Section: Cardiology

Case Series

FROM CONGESTION TO CATASTROPHE: FUROSEMIDE-INDUCED ELECTROLYTE DERANGEMENT CAUSING TORSADES DE POINTES IN ACS-HFREF" -CASE SERIES

Kalyan Munde[1], Anant Munde[2], Suvarna Thorat[3], Sandip Ghoti[3], Jayakrishna Niari[2], Samkit Mutha[2], Hariom Kolapkar[2]

[1]Head of Department, Department of Cardiology, GGMC and Sir JJ Hospital, Mumbai, Maharashtra, INDIA
[2]Assistant Professor, Department of Cardiology, GGMC and Sir JJ Hospital, Mumbai, Maharashtra, India
[3]Senior Resident, Department of Cardiology, GGMC and Sir JJ Hospital, Mumbai, Maharashtra, India

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Corresponding Author:
Dr. Suvarna Thorat,
Email: sandipghoti4@gmail.com

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ABSTRACT

Background: Patients with acute coronary syndrome (ACS) complicated by heart failure with reduced ejection fraction (HFrEF) are at increased risk for malignant ventricular arrhythmias due to ischemia-related electrical instability and impaired repolarization reserve. Loop diuretics are frequently required for the management of congestion; however, furosemide-induced renal potassium and magnesium wasting may produce an acquired electrolyte phenotype resembling Bartter or Gitelman syndromes. In the proarrhythmic milieu of ACS and HFrEF, this disturbance can precipitate QT prolongation and torsades de pointes (TdP). **Case Summary:** We report Five patients with ACS and HFrEF who developed severe hypokalemia and hypomagnesemia during furosemide therapy, resulting in marked QT prolongation and TdP. All patients were successfully treated with prompt rhythm stabilization, intravenous magnesium, aggressive potassium repletion, withdrawal or modification of precipitating factors, and heart-rate augmentation when required. No patient experienced arrhythmia recurrence after correction of electrolyte abnormalities. **Conclusion:** In patients with ACS and HFrEF, the occurrence of TdP should prompt immediate evaluation for diuretic-induced electrolyte depletion representing a reversible acquired pseudo–Bartter/Gitelman syndrome. Early recognition and targeted correction are lifesaving and may prevent unnecessary long-term device therapy.

INTRODUCTION

Acute coronary syndrome (ACS) is a well-recognized trigger for malignant ventricular arrhythmias owing to myocardial ischemia, autonomic imbalance, altered calcium handling, and increased dispersion of repolarization.[1,2] This arrhythmic risk is further amplified in patients with heart failure with reduced ejection fraction (HFrEF), in whom ventricular remodeling, fibrosis, and down-regulation of potassium channels reduce repolarization reserve and facilitate polymorphic ventricular tachycardia.[1,2] Loop diuretics, particularly furosemide, remain a cornerstone of therapy for congestion in ACS complicated by HFrEF. By inhibiting the $Na^+ –K^+ –2Cl^-$ cotransporter in the thick ascending limb of Henle, furosemide promotes natriuresis but also causes renal potassium and magnesium wasting.[3-8] Sustained or high-dose therapy may result in hypokalemia, hypomagnesemia, metabolic alkalosis, and secondary hyperaldosteronism—closely mimicking inherited Bartter or Gitelman syndromes.[6,7] When this acquired electrolyte disturbance occurs in the setting of ACS and HFrEF, it creates a potent substrate for QT prolongation and torsades de pointes (TdP).[3,5] Despite the widespread use of loop diuretics in this population, this mechanism remains under recognized. We present a case series highlighting this reversible but potentially fatal entity.

CASE PRESENTATION

Case 1

A 66-year-old woman presented with non–ST-elevation myocardial infarction and acute decompensated heart failure. Transthoracic echocardiography revealed a left ventricular ejection fraction (LVEF) of 30%. She was treated with

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guideline-directed ACS therapy and intravenous furosemide. Forty-eight hours later, she developed recurrent syncope. Telemetry demonstrated polymorphic ventricular tachycardia consistent with Torsades de pointes. Electrocardiography showed QTc prolongation to 560 ms (by Bazett formula). Serum potassium was 2.7 mmol/L and magnesium 1.2 mg/dL. She required immediate defibrillation followed by intravenous magnesium sulfate and aggressive potassium repletion, in accordance with guideline-recommended management.[1,3,4] Furosemide dosage was reduced and QT-prolonging medications were discontinued. No further ventricular arrhythmias occurred after electrolyte normalization.

