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Differential Diagnosis Clinical Presentation and Minimal Risk Factors – ST Segment Elevation Myocardial Infarction vs Myopericarditis: A Case Report

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KEYWORDS
STEMI; myopericarditis; differential diagnosis; clinical features; minimal risk factors

ABSTRACT
The purpose of this study is to find out differential diagnosis clinical presentation and minimal risk factors – st segment elevation myocardial infarction vs myopericarditis. The condition of STEMI (ST Segment Elevation Myocardial Infarction) can resemble infectious diseases of the heart such as Myopericarditis with the presence of dominant gastrointestinal symptoms, minimal demographic characteristics and cardiovascular risk factors as well as limited supporting examinations, making it a challenge in determining the diagnosis of cardiovascular disease. Case Report: 31 year old male with complaints of pain in the pit of the stomach that was difficult to describe and discomfort that extended to the chest that was not specific, a history of fever for 2 days, nausea and vomiting and a history of the habit of only consuming alcohol without smoking and the patient doing routine physical activities. ECG examination showed sinus rhythm with ST segment elevation in the precordial (anterior and lateral), inferior and LAD (Left Axis Deviation) leads as well as PR segment depression, Troponin I increased and Echocardiography showed a hypokinetic pattern in the posterior and septal segments of the heart. Fibrinolytics improved the patient's symptoms but not serial EKGs, giving rise to suspicion of STEMI which resembles Myopericarditis. Conclusion: Differential diagnosis with the possibility of myopericarditis was based on clinical findings and minimal risk factors, history of the course of the disease, ECG examination, Cardiac Biomarkers, TTE and Coronary Angiography.

Introduction

Gastrointestinal symptoms are symptoms that are not specific to cardiovascular cases. With the characteristics of young patients and minimal cardiovascular risk factors as well as limited supporting examination facilities, it will make it difficult to diagnose cardiovascular cases. We present this case report showing the clinical presentation along

with findings in several examinations that support the diagnosis of STEMI (ST Segment Elevation Myocardial Infarction) compared with acute myopericarditis. Myopericarditis as a differential diagnosis is a pericarditis syndrome with inflammatory involvement of the myocardium (Rami Reddy & Cappell, 2017).

Case Illustrations

31 year old man with the main complaint of pain in the pit of the stomach that was difficult to describe and felt uncomfortable to the chest which was non-specific. It did not improve after giving antacids and consuming food, the patient came to the emergency department (ER). It is known that 2 days before entering the ER, the patient complained of fever that came and went without taking medication, nausea and vomiting that occurred simultaneously. Before the fever occurred, the patient had consumed alcohol in minimal doses. The first onset of pain in the pit of the stomach and chest occurred after fever appeared and the patient felt that he was breaking out in a cold sweat but complaints of shortness of breath, palpitations, fatigue, swollen legs, decreased consciousness were denied. The patient's history of heart disease and other medical conditions relevant to chronic disease was unknown, history of habits such as alcohol consumption since young, the patient does not smoke and has regular physical activity. In the family history of disease, there was a male in the family who had died at a young age (18 years) suddenly after waking up, the family history of heart disease was unknown.

The patient's condition was fully conscious when he entered the ER and had vital signs such as blood pressure 135/96 mmHg, pulse 102 times per minute, respiratory rate 20 times per minute, axillary body temperature 36oC, and oxygen saturation 98% on the pain scale 4. Physical examination of the heart revealed a regular heart rhythm, no murmurs and gallops were found and determination of heart boundaries by physical examination was found to be normal. Physical evaluation of the lungs was within normal limits, there was no friction rub, there was no visible edema on the extremities and the acral was warm. A quick examination carried out while in the ER was a 12-lead electrocardiography (ECG) (Figure 1) which found sinus rhythm with ST segment elevation in the precordial (anterior and lateral), inferior and LAD (Left Axis Deviation) leads as well as PR segment depression. With clinical and examination findings supporting acute myocardial infarction, the patient was given fibrinolytic therapy with Streptokinase 1.5 million units, along with Aspirin 160 mg, Clopidogrel 300 mg, Atorvastatin 40 mg, Enoxaparin 0.6 mL in the emergency room and immediately transferred to the intensive care unit.

