

## CASE REPORT

# An Unusual Coexistence of Single Vessel Coronary Artery Disease and Hypokalaemia Periodic Paralysis

Girish Kulle<sup>1</sup>, Anita Rawat<sup>2</sup>, Kishalay Datta<sup>3</sup>**HOW TO CITE THIS ARTICLE:**

*Girish Kulle, Anita Rawat, Kishalay Datta.* An Unusual Coexistence of Single Vessel Coronary Artery Disease and Hypokalaemia Periodic Paralysis. *Ind J Emerg Med.* 2026; 12(2): 92-96.

**ABSTRACT**

We present a rare case highlighting the unusual coexistence of single vessel coronary artery disease (CAD) and hypokalemic periodic paralysis (HPP) in a middle-aged male. The patient presented to the emergency department with acute onset chest discomfort, generalized muscle weakness, and difficulty in mobility. Initial evaluation revealed hypokalemia, confirmed on serum electrolyte analysis, alongside electrocardiographic changes suggestive of myocardial ischemia. Subsequent coronary angiography demonstrated significant stenosis of a single major coronary artery. HPP, a channelopathy characterized by recurrent episodes of muscle weakness due to low serum potassium, is rarely reported in conjunction with structural cardiac disease such as CAD. This unique presentation posed a diagnostic and therapeutic challenge, as both conditions required simultaneous yet cautious management to avoid exacerbation of either pathology. Potassium replacement therapy resulted in complete neurological recovery, while percutaneous coronary intervention addressed the underlying ischemic burden. This case underscores the importance of considering concurrent metabolic and structural cardiac disorders in patients with atypical presentations, and highlights the need for integrated, multidisciplinary management to optimize outcomes. To our knowledge, documented reports of such coexistence remain scarce, making this case a valuable contribution to the literature.

**KEYWORDS**

• Coronary Artery Disease • Hypokalaemia Periodic Paralysis

**AUTHOR'S AFFILIATION:**

<sup>1</sup> Resident, Department of Emergency Medicine, Max Super Speciality Hospital, Shalimar Bagh, New Delhi, India.

<sup>2</sup> Principal Consultant, Emergency Medicine, Max Super Speciality Hospital, Shalimar Bagh, New Delhi, India.

<sup>3</sup> HOD and Director, Emergency Medicine, Max Super Speciality Hospital, Shalimar Bagh, New Delhi, India.

**CORRESPONDING AUTHOR:**

**Girish Kulle**, Resident, Department of Emergency Medicine, Max Super Speciality Hospital, Shalimar Bagh, New Delhi, India.

E-mail: gkulle108@gmail.com

Received : 17-01-2026

Accepted : 20-02-2026



## INTRODUCTION

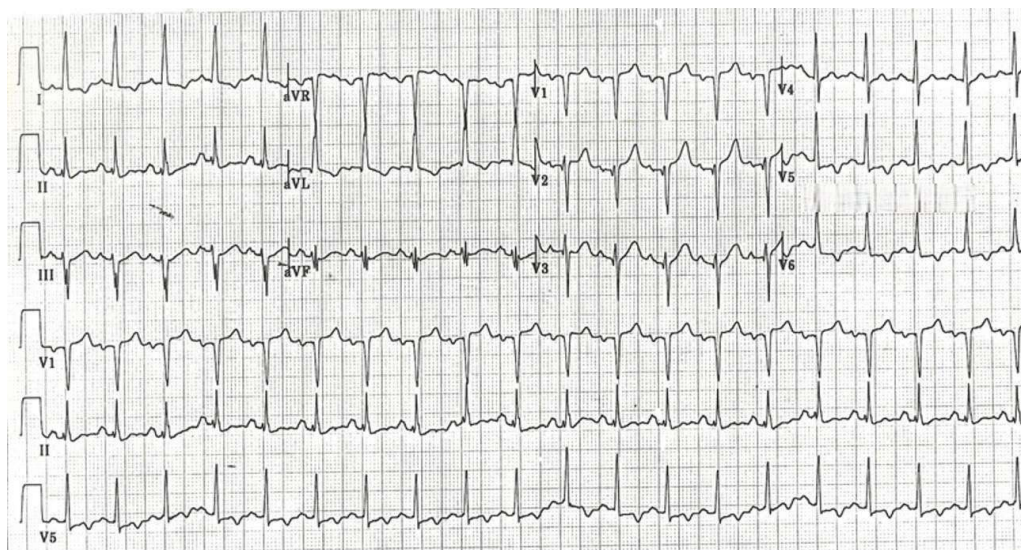
Hypokalemic periodic paralysis (HPP) is a rare disorder marked by sudden onset flaccid muscle weakness associated with low serum potassium levels, often precipitated by carbohydrate-rich meals, strenuous exercise, or physiological stress.<sup>1,2</sup> Coronary artery disease (CAD), in contrast, is a prevalent atherosclerotic condition responsible for significant cardiovascular morbidity and mortality worldwide.<sup>3</sup> The simultaneous occurrence of HPP and CAD is rarely documented. In such cases, diagnostic confusion can arise, as muscle weakness in CAD patients may be attributed to fatigue or low cardiac output. Moreover, unrecognized hypokalemia in acute coronary syndrome (ACS) can exacerbate arrhythmia risk during interventions. We report a case of single vessel CAD coexisting with HPP, where acute coronary ischemia acted as the likely trigger for a paralytic episode.

