Section: General Medicine

Case Series

ELECTROCARDIOGRAPHIC SPECTRUM OF CARDIAC ARRHYTHMIAS COMPLICATING ACUTE MYOCARDIAL INFARCTION: A CASE SERIES OF 8 PATIENTS

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ABSTRACT

Background: Arrhythmias are a common and potentially fatal complication of acute myocardial infarction (AMI), occurring in up to 90% of affected patients. The 12-lead electrocardiogram (ECG) is the primary tool for their identification and characterization in the clinical setting. Aims: This case series documents the electrocardiographic spectrum of arrhythmias encountered in 8 patients with AMI, illustrating the diagnostic value of the 12-lead ECG and its role in guiding management decisions

Materials and Methods: Eight consecutive patients with AMI who developed clinically significant arrhythmias during hospitalization were included. Patient histories, clinical findings, relevant investigations, and 12-lead ECG tracings were reviewed and reported in a de-identified format.

Results: The series comprised four male and four female patients with a mean age of 59 years (range 47–72). Arrhythmias documented included second-degree AV block Mobitz Type I (Wenckebach phenomenon) in two cases (Cases 1 and 7); complete (third-degree) AV block in two cases (Cases 2 and 3), one complicating inferior MI and one anterior MI, the latter with a ventricular escape rate of 25 beats per minute requiring emergency temporary pacing; atrioventricular nodal re-entrant tachycardia (AVNRT) at 236 bpm in one case (Case 4); atrial fibrillation with fast ventricular response (Case 5); and supraventricular tachycardia (SVT) in the context of NSTEMI with diffuse subendocardial ischemia in two cases (Cases 6 and 8). ECG features of the underlying infarction were present concurrently in most tracings.

Conclusion: The series illustrates the breadth of arrhythmias that may complicate AMI. Systematic 12-lead ECG analysis, interpreted alongside the clinical picture, is indispensable for diagnosis, localization of myocardial injury, and directing timely intervention.

Keywords: Acute Myocardial Infarction, Arrhythmia, Electrocardiography, Atrioventricular Block, Supraventricular Tachycardia, Atrial Fibrillation.

INTRODUCTION

Acute myocardial infarction (AMI) is a leading cause of cardiovascular morbidity and mortality worldwide. Alongside its direct hemodynamic consequences, AMI is complicated by a broad spectrum of cardiac rhythm disturbances. Nearly 90% of patients with AMI develop some form of arrhythmia, and approximately 25% manifest a conduction disturbance within 24 hours of infarct onset.[1] These arrhythmias range from self-limiting bradycardias to malignant ventricular tachyarrhythmias, and their timely recognition directly influences treatment decisions and patient outcomes.

The 12-lead electrocardiogram (ECG) is the cornerstone of arrhythmia diagnosis in the acute cardiac setting. Beyond identifying ST-segment changes that localize the site of coronary occlusion, it provides real-time data on cardiac rhythm, AV conduction, and QRS morphology.[2] These data collectively enable the clinician to assess the severity

of ischemia, anticipate likely complications, and direct urgent management. Standard criteria for ECG interpretation in acute ischemia, including criteria for AV block, bundle branch block, and ST changes, have been formalized in joint guidelines from major cardiology societies.[3]

The type of arrhythmia encountered in AMI is closely linked to the territory of ischemia. Inferior STEMI, most commonly caused by right coronary artery (RCA) occlusion, frequently compromises the AV nodal artery — an RCA branch in approximately 90% of individuals. This produces AV nodal arrhythmias such as sinus bradycardia, first-degree AV block, and second-degree AV block Mobitz Type I (Wenckebach phenomenon). Although Mobitz Type I block most commonly reflects AV-nodal ischemia in inferior MI, it may occasionally complicate anterior MI through transient ischemia of the proximal conduction system, as illustrated later in this series. Complete (third-degree) AV block complicating inferior MI is usually transient, AV-nodal in origin, and responds to atropine or temporary pacing. By contrast, complete AV block in anterior MI reflects ischemia of both bundle branches simultaneously, carries a higher mortality, and typically requires emergency temporary transvenous pacing.

