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Myocardial infarction in young individual: A case report of polycythemia vera-induced acute inferior wall myocardial infarction

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Abstract

Acute coronary syndrome is commonly associated with traditional cardiovascular risk factors such as smoking, hypertension, diabetes, and hyperlipidemia. Myocardial infarction in a young person presents a significant challenge because its etiology is least likely associated with atherosclerosis. Polycythemia vera refers to one of the rare causes of myocardial infarction, which involves enhanced erythrocyte levels, leukocytosis, thrombocytosis, splenomegaly, and a greater chance of vascular occlusion due to clotting in coronary arteries. A 22-year-old male from Pakistan, Asia without typical risk factors, presented with severe chest pain. Electrocardiography indicated acute inferior wall myocardial infarction, and streptokinase was administered. Subsequent investigations confirmed polycythemia vera. Treatment with hydroxyurea and aspirin was initiated, whereas normal coronary arteries in CT coronary angiogram were observed. This case highlights polycythemia vera's rare role in young individuals' heart attacks without known risk factors, emphasizing the need for early detection and specialized treatments involving hematologists to prevent future thrombotic episodes.

Keywords

Polycythemia vera, myocardial infarction, inferior wall ST elevated myocardial infarction, antiplatelet therapy, fibrinolytic therapy, hematology

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Introduction

Several cardiac risk factors are commonly observed in individuals with acute coronary syndrome (ACS). Traditional risk factors for coronary artery disease include smoking, hypertension, diabetes, hyperlipidaemia, and a hereditary predisposition to the condition.[1] Young people facing myocardial infarction (MI) encounter significant challenges as the disease is less likely caused by atherosclerosis. ACS manifestations in this age group are often triggered by substance abuse and hypercoagulable conditions.[2]

A very uncommon cause of acute MI is polycythaemia vera (PV), characterized by an absolute rise in erythrocytes, leucocytosis, and thrombocytosis. It increases the propensity for thrombus formation in the coronary arteries by making the blood more viscous and increasing platelet activity.[3–5] In this paper, we present a rare case of acute inferior wall MI in a 22-year-old Asian man caused by PV, lacking any conventional risk factors associated with cardiovascular diseases.

Case presentation

A 22-year-old Asian man arrived at the coronary care unit through the emergency room, experiencing severe chest pain for the past 4 h. The pain, was substernal, radiated to the left shoulder and jaws, accompanied by diaphoresis and vomiting. Despite lacking prior cardiovascular risk factors, recreational drug use, and being a non-smoker, he frequently used diclofenac sodium for headaches. On examination, he appeared unwell but afebrile, having a healthy weight,

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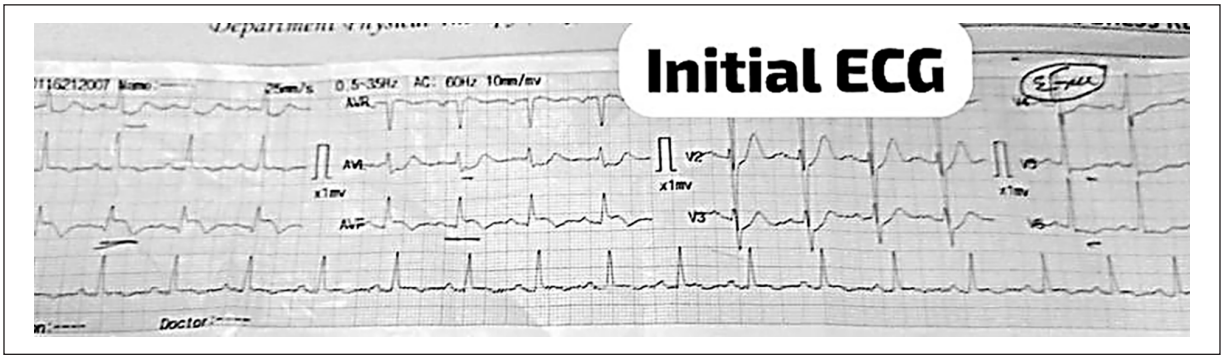

Figure 1. Standard 12-lead electrocardiogram, showing ST segment elevation of 2 mm in in III, AVF and ST segment depressions in V1–V6, I and AVL (reciprocal changes).