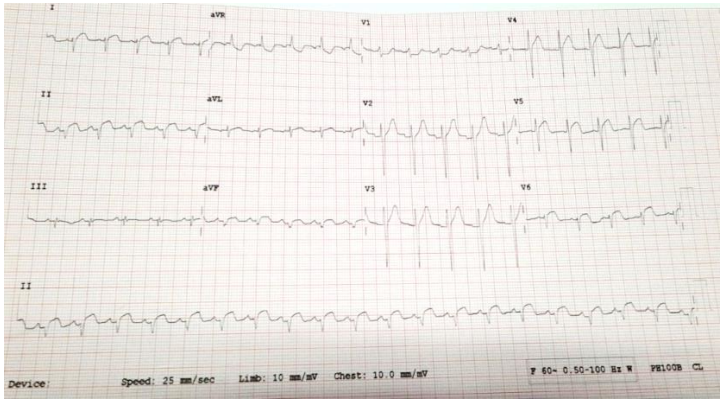

Figure 1. Results of the first electrocardiography (ECG) examination showing ST segment elevation in the precordial (anterior and lateral), inferior and LAD (Left Axis Deviation) leads as well as PR segment depression.

At around 22.15 (UTC+8), the patient's condition was good with compos mentis consciousness, and complaints of chest pain were no longer felt. At around 00.05 (UTC+8) the patient's heart rate was 123 beats per minute and additional therapy was given in the form of Bisoprolol 2.5mg. At around 03.30 (UTC+8) the patient complained of nausea and pain in the pit of the stomach and the patient received Omeprazole 40mg and Sucralfate 2g therapy (Wu et al., 2015).

At around 08.00 (UTC+8), a serological examination was carried out in the form of Troponin I >15 ng/mL, Liver Function in the form of SGPT 179 U/L and SGOT 289 U/L, Current Blood Sugar 146 mg/dL, Urea 27 mg/dL, Blood Creatinine 0.8 mg /dL, Electrolytes in the form of Blood Sodium 142 mmol/L, Blood Potassium 3.8 mmol/L, Blood Chloride 105 mmol/L, Hematology in the form of Leukocytes 10.3 x 10^3/uL, Number of Erythrocytes 5.1 x 10^6/uL, Hemoglobin 15.0 g /dL, Hematocrit 44.4%, MCV 87.1 fL, MCH 29.4 pg, MCHC 33.8 g/L, Platelet Count 193 x 10^3/uL, Neut% 67.7%, Lymp% 18.3%, Mono% 11.6%, Eosi% 2.0% , Baso% 0.4%.

A Transthoracic Echocardiography (TTE) examination was also carried out (Figure 2) with the results of normal heart chamber dimensions, no LVH (Left Ventricular Hypertrophy), normal left ventricular systolic function (55.21%), normal left ventricular diastolic function, normal right ventricular contractility, movement of the left ventricular wall has positive RWMA (Regional Wall Motion Analysis), that is, there was hypokinetic in the posterior and septal part of the heart, in other regions it was normokinetic, there were no abnormalities in the heart valves and no pericardial effusion was found (Baker, 2016).