Supraventricular tachyarrhythmias complicate AMI in 6–21% of patients and independently worsen outcomes.[4] Atrial fibrillation (AF) is the most common sustained supraventricular arrhythmia in this context, arising through sympathetic activation, atrial ischemia, and hemodynamic stress. It impairs cardiac output by abolishing the atrial contribution to ventricular filling and can precipitate hemodynamic deterioration, particularly when left ventricular function is already compromised. AVNRT, while less commonly reported in the acute MI setting, can occur as a consequence of altered autonomic tone and local ischemia involving the perinodal tissues, producing a narrow-complex tachycardia at high rates that may further compromise myocardial perfusion.

SVT in the context of NSTEMI with diffuse subendocardial ischemia presents a particular diagnostic challenge. The pattern of widespread ST-segment depression across multiple leads with reciprocal ST elevation in leads aVR and V1 is the ECG signature of left main coronary artery (LMCA) or proximal LAD occlusion. When this ischemic pattern coexists with a concurrent tachyarrhythmia, the diagnosis may be delayed if attention is focused solely on the rhythm. Ventricular tachyarrhythmias, bradyarrhythmia, and atrial tachyarrhythmias are among the most frequently encountered rhythm disturbances during the acute phase of myocardial infarction. Although advances in reperfusion therapy, including primary percutaneous coronary intervention and thrombolytic treatment, have substantially reduced the incidence of these arrhythmias by facilitating early restoration of coronary blood flow, prompt electrocardiographic recognition remains crucial for timely diagnosis, risk stratification, and appropriate therapeutic management.[5]

The clinical outcome of arrhythmias in AMI is influenced by the arrhythmia type, its hemodynamic impact, the territory of infarction, and the speed of recognition and treatment. A systematic approach to 12-lead ECG interpretation is therefore essential in the coronary care unit (CCU). This case series presents 8 patients with AMI in whom distinct arrhythmias were documented electrocardiographically.

CASE SERIES

Case 1

A 55-year-old male presented to the emergency department with a 2-hour history of central chest pain radiating to the left arm, associated with profuse diaphoresis and nausea. He had a known history of hypertension managed with amlodipine. He had no prior cardiac history and was a non-smoker. On examination, his blood pressure was 100/68 mmHg and pulse was 44 beats per minute (bpm), irregular. He was afebrile. The jugular venous pressure was not elevated. Heart sounds were normal with no added sounds. Chest auscultation revealed no crepitations. Serum troponin I was markedly elevated at 12.4 ng/mL (reference < 0.04 ng/mL). Chest radiograph showed a mildly enlarged cardiac silhouette with no pulmonary oedema. Bedside echocardiography demonstrated inferoposterior wall hypokinesia with an estimated ejection fraction of 45%. The 12-lead ECG showed ST-segment elevation in leads II, III, and aVF with reciprocal ST depression in leads I and aVL, consistent with acute inferior STEMI. The rhythm demonstrated prolongation of the PR interval with alternating conducted and non-conducted P wave, with an underlying sinus bradycardia, in keeping with a Mobitz type II [Figure 1].

The ECG demonstrating second-degree AV block Mobitz Type II with sinus bradycardia is shown below

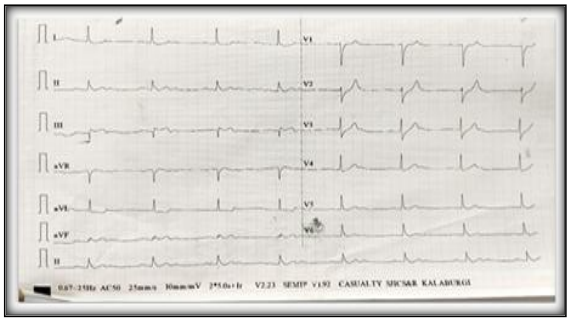

Figure 1: Twelve-lead ECG demonstrating second-degree atrioventricular (AV) block Mobitz Type II in a patient with acute inferior STEMI. Prolongation of the PR interval with alternating conducted and non-conducted P wave.

The patient was treated with aspirin 300 mg, ticagrelor 180 mg, and unfractionated heparin.

Coronary angiography demonstrated total occlusion of the RCA at its mid-segment. Primary PCI was performed with successful stent deployment. The AV conduction abnormalities resolved within 36 hours of revascularization.

Case 2

A 63-year-old male presented with a 3-hour history of severe retrosternal chest pain and two episodes of presyncope. He had a background of hypertension and type 2 diabetes mellitus on oral medication. He was an ex-smoker with a 20-pack-year history.