## CASE

A 52-year-old male, known hypertensive on amlodipine 5 mg daily, non-smoker, presented to the emergency department with sudden-onset retrosternal chest pain lasting 45 minutes, associated with profuse sweating. About 30

minutes later, he developed weakness in both lower limbs and mild weakness in the upper limbs. There was no history of preceding fever, diarrhoea, or excessive exertion. On examination in the emergency department, the patient was alert and oriented. His initial vital signs in the ED: heart rate 88 beats per minute, blood pressure 140/90 mmHg, respiratory rate 18 breaths per minute, oxygen saturation 98% on room air, temperature 36.7°C. The primary survey for the patient was not significant (ABCDE). Neurological examination was showed flaccid paraparesis (MRC grade 2/5 in both lower limbs) and mild upper limb weakness (4/5), absent deep tendon reflexes in the legs, preserved cranial nerve function. Electrocardiography was done (image). An urgent MRI brain scan was advised with a differential diagnosis of any intracranial pathology (cerebrovascular accident - Stroke), which came out to be normal.

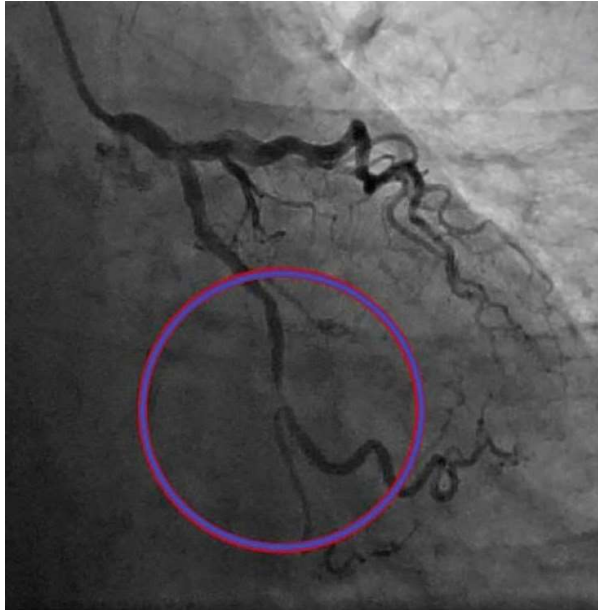
Laboratory investigation was sent with a main focus on serum electrolytes and troponin I levels, which, Troponin-I: 0.64 ng/mL, serum potassium: 2.4 mEq/L (3.5-5.5), serum sodium 139 mEq/L, serum calcium 9.1 mg/dL, serum magnesium 1.9 mg/dL. 2D echocardiography was done - Mild anterolateral wall hypokinesia, LVEF 50%. Patient was now diagnosed with



**Figure 1:** Electrocardiography findings showing changes in lead V5 and V6  
(Image Courtesy: Department of Emergency Medicine, Max SSH SHBG, New Delhi)

The patient received intravenous potassium chloride (40 mEq over 4 hours) with continuous cardiac monitoring. Serum Potassium normalized to 4.0 mEq/L, and

weakness resolved within 6 hours. The patient was then shifted to the cath lab for PCI. Drug-eluting stent to LAD was performed without periprocedural arrhythmia (image).