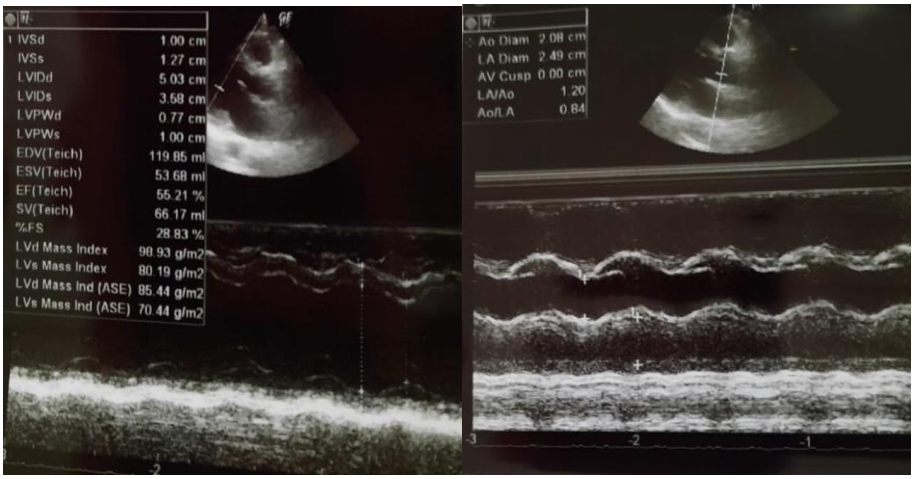

Figure 2. (A) Results of Transthoracic Echocardiography (TTE) examination showing left ventricular dimensions (LVIDd) and left ventricular ejection fraction; (B) Showed the size of the diameter of the left atrium (LA Diam).

12-lead Electrocardiography (ECG) examinations were carried out in a sequence during the period of stay in the intensive care unit. On the second ECG examination (Figure 3), ST segment elevation in the precordial leads and VES (Ventricular Extra-systole) rhythm were found in a unifocal and sequential (Couplet) form, and occur episodically. Serial ECG examinations in the precordial leads did not show any ECG changes or evolution leading to a diagnosis of ST-Elevation Myocardial Infarction (STEMI), thus directing the differential diagnosis towards myopericarditis. In addition, on the second ECG carried out in the intensive room, there were changes in the isoelectric line of the depressed PR segment (Torsson et al., 2017).

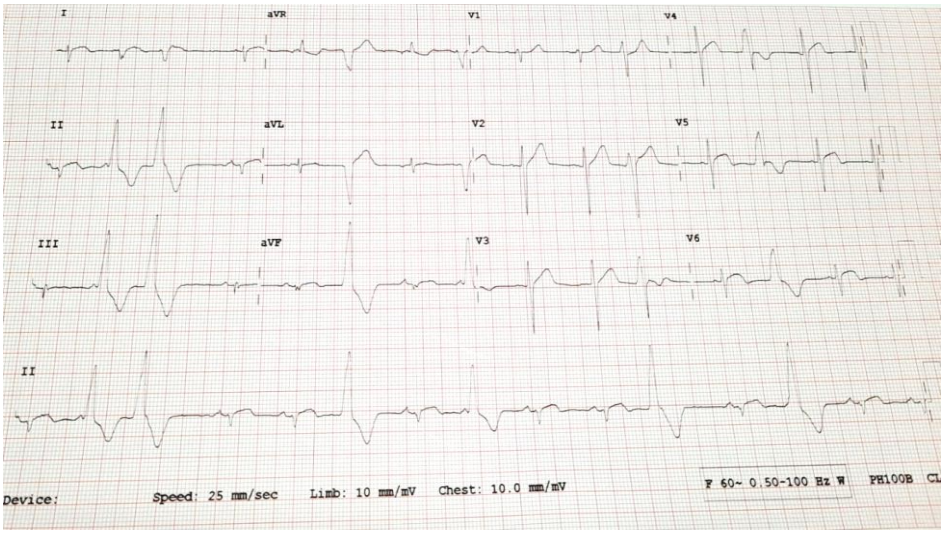

Figure 3. Results of the second ECG examination which showed that there was a Ventricular Extra-systole rhythm that was unifocal and sequential (Couplet), and occurred episodically

On the third day after being admitted to the hospital, the patient was moved from the intensive room to a regular room and the patient had a nosebleed once when he sneezed. At first the patient did not report the incident. On the fourth day after being admitted to hospital, the patient had another nosebleed for the second time and reported it to the staff so that Aspirin, Clopidogrel and Enoxaparin therapy was stopped. The patient went home after being treated for 5 days at our hospital and received home therapy of Clopidogrel 1x75 mg, Bisoprolol 1x2.5 mg, Atorvastatin 1x40 mg, Amiodarone 2x100 mg and Sucralfate Suspension 3xC2. After 2 weeks post-hospitalization the patient came for further examination and therapy in the outpatient setting. When patients undergo outpatient treatment, they are advised to undergo a Coronary Angiography examination and receive home therapy in the form of Aspirin 1x80 mg, Clopidogrel 1x75 mg, Atorvastatin 1x20 mg and Bisoprolol 1x2.5 mg.

