

CASE REPORT

Non-ST-segment Elevation Myocardial Infarction Associated with Multiple Comorbidities in a Patient with a Ventriculoperitoneal Shunt for Obstructive Hydrocephalus Following Traumatic Brain Injury

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ABSTRACT

Non-ST elevation myocardial infarction (NSTEMI) has the highest long-term mortality rates of all acute coronary syndromes. Usually, NSTEMI occurs in elderly patients (>75 years of age) with multiple associated diseases. The complication rate for NSTEMI, including heart failure or hemorrhages, is significantly higher than that in ST-elevation myocardial infarction patients. The case reported is of a 70 year-old male, with a history of ventriculoperitoneal shunt for obstructive hydrocephalus following a traumatic brain injury, who presented with NSTEMI.

Keywords: NSTEMI, multiple comorbidities, ventriculoperitoneal shunt

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INTRODUCTION

Acute myocardial infarction (MI) remains one of the leading causes of death worldwide.¹ Of all the types of acute coronary syndromes (ACS), ST-segment elevation myocardial infarction (STEMI) has the highest mortality rates during hospitalization, followed by non-ST elevation MI (NSTEMI) and unstable angina (UA). However, during the six-month follow-up period, the highest death rate is recorded among NSTEMI patients, followed by STEMI and UA.² According to the OPERA registry, one-year mortality rates are also higher for NSTEMI compared to STEMI patients (11.6% vs. 9%).³

The primary cause of the high mortality associated with NSTEMI may be due to the severity of the patients' clinical status. It is well-known that in a relatively high percentage, NSTEMI patients are significantly older, with various associated comorbidities, which in turn lead to an increased risk of cardiovascular events. Approximately one-third of patients presenting with NSTEMI are aged over 75 years, and this age factor has been proven to be one of the most significant risk predictors of NSTEMI, the incidence of complications increasing with age.⁴ Patients that present with non-ST elevation acute coronary syndromes frequently present with several comorbidities,

such as arterial hypertension, diabetes mellitus, chronic kidney disease, as well as atypical symptoms of ACS. They are also susceptible to more severe complications, such as acute heart failure and hemorrhages.⁴

Interestingly, NSTEMI patients have a less frequent rate of invasive procedures performed during hospitalization, probably due to the lower perception of high cardiovascular risk in these patients, the presence of multiple comorbidities, the advanced age or the refusal to undergo invasive treatment.^{5,6}

The case presented is of a 70-year-old male with non-ST-elevation acute coronary syndrome and multiple associated pathologies.

CASE PRESENTATION

A 70-year-old male, with a history of ventriculoperitoneal shunt (VP shunt) for obstructive hydrocephalus following a football injury in 1980, and post-traumatic seizures, presented in the Emergency Room (ER) with dizziness, confusion, sleepiness and slurred speech. Previously, the patient had multiple craniotomies for the VP shunt left hemiparesis and had been wheelchair-bound for several years. He had fallen from his bed, and was found by his wife. Hetero-anamnesis revealed that in the past the patient had a suicidal attempt by phenytoin overdose and that he suffers from chronic headaches. No recent seizures were reported.

The patient gave informed consent to the publication of this case report and its accompanying images.

Vital sign assessment in the ER showed a blood pressure (BP) of 157/84 mmHg, normal sinus rhythm, a temperature of 35.6 °C, respiratory rate – 18/min, SpO₂ – 100% in the room air, weight of 82.4 kg. The general clinical examination did not reveal any abnormal signs, with the exception of a bilateral lower extremity muscle atrophy, slurred speech, confusion, left hemiparesis and minimal movement in the left upper and lower extremities.

The initial ER laboratory evaluations are shown in Table 1.

In addition to the initial ER draw, a complete myocardial necrosis panel was requested:

- CPK (creatinine phosphokinase) – 600 (normal range: 55–400 U/L)
- CK-MB (CK myocardial and brain isoenzyme) – 6.8 (normal range: 0.0–3.8 ng/mL)
- Troponin I – 5.96 (normal range 0–0.08 ng/mL).

The electrocardiography (ECG) showed sinus rhythm, a normal P wave axis, a ventricular rate of 69 bpm, right bundle branch block (RBBB) and left posterior fascicular block (LPFB), baseline wander in leads V6 and a corrected QT interval of 533 milliseconds (Figure 1).