Case 2

A 58-year-old man with inferior wall ST-elevation myocardial infarction and HFrEF (LVEF 28%) developed pulmonary edema requiring high-dose loop diuretics. He subsequently experienced recurrent non-sustained polymorphic ventricular tachycardia initiated by long–short RR sequences, characteristic of pause-dependent TdP.[1,3] QTc was prolonged to 540 ms. Serum potassium was 2.9 mmol/L and magnesium 1.4 mg/dL, consistent with an acquired Gitelman-like electrolyte pattern.[6] Intravenous magnesium and potassium repletion were initiated. Due to bradycardia-dependent TdP, isoproterenol infusion was used for heart-rate augmentation, resulting in complete arrhythmia suppression.[1,3]

Case 3

A 42-year-old woman with ACS and baseline sinus bradycardia had HFrEF (LVEF 32%) and was treated with escalating doses of oral furosemide. She suffered sudden cardiac arrest due to torsades de pointes and was successfully resuscitated. Post-resuscitation electrocardiography demonstrated marked QTc prolongation to 600 ms. Laboratory evaluation revealed potassium 2.5 mmol/L and magnesium 1.0 mg/dL, consistent with acquired pseudo-Bartter syndrome.[7] She was treated with defibrillation, intravenous magnesium, aggressive potassium repletion, and temporary transvenous overdrive pacing. No recurrence of ventricular arrhythmias occurred following electrolyte normalization

Case 4

A 61-year-old man presented with anterior wall ST-elevation myocardial infarction complicated by acute pulmonary edema and cardiogenic shock. Transthoracic echocardiography demonstrated severe left ventricular systolic dysfunction with an LVEF of 25%. He was managed with primary percutaneous coronary intervention, inotropic support, and high-dose intravenous furosemide for refractory congestion. On day 3 of hospitalization, the patient developed recurrent presyncope with documented episodes of polymorphic ventricular tachycardia on telemetry. Electrocardiography revealed marked QTc prolongation to 570 ms. Laboratory evaluation showed severe hypokalemia (2.8 mmol/L) and hypomagnesemia (1.3 mg/dL), consistent with an acquired Bartter-like electrolyte pattern secondary to loop diuretic therapy. Immediate treatment included intravenous magnesium sulfate, aggressive potassium replacement, discontinuation of QT-prolonging agents, and reduction of loop diuretic dose. Due to recurrent pause-dependent episodes, temporary transvenous overdrive pacing was instituted, resulting in complete suppression of Torsades de pointes. Following normalization of electrolytes, the QT interval shortened, and no further ventricular arrhythmias were observed.

Case 5

A 54-year-old woman with non–ST-elevation acute coronary syndrome and a background of hypertension presented with worsening dyspnea and volume overload. Echocardiography demonstrated global left ventricular hypokinesia with an LVEF of 35%. She was treated with guideline-directed medical therapy and intravenous furosemide for decongestion. Seventy-two hours after initiation of diuretic therapy, she experienced sudden syncope. Continuous monitoring revealed self-terminating runs of polymorphic ventricular tachycardia consistent with torsades de pointes. Twelve-lead electrocardiography demonstrated sinus rhythm with profound QTc prolongation to 585 ms. Serum biochemistry revealed potassium of 2.6 mmol/L and magnesium of 1.1 mg/dL, consistent with an acquired Gitelman-like electrolyte disturbance. She was managed with immediate intravenous magnesium sulfate, high-dose potassium repletion, and withdrawal of furosemide. Given underlying sinus bradycardia and recurrent pause-dependent arrhythmia, isoproterenol infusion was initiated for heart-rate augmentation, leading to prompt arrhythmia resolution. No recurrence of torsades de pointes occurred after correction of electrolyte abnormalities, and QTc normalized prior to discharge.

ECG 1. Telemetry Strip Showing Recurrent Torsades Episodes

The rhythm strip demonstrates recurrent, self-terminating episodes of polymorphic ventricular tachycardia with waxing and waning QRS amplitude. This indicates an electrically unstable myocardium with a persistent arrhythmogenic substrate, typical of ongoing hypokalemia and hypomagnesemia.