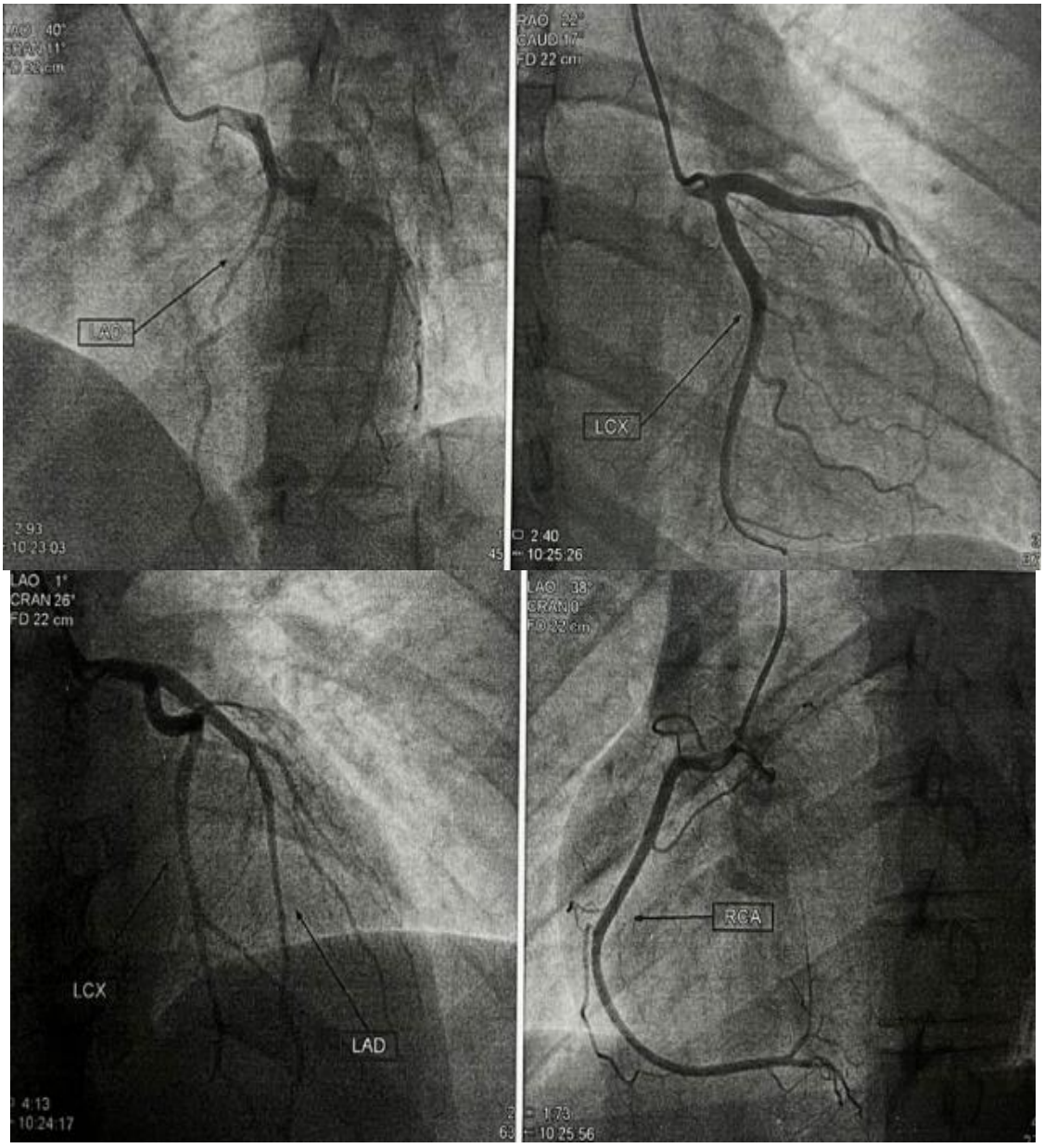

Figure 4. Coronary Angiography examination results showed normal results in the LM (Left Main), LAD (Left Anterior Descenden), and LCX (Left Circumflex); there is non-significant stenosis in the RCA (Right Coronary Artery).

Several weeks after the patient underwent outpatient treatment, the patient underwent a Coronary Angiography examination with normal results on the Left Main, LAD (Left Anterior Descenden) and LCX (Left Circumflex); and there was non-significant stenosis in the RCA (Right Coronary Artery). With the results of a Coronary Angiography examination like this, the conclusion is Non-Significant Stenosis in the RCA.

Research Methods

This type of research is library research. Literature study is a type of research conducted by collecting data from literature such as books, journals, research reports and other data sources. This research uses a documentation-descriptive approach, because the source of data and discussion of research in library research studies is in the form of

descriptions of words where the written data will be studied based on the context, Descriptive-documentation research is a method that seeks to describe and interpret objects according to what they are with documentary analysis, namely by analyzing documentation data from books, journals, research reports and others Etc.

Results and Discussions

Symptoms, a wide increase in the ST segment in the precordial, inferior and LAD (Left Axis Deviation) leads until there was an increase in Troponin I concentration could suggest that the patient had been diagnosed with ACS (Acute Coronary Syndrome). With fibrinolytic therapy given there was improvement in symptoms but the ECG showed no evolution leading to ACS (STEMI). A history of illness with fever, nausea and vomiting for 2 days, and the absence of risk factors for cardiovascular disease (age, comorbidities and habits) created confusion in the diagnosis of STEMI. The demographic characteristics of ACS and Myopericarditis cases in terms of gender are the same, namely male (Wieczorkiewicz et al., 2022).