The initial assessment established the probable diagnosis of acute toxic encephalopathy due to phenytoin overdose, for which the cessation of phenytoin administration was required and serial neurological checks were requested together with continuation of stroke care pathway, as, at this point in time, a stroke could not be ruled out.

MRI examination was not possible due to the presence of the VP shunt, and a head CT was performed to rule out a stroke or a transient ischemic attack (TIA). CT results are shown in Figure 2.

A cardiology assessment was requested due to the presence of elevated myocardial necrosis enzymes and the ECG changes. Due to the presence of atypical chest pain, reassessment of cardiac enzymes and echocardiographic evaluation were recommended, and aspirin, 300 mg daily, was initiated together with 5000 units of unfractionated heparin at every eight hours for the prophylaxis of deep vein thrombosis.

A trans-thoracic echocardiography was performed with difficulty, showing no evidence of mass or embolic thrombus from other cardiogenic sources. The contrast study was negative for atrial septum defects or persistent foramen ovale. The left ventricular (LV) systolic function was normal, with an ejection fraction of 55–60% and mild hypokinesia of the mid anteroseptal, anteroapical and septal walls. The cavity sizes and wall thickness of the LV and the right ventricle (RV) were normal, with no significant valvular abnormalities, and there was an impaired relaxation pattern of diastolic filling (Figure 3).

TABLE 1. Laboratory results at presentation in the emergency room

Laboratory biomarker	Value	Reference range
Sodium (mmol/L)	137	135–145
Potassium (mm/L)	4.0	3.5–5.3
Chloride (mmol/L)	104	99–109
Glucose (mg/dl)	166	65–99
Urea (mg/dl)	17	8–25
Creatinine (mg/dl)	0.7	0.7–1.3
Anion Gap (mmol/L)	9	5–16
White blood cell count (*10 ³ /uL)	13.39	3.80–11.00
Red blood cell count (*10 ⁶ /uL)	5.43	4.20–5.70
Hemoglobin (g/dl)	15.8	13.2–17.0
Hematocrit (%)	46.1	39.0–50.0
Platelet count (*10 ³ /uL)	276	150–400
International Normalised Ratio (INR)	1.1	0.9–1.2
Thrombin Time (s)	16.5	15.5–19.2
Total Phenytoin serum levels (ug/mL)	29.6–31.9	10.0–20.0

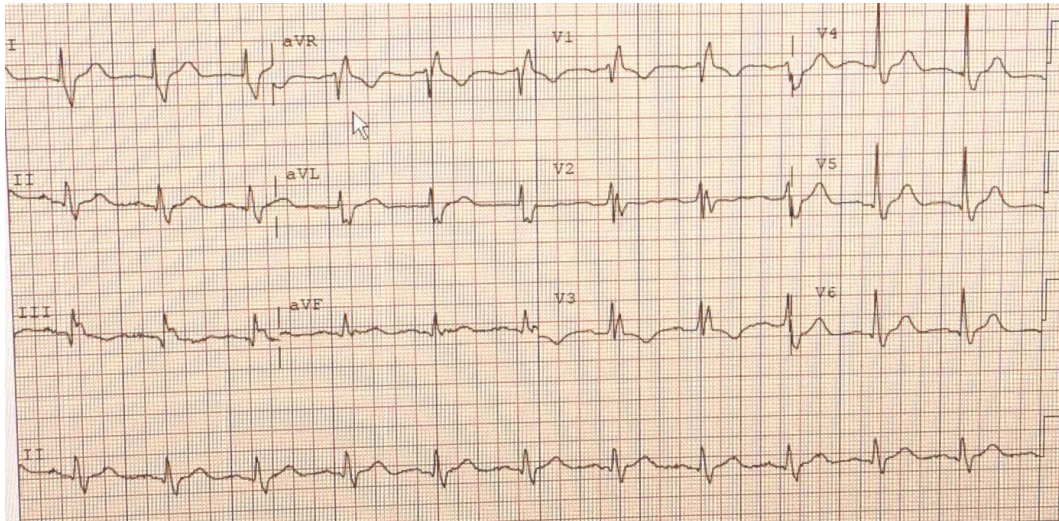


FIGURE 1. Baseline electrocardiographic trace

Due to the presence of abdominal distension, the Gastroenterology Service was asked to perform an assessment. The abdominal X-ray was consistent with small bowel obstruction. Therefore abdominal CT and a surgical assessment were requested, which raised the suspicion of a small bowel obstruction versus ileus. A nasogastric tube placement with suction and close follow-up of the output was recommended. The aspect of the abdominal CT is presented in Figure 4.



