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Dr. Hemant Gupta
Professor, Head, Department of Internal Medicine, MGM Medical College, Hospital and Research Centre, Vashi, Navi Mumbai, Maharashtra, India

Dr. Akash Parab
Assistant Professor, Department of Internal Medicine, MGM Medical College, Hospital and Research Centre, Vashi, Navi Mumbai, Maharashtra, India

Dr. Sahil Kubal
Assistant Professor, Department of Internal Medicine, MGM Medical College, Hospital and Research Centre, Vashi, Navi Mumbai, Maharashtra, India

Dr. Aishwarya Mokal
Intern, Office of the Medical Director, MGM Medical College, Hospital and Research Centre, Vashi, Navi Mumbai, Maharashtra, India

Dr. Panchami Pradeep Kumar
Senior Resident, Department of Internal Medicine, MGM Medical College, Hospital and Research Centre, Vashi, Navi Mumbai, Maharashtra, India

Corresponding Author:
Dr. Sahil Kubal
Assistant Professor, Department of Internal Medicine, MGM Medical College, Hospital and Research Centre, Vashi, Navi Mumbai, Maharashtra, India

A diagnostic challenge: Posterior wall myocardial infarction triggered by acute pyelonephritis and uremia in a chronic alcoholic patient

Hemant Gupta, Akash Parab, Sahil Kubal, Aishwarya Mokal and Panchami Pradeep Kumar

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Abstract
A 67-year-old man with long-standing diabetes, hypertension, chronic kidney disease, and heavy alcohol use presented with fever, flank pain, dysuria and myalgia. He was diagnosed with acute pyelonephritis and treated with intravenous antibiotics. Initial vitals were stable, but nine hours later he developed profound bradycardia (30 bpm) and severe hypoxemia (SpO_2 47% on room air). ECG revealed marked ST-segment depression in leads V4-V6, consistent with a posterior wall infarction. Laboratory tests showed severe uremia (BUN 66.2 mg/dL, creatinine 8.74 mg/dL) and hyperkalemia. He was managed with IV atropine, sodium bicarbonate and BiPAP, and subsequently stabilized. This case highlights how uremic metabolic derangements and sepsis from pyelonephritis can precipitate type 2 myocardial infarction by creating an acute imbalance of myocardial oxygen supply and demand [1]. We review the diagnostic considerations and pathophysiological mechanisms linking uremia, systemic infection and posterior wall infarction.

Keywords: Uremia, pyelonephritis, type 2 myocardial ınfarction, hyperkalemia, sepsis

Introduction

Posterior myocardial infarction (MI) is relatively uncommon and often underdiagnosed because standard 12-lead ECG does not directly image the posterior heart wall [2]. Instead of the typical ST-elevation seen in other infarcts, isolated posterior MI produces reciprocal ST-segment depressions in the anterior precordial leads (especially V1-V3) [3]. For example, Figure 1 shows an ECG with horizontal ST depressions in V1-V3 and tall R-waves (arrows), the "mirror image" of posterior ST-elevations [4]. Such subtle ECG changes make posterior MI a diagnostic challenge [5, 6].

Acute pyelonephritis (APN) is a severe bacterial kidney infection that classically presents with fever, flank pain, and urinary symptoms [8]. Complicated APN can progress to sepsis and multi-organ failure [9]. Chronic kidney disease (CKD) carries an enormous cardiovascular burden; CKD and end-stage renal disease (ESRD) patients have a 5-10-fold increased risk of cardiovascular disease compared to age-matched controls [10]. This excess risk is only partly explained by traditional factors (hypertension, diabetes) and is largely driven by nontraditional CKD-related factors such as uremic toxins, chronic inflammation, oxidative stress and vascular calcification [11]. These factors accelerate atherosclerosis and destabilize plaques in uremia [12].

During severe systemic infection (sepsis), inflammatory mediators (e.g. TNF-α, IL-1) and endothelium-derived factors impair coronary perfusion and myocardial contractility [13]. Sepsis-induced cardiac dysfunction is well-recognized and often reversible, but it simultaneously increases myocardial oxygen demand (due to fever and tachycardia) while reducing effective supply (via hypotension, microvascular dysfunction) [14]. In this setting, even modest coronary disease can precipitate ischemia. The concept of a type 2 myocardial infarction (MI) - myocardial necrosis from a supply-demand imbalance without acute plaque rupture - is increasingly applied to such cases [15].

Here we report a posterior wall infarction in an elderly alcoholic diabetic with APN and uremia, emphasizing the interplay of infection, uremic metabolic derangements, and pre-existing cardiovascular risk. The case underscores the diagnostic challenge: pyelonephritis symptoms can mask cardiac ischemia, and uremic ECG changes or hyperkalemia may mimic or precipitate infarction [16]. We review mechanisms by which acute pyelonephritis and uremia can jointly trigger myocardial ischemia, and discuss the clinical implications.