Myocardial cell involvement causing acute myocarditis alone cannot be excluded because this patient had unclear signs of infection such as a history of fever, nausea and vomiting 2 days before being admitted for chest pain. Based on a case report by Ko H et al, acute myocarditis can be accompanied by non-specific symptoms in the form of gastrointestinal complaints (nausea, vomiting, abdominal pain and decreased appetite) and fever as in this case. This clinical presentation can occur due to viral infection which is the etiology of acute myocarditis with a direct viral mechanism and an adaptive immune response that damages myocardial cells, causing myocellular necrosis and apoptosis which mimics the signs and symptoms of ACS with a different pathophysiology, namely ischemia or infarction due to atherosclerotic plaque thrombi. (increase in precordial and inferior ST segment leads and increase in Cardiac Biomarkers) (Ko et al., 2022).

The various clinical presentations that patients complaint about, created several differential diagnoses that could occur in this case. One of the differential diagnoses in this case was acute pericarditis where there were 2 of 4 criteria such as 1) sharp or pleuritic chest pain, which improved when sitting and leaning the chest forward, 2) pericardial friction rub, 3) widespread ST segment elevation that new or depressed PR segment and 4) pericardial effusion.

Acute pericarditis could be established in this case because it met these 2 criteria, namely criteria 1 and 3. However, if these 2 criteria are added to the results of an increase in the concentration of the Cardiac Biomarker (Troponin I), the differential diagnosis can be myopericarditis. Myopericarditis is a new entity defined as a pericarditis syndrome with involvement of the myocardium (Prepoudis et al., 2022).