FIGURE 2. Head CT examination revealed no intracranial hemorrhage, no evidence of mass, no midline shift or CT findings of acute infarction, no subdural or epidural collections identified, and right occipital shunt unchanged.

On the sixth day after admission, the patient presented with hematemesis, and underwent esogastroduodenal endoscopy which revealed an ulcer in the cardia with a visible vessel for which a hemoclip was placed, and five injections of epinephrine were administered producing successful hemostasis. After the procedure, the patient complained of abdominal pain and distension, for which an abdominal X-ray was performed. The results of the X-ray showed free air under the diaphragm, and the surgical assessment concluded that the patient had an early perforation, for which an emergency laparotomy was performed.

The postoperative diagnosis consisted of the presence of a perforated gastric ulcer, dense adhesions of the jejunum with partial obstruction, and an in-dwelling ventriculoperitoneal shunt. The surgical treatment consisted of a partial anterior gastrectomy with primary closure, the removal of the VP shunt and the lysis of the adhesions. The surgeon's opinion was that the jejunum adhesions had occurred due to the long-standing shunt drainage.

Post-surgery the patient developed a hospital-acquired pneumonia, with a left abdominal fluid collection and a possible abscess, for which dual antibiotic therapy was initiated (cefepime and metronidazole). Also, the patients showed leukocytosis, that did not meet the criteria for sepsis, decreased hemoglobin and hematocrit due to the acute blood loss during the haematemesis episode (Hb – 9.3 g/dL, Htc – 27.7%) for which he had received one unit of blood, and reactive thrombocytosis.

The phenytoin toxicity had resolved by twenty days from cessation, with current levels of 20.2 µg/mL, normal range 10.0–20.0 µg/mL. The elevated white cell blood count had returned to normal, and there was no fever.