**Figure A:** Blocked distal left anterior descending artery  
(Image Courtesy: Department of Interventional Cardiology, Max SHBG, New Delhi)

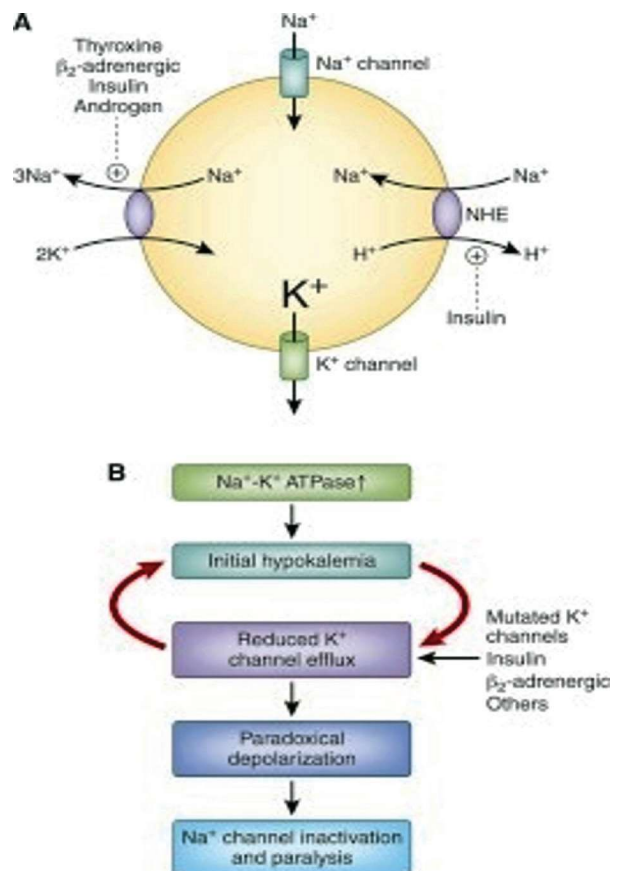


**Figure B:** Post PCI therapy to the patient  
(Image Courtesy: Department of Interventional Cardiology, Max SHBG, New Delhi)

The patient recovered well. He was discharged on the 3<sup>rd</sup> day after admission with complete recovery from the limb weakness and chest heaviness. His serum potassium level continued to be stable over 3 days. The patient was discharged on aspirin, clopidogrel, atorvastatin 40 mg, metoprolol 25 mg, and potassium-rich diet advice. At 2-week follow-up, he remained asymptomatic with serum potassium 4.2 mEq/L.

## DISCUSSION

Hypokalemic periodic paralysis (HPP) is a neuromuscular disorder that can be classified as either primary resulting from inherited ion channel dysfunction (channelopathy) or secondary, arising from underlying endocrine disturbances, renal tubular disorders, or other systemic causes.<sup>4</sup> The pathophysiological hallmark of HPP is a marked reduction in serum potassium, which induces hyperpolarization of the skeletal muscle cell membrane.<sup>5</sup> This hyperpolarization diminishes muscle fiber excitability, thereby impairing action potential generation and producing episodes of transient, flaccid weakness.<sup>5,6</sup>