Transthoracic Echocardiography (TTE) cannot differentiate inflammation and ischemia or infarction in the myocardium between myopericarditis and ACS. These findings were the same, namely abnormalities in the movement of the segmental walls of the heart (RWMA / Regional Wall Motion Abnormalities) in the form of a hypokinetic pattern. In this case, there were changes in the movement patterns of the posterior and septal walls of the heart which were possibly caused by myocyte cell necrosis but the pathophysiological basis could not be determined. In the case report from Ardiana M and Aditya M, myopericarditis might be confirmed by signs of severe or prolonged ventricular dysfunction, myocardial involvement due to thickening of the myocardial segment, and the presence of pericardial effusion from several TTE examination positions. In this case report, the suspicion of acute myocarditis was also seen from the distribution of ST

segment elevation on the ECG that did not match the segmental heart wall movement abnormalities observed on TTE. This also happened in our case, where the ECG showed an increase in the ST segment in the precordial (anterior and lateral) to inferior leads, while the segmental wall movement abnormalities of the heart were only in the posterior and septal leads (Ardiana & Aditya, 2022).

The first ECG examination that produces a picture of ST segment elevation usually occurs in ACS (STEMI) conditions, namely as a marker of epicardial coronary artery patency and a marker of myocardial tissue reperfusion. However, ST segment deviation in myopericarditis can occur as a result of 3 factors, namely pericardial effusion, superficial myocardial injury by fibrin or fluid pressure and superficial myocarditis secondary to a local inflammatory reaction in the epicardium which underlies inflammation in the pericardium. According to Le CK, ECG anomalies such as concave and diffuse ST segment elevation in pericarditis occur due to pericardial effusion or inflammation of the adjacent superficial myocardium. Myocarditis also shows non-specific ST segment abnormalities, T wave abnormalities, conduction abnormalities or decreased QRS complexes. In the case report reported by Le CK, ECG changes in myopericarditis showed low amplitude ECG, transient T waves in the precordial and lateral leads or inversion and ST segment changes. Le CK used an ECG initially to diagnose STEMI, but ST segment and T wave evolution on subsequent examinations indicated myopericarditis. In line with this case report, the evolution of the ST segment and T wave in our patient did not lead to a STEMI in the precordial leads, but rather a revolution occurred that made the differential diagnosis point to myopericarditis. Based on a case report by Choi HJ et al, a change in the isoelectric line pattern of the PR segment to depression strengthens the diagnosis of myopericarditis. This indicates atrial involvement which is responsible on the ECG as a picture of PR segment depression in cases of myopericarditis (Le et al., 2022)(Choi et al., 2022).

Serial ECG examinations carried out on this patient showed the presence of ventricular extra-systole (VES) rhythms that were unifocal and sequential (Couplet), and occurred episodically. The presence of myocyte cell death due to inflammatory reactions due to myopericarditis or infarction due to acute coronary syndrome allows the emergence of premature ventricular beats as the etiological basis. In research conducted by Jeserich M et al, the ectopic focus of an electrical spark that causes a premature ventricular beat can be a pathological substrate such as myocardial edema or scar tissue, where myocardial edema is usually found in conditions of acute myocarditis due to the acute inflammatory process or scar tissue that is caused. due to ischemia or infarction in acute coronary syndrome. This pathological substrate can be detected by performing CMR, but in this case it was not performed. In line with the research mentioned by Laloglu F and Ceviz N, which reported a 65% frequency of arrhythmias, the majority of which were atrial and ventricular extra-systole in adult myopericarditis patients. This study also evaluated myopericarditis in children and adolescents, where there were 4 out of 27 patients who experienced frequent arrhythmias and conduction disorders, namely 1 patient with Idioventricular Rhythm, 1 patient with Ventricular Couplet with Low Rate, 1 patient with Infrequent Supraventricular Premature Contraction and 1 patient with Infrequent Ventricular Premature Contraction + Wenckenbach type Atrioventricular Block. This study also provides an explanation that the frequency of arrhythmias and conduction disturbances in myopericarditis is very low and is benign so that it does not significantly cause left ventricular systolic dysfunction as evaluated by ejection fraction via TTE (Jeserich et al., 2015)(Laloğlu & Ceviz, 2022).