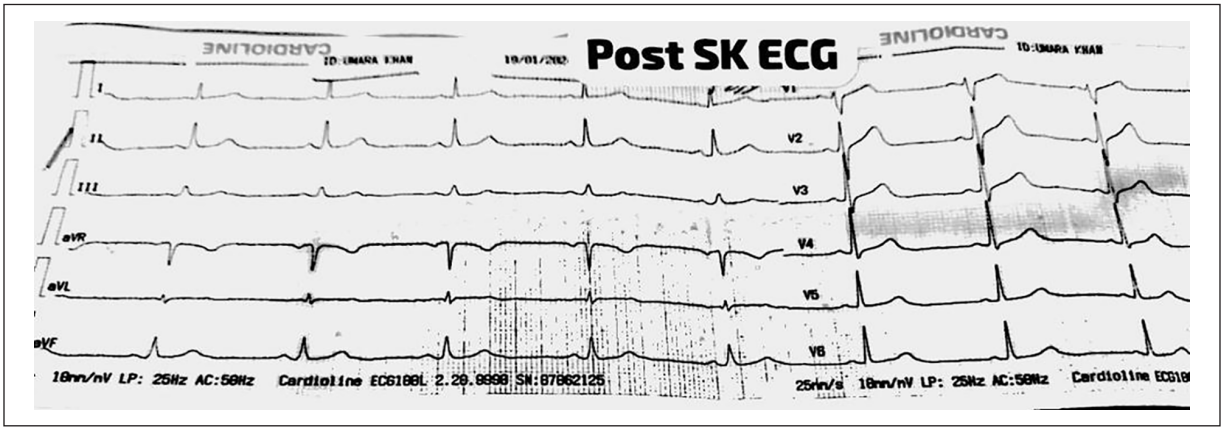

Figure 2. Post-SK electrocardiogram done at 30 min interval after streptokinase infusion, showing successful resolution of ST segment elevations and reciprocal changes.

regular pulse of 82/min, blood pressure of 100/70 mmHg, respiratory rate of 20/min, and 96% saturation at room air. Systemic examination revealed no abnormalities, with normal peripheral pulses and no evidence of elevated jugular venous distention, ankle, or sacral edema.

Upon admission, a comprehensive examination revealed noteworthy findings in various hematological parameters. The white blood cell count showed an elevation at 12.1×10^3/uL (normal range: $4.0–10 \times 10^3$/uL), indicating a predominance of neutrophilic leucocytosis. Hemoglobin levels were elevated at 18.5 g% (normal: 12–16 g%), accompanied by an increased hematocrit of 56.9% (normal: 40–54%). The Mean Corpuscular Volume measured 87 FL (normal: 80–100 FL), with Red Blood Cells numbering 6.55×10^6 (normal: $4.0–5.5 \times 10^6$/uL), and platelets at 270×10^3/uL (normal: $100–300 \times 10^3$/uL).

His highly sensitive troponin-I level, assessed upon arrival, was high at 533.5 ng/L (typical upper limit: 29.9 ng/L). Despite these hematological and cardiac abnormalities, the patient's serum electrolytes were within normal limits: Sodium (Na): 138 mmol/L (normal range: 135–145 mmol/L), Potassium (K): 3.9 mmol/L (normal range: 3.5–5.0 mmol/L), Chloride (Cl): 102 mmol/L (normal range: 98–106 mmol/L), urea: 24 mg/dL (normal range: 10–50 mg/dL), and creatinine: 1.1 mg/dL (normal range: 0.7–1.3 mg/dL).

The Lipid Profile exhibited Total Cholesterol at 165 mg/dL (normal: $<$200 mg/dL), Triglycerides at 122 mg/dL (normal: $<$150 mg/dL), Low-Density Lipoprotein Cholesterol at 72 mg/dL (normal: $<$100 mg/dL), and High-Density Lipoprotein Cholesterol at 36 mg/dL (normal: 40–60 mg/dL).