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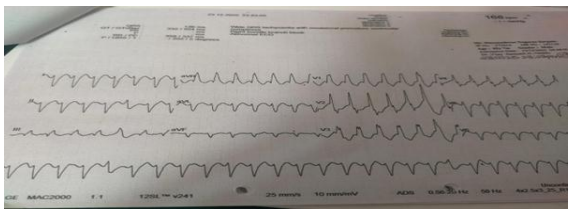
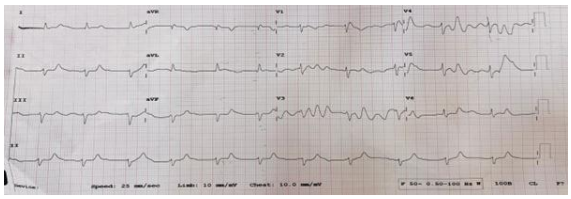
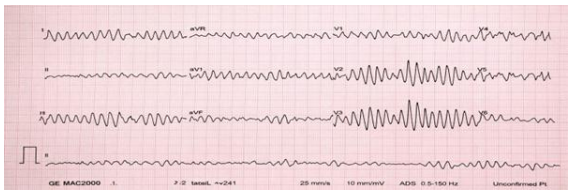


ECG 2. Polymorphic Ventricular Tachycardia (Torsades de Pointes)
This ECG shows rapid polymorphic wide-complex tachycardia with continuous beat-to-beat variation in QRS morphology and axis, characteristic of torsades de pointes. The pattern reflects triggered ventricular activity arising in the setting of prolonged repolarization due to electrolyte imbalance



ECG 3. Sinus Rhythm With Marked QT Prolongation
This ECG shows intermittent torsades de pointes which later lead to malignant ventricular arrhythmias.



ECG 4 Electrocardiogram showing torsades de pointes, characterized by polymorphic wide-complex ventricular tachycardia with beat-to-beat variation in QRS morphology and a waxing–waning amplitude pattern ("twisting of the points").

Table 1: ?

Case	Age / Sex	ACS Type	LVEF (%)	Diuretic Exposure	K^+ (mmol/L)	Mg^{2+} (mg/dL)	QTc (ms)	TdP Trigger	Acute Management	Outcome
1	66 / F	NSTEMI	30	IV furosemide	2.7	1.2	560	Electrolyte depletion	Defibrillation, IV Mg, K^+	No recurrence
2	58 / M	STEMI (IWMI)	28	High-dose IV furosemide	2.9	1.4	540	Bradycardia, long–short RR	IV Mg, K^+ , isoproterenol	No recurrence
3	42 / F	ACS	32	Escalating oral furosemide	2.5	1.0	600	Severe electrolyte loss	Defibrillation, IV Mg, K^+ , pacing	No recurrence
4	61 / M	STEMI (AWMI)	25	High-dose IV furosemide	2.8	1.3	570	Pause-dependent TdP	IV Mg, K^+ , overdrive pacing	No recurrence
5	54 / F	NSTEMI	35	IV furosemide	2.6	1.1	585	Bradycardia + electrolyte loss	IV Mg, K^+ , isoproterenol	No recurrence

DISCUSSION

Torsades de pointes in patients with ACS and HFrEF represents the culmination of a complex interaction between ischemia, structural heart disease, autonomic influences, pharmacologic exposures, and electrolyte disturbances.[1-3] This case series highlights an under-recognized but clinically critical mechanism: furosemide-induced acquired pseudo–Bartter/Gitelman syndrome, which markedly lowers the threshold for malignant ventricular arrhythmias.

Reduced Repolarization Reserve: Repolarization reserve reflects the redundancy of outward potassium currents that stabilize ventricular repolarization.[1,2] In HFrEF, this reserve is intrinsically reduced due to ventricular remodeling, fibrosis, and down-regulation of potassium channels. Consequently, superimposed insults—such as hypokalemia, hypomagnesemia, ischemia, or QT-prolonging drugs—can precipitate marked QT prolongation and TdP.[1,2]

Electrolyte Abnormalities as Primary Triggers: Hypokalemia suppresses IKr and IK1 currents, prolonging action potential duration and promoting early afterdepolarizations.[3,9] Hypomagnesemia further destabilizes myocardial electrophysiology by impairing Na^+ –K^+ ATPase function, facilitating intracellular potassium loss, and enhancing calcium influx.[8,9] These combined effects create a highly arrhythmogenic substrate.[3,4] Importantly, serum magnesium levels may underestimate total body deficiency, explaining the consistent efficacy of intravenous magnesium in terminating TdP regardless of baseline levels.[3,4]

Acquired Pseudo–Bartter/Gitelman Syndrome
Loop diuretics reproduce key features of inherited Bartter syndrome by blocking the Na^+ -K^+ 2-Cl^- cotransporter, resulting in renal salt wasting, hypokalemia, metabolic alkalosis, and secondary hyperaldosteronism.[6,7] Chronic exposure also impairs distal tubular magnesium reabsorption, creating a Gitelman-like phenotype.[6] Unlike genetic tubulopathies, this acquired condition is fully

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reversible, emphasizing the importance of early recognition.

