodynamic condition caused by ischemic inflammation [3]. Second, systemic inflammation has effects such as inhibition of fibrinolysis and down-regulation of anticoagulant pathways [4]. In this patient, ST segment elevation and elevated troponins in the anterior leads were due to severe transmural ischemia, possibly secondary to increased oxygen demand. Apart from ischemia, in the setting of septic shock other factors may have contributed to myocardial cell damage. The

patient completed intravenous antibiotic therapy for the treatment of septic shock secondary to urosepsis and klebsiella pneumonia bacteremia. The patient was prescribed cardiological treatments.

## Conclusion

Septic shock, by definition, entails end-organ damage. The presence of ST segment elevations in the anterior leads in this patient was most likely secondary to ischemia and increased oxygen demand. Coronary angiography was conducive to the diagnosis of this patient.

## References

1. Rich M M, Mike L McGarvey, James W Teener, Lawrence H Frame (2002) ECG changes during septic shock. *Cardiology* 97(4): 187-196.
2. Rudiger A, M Singer (2007) Mechanisms of sepsis-induced cardiac dysfunction. *Crit Care Med* 35(6): 1599-1608.
3. Thygesen K, Joseph S Alpert, Allan S Jaffe, Maarten L Simoons, Bernard R Chaitman, et al. (2012) Third universal definition of myocardial infarction. *J Am Coll Cardiol* 60(16): 1581-1598.
4. Taylor F B Jr (2001) Response of anticoagulant pathways in disseminated intravascular coagulation. *Semin Thromb Hemost*, 27(6): 619-631.

ISSN: 2574-1241

DOI: 10.26717/BJSTR.2023.53.008432

Emre Paçacı. Biomed J Sci & Tech Res



This work is licensed under Creative Commons Attribution 4.0 License

Submission Link: <https://biomedres.us/submit-manuscript.php>



### Assets of Publishing with us

- Global archiving of articles
- Immediate, unrestricted online access
- Rigorous Peer Review Process
- Authors Retain Copyrights
- Unique DOI for all articles

<https://biomedres.us/>