The Electrocardiography at admission indicated ST segment elevation of 2mm in the inferior leads (II, III, and AVF), along with T-wave inversions in the anterolateral leads. A diagnosis of acute inferior wall ST-segment elevation myocardial infarction (STEMI) was made (Figure 1). The patient received loading doses of aspirin (300 mg), clopidogrel (300 mg), enoxaparin (1 mg/kg), and rosuvastatin (20 mg) immediately. After obtaining consent and ruling out any contraindications, the patient was thrombolyzed with 1.5 million units of streptokinase IV over the course of an hour using an infusion pump. This was done because the cardiac catheterization lab and primary percutaneous intervention (PCI) facility were not available, and transfer to the closest facility was not possible within the 120-min interval. After thrombolysis, the patient's chest pain and ST elevations subsided, as shown in the post-streptokinase electrocardiogram at 30-min intervals (Figure 2).

Additional tests were conducted to confirm suspicions raised by the initial baseline studies. Arterial blood gas

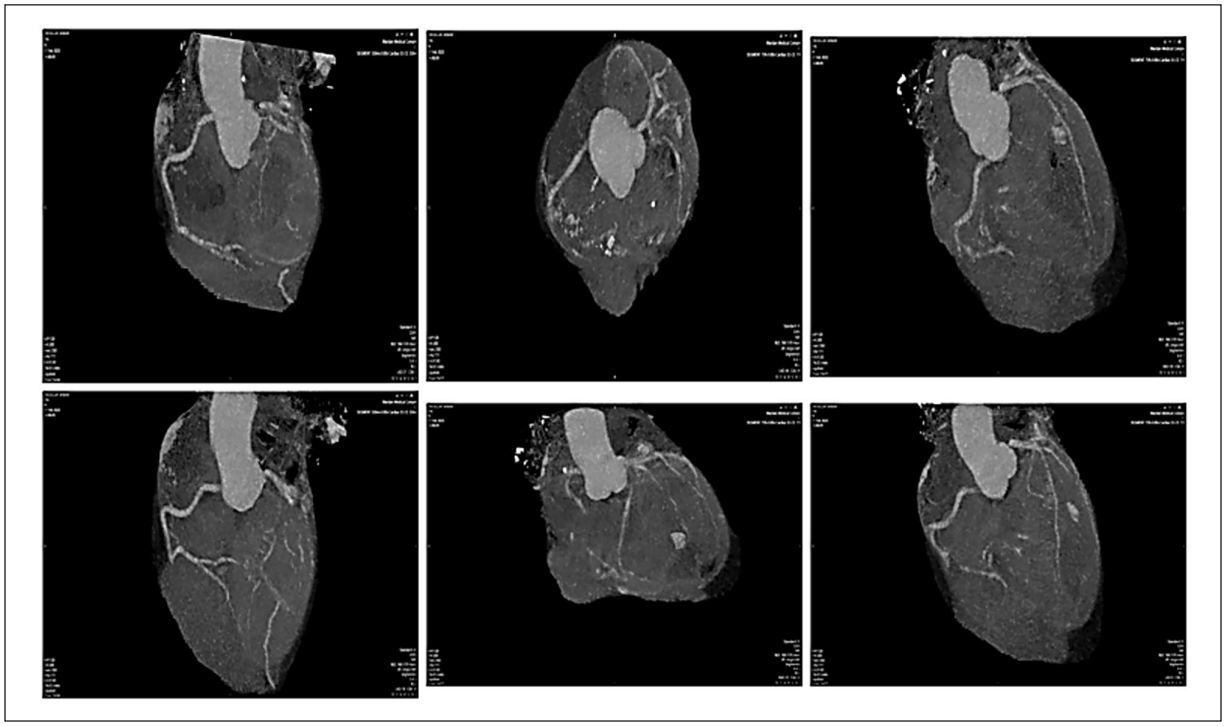

Figure 3. CT angiogram of the patient showing normal left coronary artery, left circumflex artery, and right coronary artery.

analysis showed no hypoxia, and the erythropoietin level was low at 1.8 mIU/ml (normal range: 3.3–16.6 mIU/ml). Janus Kinase 2 gene (JAK2) exon 12 mutations were present, while the autoimmune profile was negative, and abdominal ultrasound results were normal. Echocardiography revealed inferior segment hypokinesia, decent left ventricular systolic function with a 64% ejection fraction, and no signs of intracardiac thrombus.