Pause-Dependent and Bradycardia-Mediated TdP: TdP is classically pause-dependent and initiated by long–short RR sequences.[1,3] Bradycardia—due to inferior wall ischemia, enhanced vagal tone, or β-blocker therapy—exaggerates QT prolongation and facilitates early afterdepolarizations. Suppression of arrhythmia with isoproterenol or overdrive pacing in our patients confirms the central role of rate-dependent repolarization dynamics.[1,3]

Ischemia and Structural Substrate: Acute myocardial ischemia alters ionic gradients, impairs ATP-dependent ion pumps, and increases dispersion of repolarization, promoting polymorphic ventricular arrhythmias.[1,2] In HFrEF, structural remodeling and fibrosis further amplify this vulnerability, explaining why TdP may occur with electrolyte abnormalities that might otherwise be tolerated in structurally normal hearts.[1,2]

Drug and Autonomic Influences: QT-prolonging medications synergize with electrolyte abnormalities to increase TdP risk.[3,5] Importantly, polymorphic VT in this context should not be reflexively treated with amiodarone, which may worsen QT prolongation.[1,5] Lidocaine is preferred if pharmacologic therapy is required.[1]

Therapeutic and Prognostic Implications: These cases demonstrate that TdP in ACS with HFrEF is frequently reversible. Management should focus on immediate arrhythmia termination, aggressive electrolyte correction, heart- rate augmentation, and removal of precipitating factors.[1-4] Implantable cardioverter- defibrillator implantation is not indicated once reversible causes are corrected, in accordance

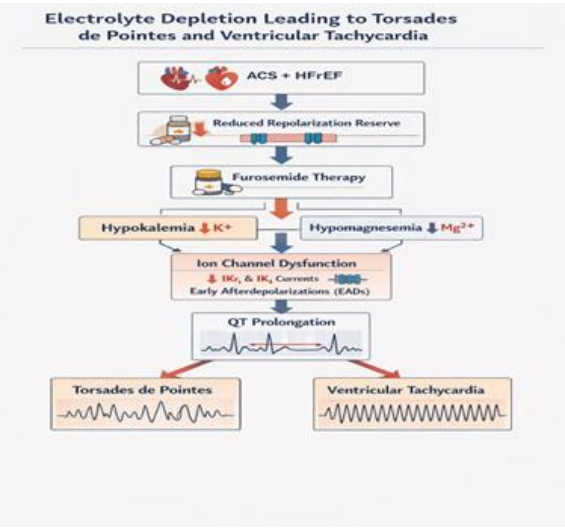

Figure 1: Mechanism of Electrolyte-Mediated Ventricular Arrhythmogenesis

Loop diuretic–induced hypokalemia and hypomagnesemia reduce IKr-mediated repolarizing currents and impair Na^+ /K^+ -ATPase activity, leading to prolonged ventricular action potential duration and QT interval prolongation.

Hypomagnesemia facilitates L-type Ca^{2+} channel reactivation and sustains potassium depletion, promoting early after-depolarizations.

In the setting of reduced repolarization reserve (acute ischemia and HFrEF), these triggers initiate pause-dependent polymorphic ventricular tachycardia (torsades de pointes), which may degenerate into sustained VT or ventricular fibrillation.[1-3,10-12]

Treatment and Acute Management Strategy: The management of torsades de pointes (TdP) in patients with acute coronary syndrome (ACS) and heart failure with reduced ejection fraction (HFrEF) requires a structured, stepwise, and mechanism-based approach, targeting both immediate arrhythmia termination and reversal of the underlying electrophysiological substrate. Given that TdP in this setting is typically functional and reversible, prompt recognition and appropriate intervention are often curative and prevent recurrence.[1-3]

Immediate Recognition and Hemodynamic Stabilization: Torsades de pointes is a polymorphic ventricular tachycardia that may be self-terminating or degenerate into ventricular fibrillation. In patients presenting with hemodynamic instability, syncope, or cardiac arrest, immediate electrical defibrillation is mandatory and lifesaving.[1,2] Early defibrillation interrupts recurrent TdP episodes and restores organized cardiac rhythm. Continuous cardiac monitoring is essential, as TdP is often episodic and pause-dependent.[3]