With the clinical presentation and history of the patient's disease, findings from serial ECG and TTE examinations, we had a differential diagnosis and suspicion of myopericarditis. This was in accordance with the case report of Hasibuan FS et al, where the differential diagnosis of myopericarditis was determined from the results of the patient's clinical presentation with supporting examinations in the form of serial ECGs and TTE and excluding differential diagnoses from other non-cardiac causes. The case report by Hasibuan FS et al requested a referral to a tertiary hospital for coronary angiography examination but the patient and family refused because the patient was being treated in a hospital that had limited supporting facilities (Hasibuan et al., 2020).

Additional supporting examinations were carried out in the form of hematology, and leukocytosis was found which created a basic suspicion regarding the pathophysiology of the clinical presentation, ECG changes and an increase in Cardiac Biomarkers based on an inflammatory process due to infection. The limitation experienced by this patient was that they did not undergo CRP (C-Reactive Protein) examination as a marker of acute inflammation which could at least objectively differentiate myopericarditis from acute coronary syndrome, by determining the CRP/Troponin ratio. However, the troponin used was T-type troponin in this ratio. This is in accordance with research conducted by Meisel SR et al, who differentiated myopericarditis and acute coronary syndrome using the CRP/Troponin ratio. Where a CRP/Troponin ratio >250 can support the possibility of myopericarditis and >500 strengthens the definite diagnosis of myopericarditis. However, even though it strengthens a definite diagnosis for myopericarditis, imaging examinations must still be carried out as the gold standard in diagnosing myopericarditis. The condition of leukocytosis in this patient could not establish an inflammatory process due to myopericarditis as a pathophysiological basis because this could also occur in ACS (Meisel et al., 2021).

Based on case report by Ardiana M and Aditya M, therapy with NSAIDs (Non-Steroid Anti-Inflammatory Drugs) needed to be avoided because it could worsen myocardial damage by increasing sodium retention, thereby increasing fluid, worsening renal hypoperfusion and increasing the risk of death. This drug worsens myocardial inflammation and necrosis by reducing pro-inflammatory factors such as interferon, thereby increasing the viral load. Another mechanism was explained by the case report of Kurtul A et al, that NSAIDs inhibit the COX-1 and COX-2 enzymes and then inhibit prostacyclin synthesis competitively and irreversibly, thereby increasing the risk of coronary thrombosis. COX-2 inhibitor NSAID therapy could trigger platelet hyperactivity and an imbalance between thromboxane / prostacyclin resulting in prothrombotic endothelium. Administration of NSAIDs would increase the risk of cardiovascular disease with or without other cardiovascular risk factors because NSAIDs would cause coronary artery spasm and rupture of vulnerable atherosclerotic plaque (Kurtul et al., 2020).

Conclusion

With a clinical presentation predominantly gastrointestinal symptoms and minimal risk factors, it was difficult to determine whether ST segment elevation was caused by infarction or inflammation of the myocardium or pericardium. By carrying out a Coronary Angiography examination, the diagnosis of STEMI could be excluded so that in our case report, we got a working diagnosis of Myopericarditis. Other supporting data for this working diagnosis were meeting the criteria for myopericarditis (sharp or pleuritic chest pain, which was improved when sitting and leaning the chest forward, new widespread ST segment elevation or PR segment depression and myocardial involvement (increased Cardiac Biomarker (Troponin I) concentration); 3 as well as the distribution of ST segment elevation on the ECG did not correspond to the cardiac segmental wall movement abnormalities observed on TTE.4 In this case report, in the determination of Myopericarditis in the absence of CMR and immuno-histological evidence from EMB (Endomyocardial Biopsy). The patient's discharge showed no changes in the ECG pattern like STEMI, but the appearance of Ventricle Extra-systole has corrected itself.

References

Ardiana, M., & Aditya, M. (2022). Acute Perimyocarditis–an ST-Elevation Myocardial Infarction Mimicker: A Case Report. *The American Journal of Case Reports*, *23*, e936985-1.