Based on the clinical manifestation and blood reports, the patient was diagnosed with PV, meeting the World Health Organization (WHO) diagnostic criteria. Therefore, additional biopsy testing was deemed unnecessary. Hydroxyurea and 75 mg of aspirin were initiated after consultation with the internal medicine department on the third day following streptokinase administration. The patient demonstrated improvement without complications throughout the hospital stay and the initial follow-up. A CT coronary angiography conducted 2 weeks later revealed healthy coronary arteries (Figure 3).

Discussion

PV has been primarily recognized for its effect on red, white, and platelet cell proliferation, and is typically caused by alterations in the JAK2 gene.[6] However, the association between PV and subsequent blood hyperviscosity with thrombotic events, particularly coronary events, is increasingly being recognized. JAK2 gene alterations seem not to be the only factor contributing to PV's involvement in coronary events; literature reporting JAK2-negative PV patients developing STEMI[7] also hints at other hematological factors involved in PV, such as blood hyperviscosity, as potential factors in the causal pathway of myeloproliferative neoplasms and coronary events.

The literature offers several relevant case reports that serve to contextualize our findings, each bringing unique aspects to the fore. Hirsch et al. reported a case of PV presenting as STEMI, highlighting the diverse clinical presentations associated with these myeloproliferative neoplasms.[8] In contrast, Davis et al. detailed a case where PV led to cardiac arrest, emphasizing the need for novel management strategies in such critical situations.[9] Additionally, Rattarittamrong et al. presented a unique case of acute non-atherosclerotic STEMI in an adolescent with concurrent hemoglobin H-Constant Spring disease and PV, expanding the understanding of the disease spectrum.[10]

Our case contributes to this evolving narrative, providing further insights into the diagnosis and management of PV-related acute MI. The utilization of WHO criteria for diagnosis, incorporating both major and minor criteria, aligns with the established guidelines.[11] However, the scarcity of a standardized approach to acute MI management in PV patients necessitates a comprehensive review of existing literature.

One notable consideration is the management of streptokinase in PV patients experiencing acute MI. The literature lacks a consensus on this aspect, and our case prompts a critical reflection on the appropriate use of thrombolytic agents. The case by Davis et al. emphasizes the need for novel management strategies, possibly reflecting the diversity in clinical

approaches.[9] Our unique case, where PV manifested as an acute MI, adds a layer of intrigue, especially given the absence of any identified blockage during the CT angiography. This observation hints at the potential resolution of thrombus following streptokinase administration, presenting a distinctive aspect in the understanding of PV-associated cardiovascular events.

It is imperative to highlight the lessons learned from our case, especially in regions where catheterization lab might not be readily available. The successful management of our patient, in the absence of a standardized protocol, prompts a broader discussion on alternative strategies like utilization of other thrombolytic agents along with anticoagulants and antiplatelet. This becomes particularly relevant in scenarios where PCI may not be feasible, making our case a potential reference for clinicians facing similar challenges globally.

Conclusion

In summary, our case highlights the intricate connection between hematologic disorders, specifically polycythemia vera (PV), and cardiovascular events, such as acute MI. The patient's absence of typical cardiovascular risk factors underscores the need to consider less common causes in young individuals with MI symptoms. Our findings align with previous case reports, contributing to the understanding of the diverse clinical presentations of PV-related cardiovascular events. The complexity of PV, impacting red, white, and platelet cell proliferation, emphasizes the importance of a nuanced understanding of its association with thrombotic incidents, particularly in the coronary arteries. The challenges in diagnosing and managing PV-related acute MI call for a critical examination of therapeutic approaches. The absence of a standardized protocol necessitates a broader discussion on alternative thrombolytic agents, anticoagulants, and antiplatelets. Our case offers insights for clinicians globally, serving as a potential reference for effective management strategies in the absence of PCI availability.

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Authors contribution

S.R., J.K. made substantial contributions to the conception and design of the work. All authors (A.M.S., J.K., M.K.) were involved in drafting the work and revising it critically for important intellectual content and final approval of the version to be published. They agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Availability of data and material

All data underlying the results are available as part of the article and no additional source data are required.

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Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written Informed Consent was obtained from the patient for their anonymized information to be published in this article

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