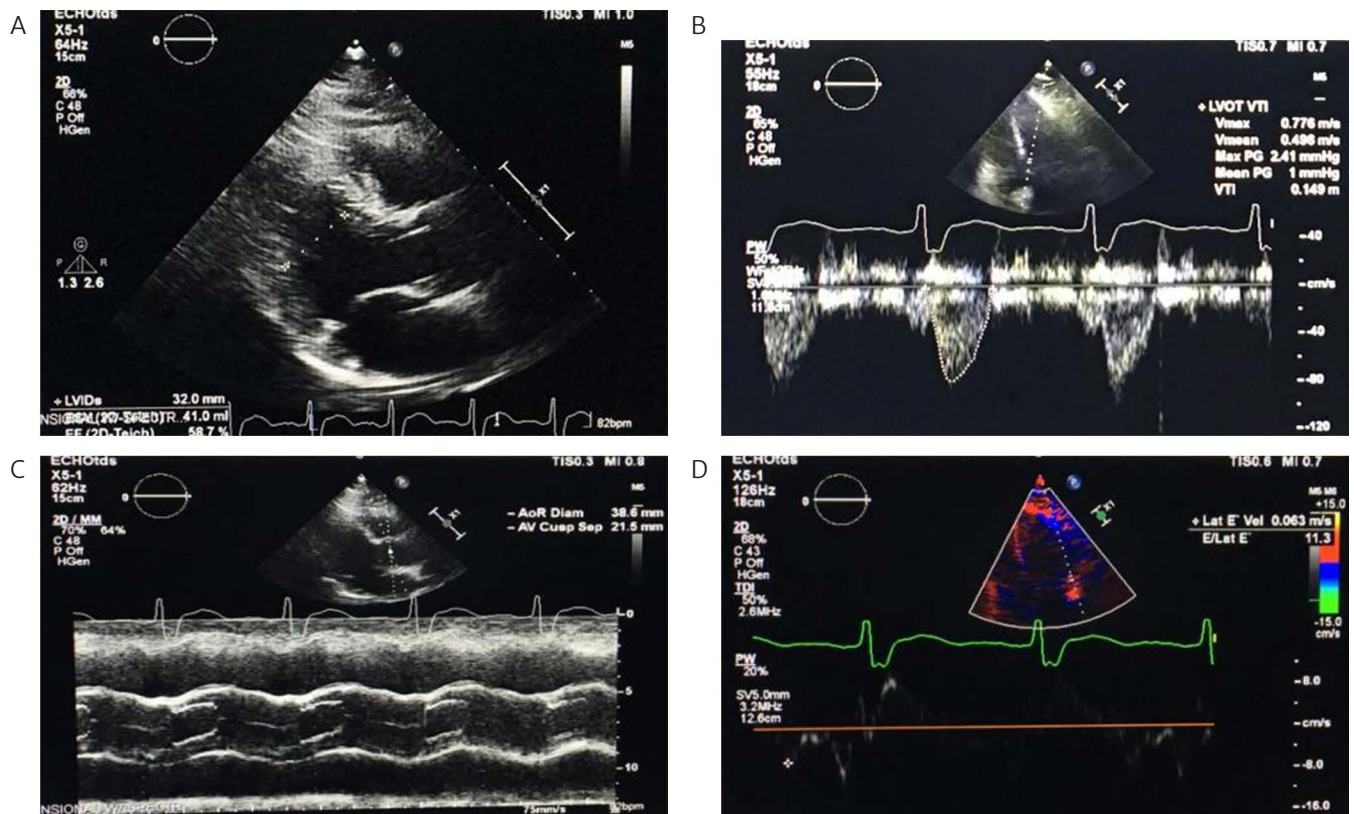


FIGURE 3. Echocardiography assessment. A – 2D examination, parasternal long axis view, showing normal cardiac chambers. B – Doppler flow in the left ventricular outflow tract – normal trace. C – M-mode examination at the level of the aortic leaflets showing normal opening of the aortic valve. D – Tissue Doppler examination showing normal ventricular function.

The long QT interval had improved from 533 ms to 488 ms.

During the night of the twentieth day following admission, the patient had been persistently stuporous, and a digital electroencephalography (EEG) was performed utilizing scalp electrodes. The EEG results showed abnormalities that were consistent with a severe encephalopathy of non-specific etiology, though possibly metabolic, and no electrographic seizures had been captured during the examination.

Finally, after twenty-eight days in the hospital, during which time antiischemic and antibiotic treatment had been administered, the patient was discharged, with an improved overall state and normalization of the vital signs.

The principal diagnosis was acute encephalopathy. The secondary diagnoses and treatments are listed in Table 2.

DISCUSSIONS

Acute coronary syndromes (ACS) consist of a range of conditions — non-ST-segment elevation ACS, which include unstable angina and NSTEMI, and ST elevation myocar-

dial infarction, resulting from atherosclerotic plaque and formation of thrombi, which leads to acute myocardial ischemia.⁷

According to its third definition, a myocardial infarction is diagnosed by encompassing clinical, electrocardiographic and biochemical specific markers.⁸

The clinical manifestations of an acute coronary syndrome consist of chest pain, and atypical complaints including diaphoresis, nausea, weakness, palpitations, anxiety, dyspnea.⁹ Although chest pain is the most common symptom, acute coronary syndromes may present without pain, particularly in older patients, as well as diabetic or heart transplant patients.¹⁰

The first means of diagnosis of ACS patients is the 12-lead ECG. Results may be normal in most patients, but ECG changes in NSTEMI include ST-segment depression or transient elevations and T wave modifications.⁷

In case of silent, asymptomatic myocardial infarction, serological identification of cardiac necrosis is an important diagnostic tool. Cardiac troponins are the preferred biochemical markers for the diagnosis of MI, due to their increased sensitivity and specificity in detecting even small myocardial necroses.⁸