Baker, J. (2016). Functional voice disorders: Clinical presentations and differential diagnosis. *Handbook of clinical neurology*, *139*, 389–405.

Choi, H. J., Kwon, H. W., Oh, K. J., & Song, M. K. (2022). Acute perimyocarditis mimicking acute myocardial infarction in a 12-year-old boy with Duchenne muscular dystrophy. *Acute and Critical Care*, *37*(2), 258–262.

Hasibuan, F. S., Intan, R. E., Wilujeng, H. R. T., Octora, T. N., Dharmajati, B. B., Gandi, P., & Alkaff, F. F. (2020). Triangular QRS-ST-T waveform electrocardiographic pattern in acute myopericarditis: a case report from a limited-resources hospital. *The American Journal of Case Reports*, *21*, e926360-1.

Jeserich, M., Merkely, B., Olschewski, M., Kimmel, S., Pavlik, G., & Bode, C. (2015). Patients with exercise-associated ventricular ectopy present evidence of myocarditis. *Journal of Cardiovascular Magnetic Resonance*, *17*, 1–7.

Ko, H., Kim, T., Lee, H. D., Byun, J.-H., & Choo, K. S. (2022). Coronary artery spasm due to acute myocarditis in an adolescent: a case report. *BMC pediatrics*, *22*(1), 304.

Kurtul, A., Şen, F., & Bekler, Ö. (2020). *Acute anterior myocardial infarction during myopericarditis treatment in a very young adult*.

Laloğlu, F., & Ceviz, N. (2022). Subclinical Arrhythmias and Conduction Disturbances in Children with Myopericarditis: A 24-hour Holter Monitorization Study. *The Eurasian Journal of Medicine*, *54*(3), 239.

Le, C.-K., Nguyen, M. B., & Vo, A. T. (2022). ST-elevation in an adolescent with COVID-19: Myopericarditis or myocardial infarction? *The American Journal of Emergency Medicine*, *52*, 271-e1.

Meisel, S. R., Nashed, H., Natour, R., Abu Fanne, R., Saada, M., Amsalem, N., Levin, C., Kobo, O., Frimerman, A., & Levi, Y. (2021). Differentiation between myopericarditis and acute myocardial infarction on presentation in the emergency department using the admission C-reactive protein to troponin ratio. *Plos one*, *16*(4), e0248365.

Prepoudis, A., Koechlin, L., Nestelberger, T., Boeddinghaus, J., Lopez-Ayala, P., Wussler, D., Zimmermann, T., Rubini Giménez, M., Strebel, I., & Puelacher, C. (2022). Incidence, clinical presentation, management, and outcome of acute pericarditis and myopericarditis. *European Heart Journal Acute Cardiovascular Care*, *11*(2), 137–147.

Rami Reddy, S. R., & Cappell, M. S. (2017). A systematic review of the clinical presentation, diagnosis, and treatment of small bowel obstruction. *Current gastroenterology reports*, *19*, 1–14.

Torsson, E., Berg, M., Misinzo, G., Herbe, I., Kgotlele, T., Päärni, M., Roos, N., Blomström, A.-L., Ståhl, K., & Johansson Wensman, J. (2017). Seroprevalence and risk factors for peste des petits ruminants and selected differential diagnosis in sheep and goats in Tanzania. *Infection Ecology & Epidemiology*, *7*(1), 1368336.

Wieczorkiewicz, P., Przybylak, K., Supel, K., Kidawa, M., & Zielinska, M. (2022). Symptomatic Young Adults with ST-Segment Elevation—Acute Coronary Syndrome or Myocarditis: The Three-Factor Diagnostic Model. *Journal of Clinical Medicine*, *11*(4), 916.

Wu, Z., Boersema, G. S. A., Dereci, A., Menon, A. G., Jeekel, J., & Lange, J. F. (2015).

Clinical endpoint, early detection, and differential diagnosis of postoperative ileus: a systematic review of the literature. *European Surgical Research*, *54*(3–4), 127–138.