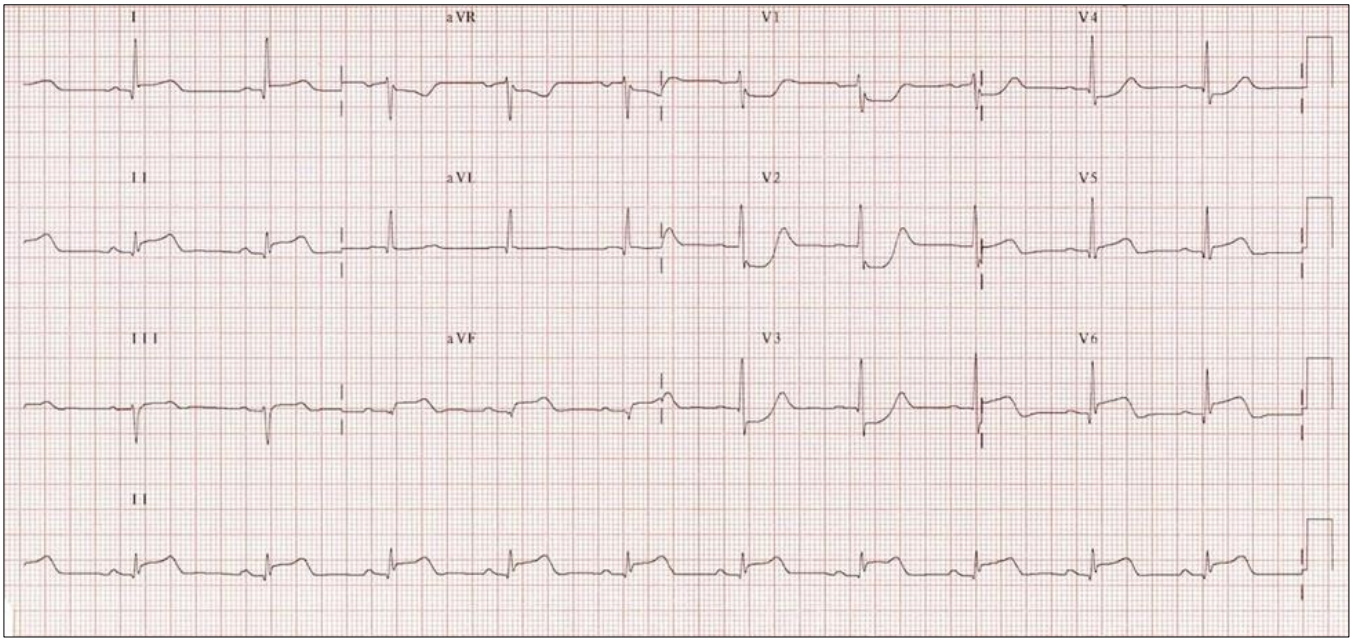

Fig 1: Example 12-lead ECG in posterior MI, demonstrating horizontal ST depressions in leads V1-V3 (arrows) with large R-waves - a classic reciprocal pattern of posterior wall infarction [7].

Case Presentation

A 67-year-old male with a history of type 2 diabetes mellitus, chronic hypertension (10 years), and daily alcohol use (5 years) was admitted with 5 days of generalized weakness, myalgia, fever (101.6°F) and 5 days of burning micturition, frequency, and left flank pain. His records indicated stage 5 CKD on hemodialysis (3 sessions 2 months ago). On exam he was febrile and appeared fatigued but hemodynamically stable (heart rate 78 bpm, blood pressure 135/78 mmHg, SpO_2 96% on room air). No new chest pain or focal deficits were noted. A Foley catheter was in place and a left internal jugular central venous catheter was present for dialysis access.

Urinalysis showed pyuria and nitrites. Blood tests revealed leukocytosis (WBC 15.2×10^9/L) and severe renal failure: BUN 66.2 mg/dL, serum creatinine 8.74 mg/dL, blood urea 358 mg/dL, uric acid 9.10 mg/dL. He was diagnosed with acute pyelonephritis. Empiric IV Cefoperazone-Sulbactam (1.5 g q12h) and Amikacin (250 mg q24h) were started, and intravenous fluids and insulin/dextrose protocol were initiated.