Intravenous Magnesium Sulfate as First-Line Therapy: Intravenous magnesium sulfate is the cornerstone of acute TdP management, regardless of baseline serum magnesium levels.[3,4] Magnesium suppresses early after-depolarizations by stabilizing myocardial cell membranes, modulating L-type calcium channel activity, and reducing triggered activity.[4,9] A bolus of 2 g intravenous magnesium sulfate administered over 10–15 minutes is recommended, followed by a continuous infusion if recurrent TdP episodes occur.[3,4] Importantly, the antiarrhythmic benefit of magnesium extends beyond simple correction of hypomagnesemia and reflects its direct electrophysiological effects.[4]

Aggressive Potassium Repletion and Restoration of Repolarization Reserve: Hypokalemia is a critical driver of QT prolongation and TdP. Reduced extracellular potassium diminishes outward potassium currents (particularly IKr and IK1), prolongs action potential duration, and promotes early afterdepolarizations.[3,9] Aggressive potassium repletion is therefore essential, with a target serum potassium in the upper-normal range (≥4.5–5.0 mmol/L).[3] Intravenous potassium chloride is preferred in patients with ongoing arrhythmia or hemodynamic instability, with close telemetry and serial electrolyte monitoring to avoid overcorrection. Restoration of potassium levels shortens the QT interval and markedly reduces arrhythmia recurrence.[3,9]

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Heart-Rate Augmentation for Pause-Dependent TdP: Torsades de pointes is classically pause-dependent and frequently initiated by long–short RR sequences, particularly in the presence of bradycardia.[3] Slower heart rates exaggerate QT prolongation and facilitate early afterdepolarizations. In patients with recurrent or bradycardia-dependent TdP, heart-rate acceleration is a critical therapeutic strategy. This can be achieved pharmacologically using isoproterenol infusion or mechanically with temporary transvenous overdrive pacing, targeting a heart rate of 90–110 beats per minute.[1,3] Heart-rate augmentation shortens repolarization duration, suppresses pauses, and effectively terminates TdP when electrolyte correction alone is insufficient.[3]

Withdrawal of QT-Prolonging Drugs and Antiarrhythmic Considerations: All QT-prolonging medications should be promptly discontinued, as drug-induced QT prolongation synergizes with electrolyte abnormalities to precipitate TdP.[5] Importantly, amiodarone should be avoided in polymorphic ventricular tachycardia associated with QT prolongation, as it may further lengthen repolarization and exacerbate TdP.[1,5] If antiarrhythmic therapy is required, lidocaine is preferred due to its neutral or QT- shortening electrophysiological effects.[1]

Optimization of Diuretic Therapy and Prevention of Recurrence: Because the arrhythmogenic substrate in these patients was driven by furosemide-induced potassium and magnesium wasting, modification of diuretic therapy is essential. Strategies include dose reduction, temporary cessation, or careful reintroduction at lower doses once congestion improves.[6-8] Long-term prevention requires scheduled electrolyte surveillance in all ACS–HFrEF patients receiving loop diuretics, particularly during the acute phase of illness. Oral potassium and magnesium supplementation should be considered in patients requiring ongoing diuretic therapy.[3,8]

Role of Implantable Cardioverter-Defibrillator Therapy: Despite the dramatic clinical presentation, implantable cardioverter-defibrillator (ICD) implantation is not indicated when ventricular arrhythmias arise from reversible metabolic or drug-induced causes.[1,2] In all cases presented, arrhythmias resolved completely following correction of electrolyte abnormalities and elimination of precipitating factors, confirming the transient nature of the arrhythmogenic substrate.

CONCLUSION

Torsades de pointes in patients with ACS and HFrEF should immediately raise suspicion for diuretic-induced hypokalemia and hypomagnesemia, representing a reversible acquired pseudo–Bartter/Gitelman syndrome. The convergence of electrolyte depletion, ischemia, bradycardia, and reduced repolarization reserve creates a highly arrhythmogenic substrate. Early recognition, aggressive electrolyte correction, and appropriate heart-rate management are lifesaving and prevent unnecessary long-term device therapy.[1–3]

Disclaimer (Artificial Intelligence)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, Copilot, etc.) or text-to-image generators were used during the writing or editing of this manuscript.

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