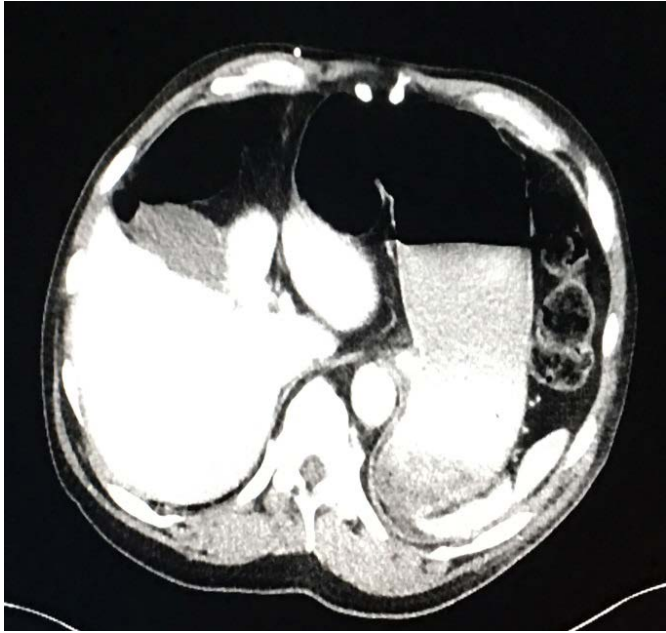


FIGURE 4. Abdominal CT: Liver – normal, no masses; Gall bladder, bile ducts – normal, no stones, no obstructions or inflammation; Spleen – normal size, no masses; Pancreas – normal, no masses or ductal obstruction; Adrenal glands: heterogeneous vascular left adrenal mass measuring 2.9 x 3.1 cm. Right adrenal appears unremarkable; Kidneys – exophytic mass of the upper pole right kidney with intermediate to high Hounsfield units, could represent a renal cyst, however, solid mass is not excluded. This measures 1 cm. No hydronephrosis. Nasogastric tube tip is in the proximal stomach. Large air fluid level in the stomach. Peritoneal cavity – a group of a few dilated small bowel loops at the proximal jejunum, in the left paraduodenal region, next to the Treitz ligament. The small bowel loops are decompressed distal to this. These findings are consistent with proximal small bowel obstruction. Paraduodenal internal hernia is possible.

Despite the high myocardial specificity, troponins may present abnormal values in other conditions, not related to the coronary atherosclerotic disease, such as myocarditis, heart failure, pulmonary embolism, septic shock, renal dysfunction, or chronic obstructive pulmonary disease (COPD).¹¹ Furthermore, an elevated troponin level may also be found in case of cerebrovascular injuries, such as subarachnoid haemorrhage or ischemic stroke.¹² Other acute troponin elevations occur in case of trauma, including contusion, ablation, surgery, biopsy, hypertension-hypotension, critical illness such as diabetes, respiratory failure, gastrointestinal bleeding, sepsis, drug toxicity with adriamycin or 5-fluorouracil, Herceptin, hypothyroidism, apical ballooning, burns, infiltrative diseases that affect the myocardium, as well as inflammatory diseases.¹³

In this particular case, the patient presented with multiple associated pathologies, including a post-traumatic

TABLE 2. Secondary diagnoses

1. Phenytoin overdose
2. NSTEMI
3. Prolonged QT interval
4. Posttraumatic seizure disorder
5. VP shunt status
6. Small bowel obstruction
7. Perforated gastric ulcer
8. Sepsis
9. Anemia secondary to acute blood loss
10. Thrombocytosis
11. Hospital-acquired pneumonia
12. Abdominal fluid collection

brain injury that caused hydrocephalus, which in turn required the permanent implantation of a ventriculoperitoneal shunt. The patient had been diagnosed with non-ST elevation myocardial infarction, despite the lack of chest pain or other symptoms that would indicate an acute coronary syndrome, without any significant electrocardiographic signs. The NSTEMI diagnosis was based on the elevated troponin level but, as mentioned above, a troponin rise may occur in several pathologies that are not related to coronary artery disease. The elevated troponin levels, in this case, could have emerged due to the cerebral injury of the VP shunt placement, due to the gastrointestinal bleeding or the anemia secondary to the acute blood loss, or it could have been caused by the infection that occurred due to the perforated gastric ulcer.

The patient also presented with regional myocardial kinetic impairment upon echocardiographic assessment, but the modifications did not appear to be of an acute nature.

The current case was remarkable because of the NSTEMI diagnosis that was based solely on the elevated troponin levels, with lack of any evident clinical, ECG or echocardiographical modifications. The NSTEMI diagnosis was not sustained because of the multiple causes that could have led to an acute rise in troponin levels. This was also the reason why the patient did not undergo an interventional diagnostic and treatment approach.

CONCLUSION

This case underlines the importance of a complex differential diagnosis in patients presenting at the Emergency Room with chest pain and raised troponin levels, to accurately establish the diagnosis of NSTEMI and differentiate the real NSTEMI cases from an isolated increase of

troponin values. This is particularly important in complex NSTEMI cases, such as the one described, when patients present with many associated comorbidities.

CONFLICT OF INTEREST

Nothing to disclose.

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