About 9 hours after admission, the patient acutely developed profound bradycardia (heart rate ~30 bpm) and his oxygen saturation fell to 47% on room air. An immediate 12-lead ECG showed deep ST-segment depressions in leads V4-V6 (Figure 1), consistent with a posterior wall ischemia (reciprocal changes). Arterial blood gas revealed hyperkalemia (estimated >6.5 mEq/L) with anion-gap metabolic acidosis. In response, he received IV atropine 0.5 mg (two doses), IV sodium bicarbonate (100 mEq), and BiPAP ventilation. By two hours later his heart rate improved to 70 bpm and SpO_2 rose to 92%. Serial ECG showed gradual resolution of the ST depressions. Over the next 24 hours he stabilized: renal acidosis improved, and repeat ECG showed T-wave inversion in V4-V6.

Case Discussion

This case illustrates several important issues. First, his CKD and uremic state predisposed to coronary disease: chronic renal failure causes accelerated atherosclerosis (via oxidative stress, inflammation, dyslipidemia and vascular calcification) and left ventricular hypertrophy [17]. Severe uremia by itself creates critical electrolyte and metabolic abnormalities. Uremic acidosis and hyperkalemia can dramatically impair cardiac conduction. Even mild hyperkalemia (>5 mEq/L) can depress pacemaker activity and slow conduction [18]. The combination of bradycardia, acidosis and volume overload - as seen in "BRASH syndrome" (bradycardia, renal failure, AV-nodal blockers, shock, hyperkalemia) - can precipitate cardiogenic shock [19]. In our patient, the rapid drop in heart rate to 30 bpm likely reflected uremic hyperkalemia and acidosis acting on the AV node. Aggressive IV bicarbonate (to correct acidosis) and atropine (to raise rate) are recommended in BRASH-like scenarios [20].

Large cohort analyses have documented that infections (including urinary tract infections) acutely elevate MI risk [21]. The proposed mechanisms include systemic inflammation activating macrophages in coronary plaques, endothelial dysfunction, and a prothrombotic state (with increased platelet activation and thromboxane) that can trigger plaque rupture (a Type I MI mechanism) [22]. Severe infection also imposes a high metabolic demand and may cause hypotension or hypoxemia, leading to oxygen supply-demand mismatch (Type II MI) [23]. In our patient, acute sepsis (fever, tachycardia, etc.) likely created a milieu for plaque destabilization or demand ischemia against a

backdrop of known atherosclerotic risk factors. By age 67 with diabetes, hypertension, and long-term alcohol use, he undoubtedly had underlying coronary artery disease risk factors [24]. Indeed, he had no prior known heart disease, but these comorbidities and acute stress tipped him into infarction.

Second, the presentation was atypical. The patient had no chest pain or classic anginal symptoms; instead, his collapse was marked by extreme bradycardia and hypoxia. Posterior wall MI often presents with subtle or "silent" symptoms, especially in diabetics. The ECG changes in posterior MI - ST depression in the anterior leads - are subtle and easily overlooked [25, 26]. In our patient, the ST depression appeared in V4-V6 (though more classically one sees it in V1-V3). This ECG finding suggests reciprocal changes to ST-elevation that would appear in posterior leads (V7-V9) if recorded. Notably, an isolated posterior infarct can be mistaken for non-diagnostic ischemic changes unless carefully considered [27, 28].

Third, the extreme bradycardia (HR ~30) is consistent with an inferior/posterior MI involving the right coronary artery (which often supplies the AV node). Inferior and inferoposterior MIs frequently cause AV nodal ischemia or vagal reflexes leading to bradyarrhythmias [29]. Up to 20% of inferior MIs develop second- or third-degree AV block [30]. The bradycardia in this setting is often transient and responds well to atropine [31], as we observed when the patient's rate improved after atropine administration. This response further supports the diagnosis of an infarction involving the AV node. The severe drop in oxygen saturation and need for BiPAP likely reflected acute pulmonary edema or cardiogenic shock from the large infarct.

The interplay of factors made diagnosis challenging. He lacked typical chest pain; his initial presentation was dominated by infection. Uremic toxins and electrolyte shifts can alter ECG morphology - making acute MI harder to spot. Clinicians should therefore maintain a high index of suspicion for myocardial ischemia in septic uremic patients with any cardiac changes. Continuous cardiac monitoring and serial ECGs are prudent in such high-risk settings.

Conclusion

This case illustrates a rare but instructive scenario: acute pyelonephritis and uremia precipitating a posterior wall myocardial infarction in a high-risk patient. It underscores that in CKD patients with infection, clinicians should vigilantly monitor for cardiac ischemia even in absence of chest pain, since sepsis and uremic toxins can acutely injure the heart [32]. Early ECG monitoring and correction of metabolic derangements (hyperkalemia, acidosis) are critical. The concept of type 2 MI is central: management focuses on stabilizing hemodynamics and treating the underlying triggers rather than routine invasive reperfusion. Finally, it highlights how uremia itself accelerates atherosclerosis and blunts physiological reserve [33]. Awareness of this mechanistic link may prompt earlier cardiac evaluation and tailored therapy in similar patients.

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