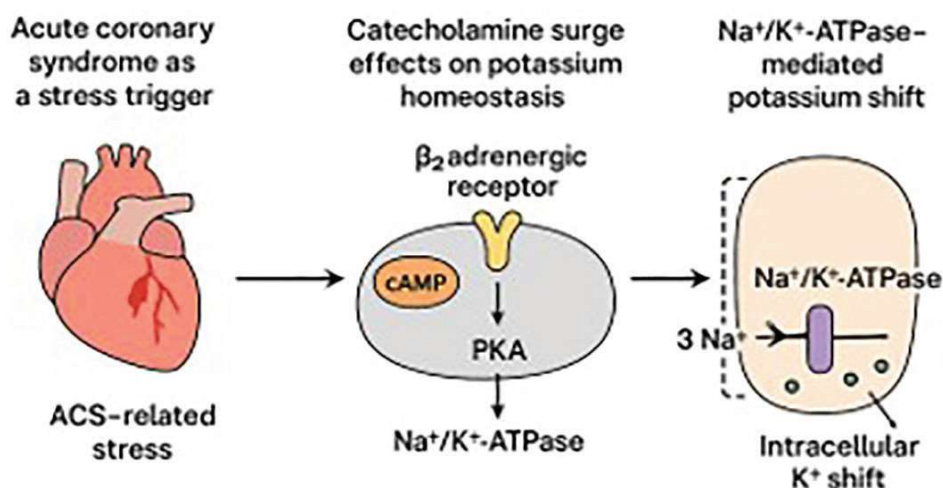
Source: Google Search Engine

In the present case, it is plausible that acute myocardial ischemia served as the precipitating factor for HPP. Acute coronary events trigger a surge in catecholamines and stress hormones, which, along with elevated insulin levels, can drive potassium into cells via  $\text{Na}^+/\text{K}^+$ -ATPase activity, precipitating a rapid fall in extracellular potassium. This intracellular shift may explain the abrupt onset of generalized weakness coinciding with the cardiac event.<sup>7</sup>

While coronary artery disease (CAD) is relatively common in middle-aged and older populations, its association with HPP remains distinctly uncommon. In acute coronary syndrome (ACS), profound weakness is more often attributed to hemodynamic instability, low cardiac output, or concurrent cerebrovascular compromise. Consequently, metabolic etiologies such as severe hypokalemia may be under-recognized in the acute phase, potentially delaying diagnosis and appropriate

treatment. This oversight carries significant implications, as uncorrected hypokalemia during ACS is a well-established risk factor for malignant ventricular arrhythmias, conduction abnormalities, and increased in-hospital mortality.<sup>7</sup> Hence, rapid identification and correction of potassium imbalance ideally prior to percutaneous coronary intervention (PCI) is essential to optimize both neuromuscular and cardiac outcomes.

### Pathophysiological link between ACS stress and hypokalemic periodic paralysis



Additionally, this case underscores the diagnostic challenge of differentiating HPP from other acute paralytic conditions in the emergency setting. Disorders such as Guillain-Barré syndrome, myasthenic crisis, and acute inflammatory myopathies may present with similar patterns of sudden weakness, but differ in onset kinetics, reflex status, and laboratory profiles.<sup>8</sup> A high index of suspicion, supported by targeted biochemical assessment, can guide early recognition of HPP, enabling timely correction of hypokalemia and prevention of potentially fatal cardiac arrhythmias.

### CONCLUSION

In CAD patients with acute weakness, clinicians should consider HPP as a potential diagnosis, especially in the presence of unexplained hypokalemia. Prompt correction of potassium before cardiac intervention can reduce arrhythmic risk and improve outcomes.

### Learning Points

1. Acute coronary events can precipitate HPP in susceptible individuals.
2. Hypokalemia should be corrected before PCI to prevent arrhythmias.
3. Muscle weakness in ACS patients should prompt evaluation for metabolic causes.

### REFERENCES

1. Fontaine B. Periodic paralysis. *Adv Genet.* 2008; 63: 3–23.
2. Statland J.M., Fontaine B., Hanna M.G., Johnson N.E., Kissel J.T., Sansone V.A., *et al.* Review of the diagnosis and treatment of periodic paralysis. *Muscle Nerve.* 2018; 57(4): 522–530.
3. Virani S.S., Alonso A., Aparicio H.J., Benjamin E.J., Bittencourt M.S., Callaway C.W., *et al.* Heart disease and stroke statistics—2021 update. *Circulation.* 2021; 143(8): e254–e743.

4. Lin S.H., Lin Y.F., Halperin M.L. Hypokalemia and paralysis. *QJM*. 2001; 94(3): 133-139.
5. Lehmann-Horn F., Jurkat-Rott K. Voltage-gated ion channels and hereditary disease. *Physiol Rev*. 1999; 79(4): 1317-1372.
6. Clausen T. Hormonal and pharmacological modification of plasma potassium homeostasis. *Fundam Clin Pharmacol*. 2010; 24(5): 595-605.
7. Goyal A., Spertus J.A., Gosch K., Venkitachalam L., Jones P.G., Van den Berghe G., *et al*. Serum potassium levels and mortality in acute myocardial infarction. *JAMA*. 2012; 307(2): 157-164.
8. Finsterer J. Differential diagnosis of weakness. *Acta Neurol Scand*. 2007; 115(2): 72-81.