On examination, blood pressure was 85/55 mmHg and pulse was 36 bpm, regular. He appeared pale and diaphoretic, with signs of reduced peripheral perfusion. The jugular venous pressure was elevated at 4 cm above the sternal angle. Heart sounds were present with a soft first heart sound. No murmurs were audible.

Troponin I was elevated at 18.7 ng/mL. Bedside echocardiography was done which showed inferior and right ventricular wall motion abnormality with an ejection fraction of 30%. The 12-lead ECG showed ST-segment elevation in leads I, aVL, V3 - V6. These findings, together with the angiographic occlusion described below, were consistent with inferior STEMI. ECG showed complete AV dissociation, with P waves and QRS complexes occurring independently at their respective rates. The ventricular escape complexes exhibited a broad left bundle branch block (LBBB) morphology, with QS complexes in leads V1–V2, poor R-wave progression, and biphasic T waves in leads V3–V6. This was indicative of an escape focus below the bundle of His. The QT interval was prolonged (QTc 451 ms) (ECG 2).

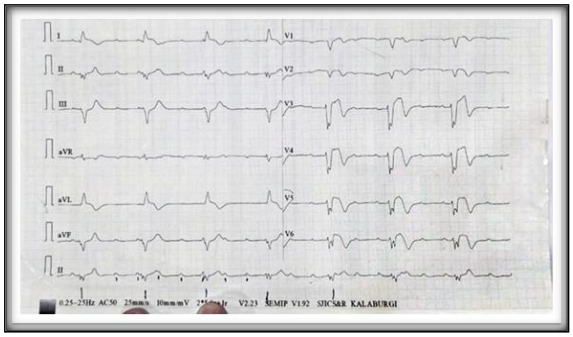

Figure 2: 12-lead ECG demonstrating complete (third-degree) atrioventricular block with a broad ventricular escape rhythm of left bundle branch block (LBBB) morphology. QS Complexes are present in leads V1–V2 with poor R-wave progression, and biphasic T waves are seen in leads V3–V6, ST-segment elevation in leads I, aVL, V3 - V6.

Emergency temporary transvenous pacing was inserted, restoring hemodynamic stability. Coronary angiography revealed proximal LCA occlusion. Primary PCI was performed successfully and permanent pacemaker was placed. The complete AV block resolved within 48 hours of revascularization.

Case 3

A 70-year-old male was brought to the emergency department by ambulance with a 4-hour history of severe chest pain, profound breathlessness, and near-syncope. He had a background of dyslipidemia but no prior known coronary artery disease.

The patient presented in cardiogenic shock: blood pressure was 75/50 mmHg, pulse was 25 bpm, and respiratory rate was 30 per minute. He was cold and clammy with markedly reduced peripheral perfusion. Crackles were audible at both lung bases. An emergency resuscitation team was activated immediately on arrival.

Troponin I was critically elevated at 45.2 ng/mL. Chest radiograph showed pulmonary oedema with cardiomegaly. The 12-lead ECG showed ST-segment elevation in leads V2–V4 consistent with acute anterior STEMI. The rhythm showed complete AV dissociation with a ventricular escape rate of approximately 25 bpm and a QRS duration of 173 ms, indicating a ventricular origin of the escape focus with severe intraventricular conduction delay. ST changes in leads V2–V4 confirmed anterior myocardial injury. Left atrial overload was an additional finding. [Figure 3]

The ECG demonstrating complete AV block complicating acute anterior STEMI with a critically slow ventricular escape rhythm is shown below.

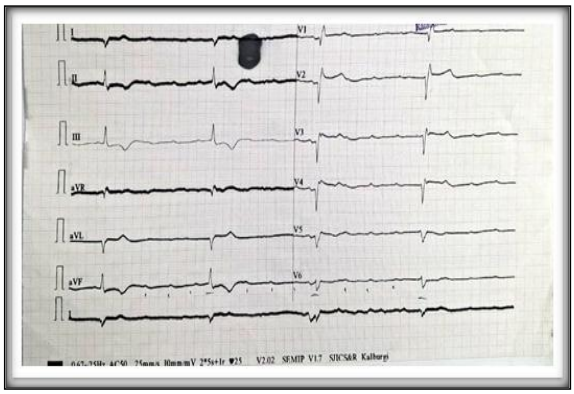

Figure 3: Twelve-lead ECG showing complete (third-degree) atrioventricular block complicating acute anterior myocardial infarction. The ventricular escape rate is approximately 25 beats per minute with a QRS duration of 173 ms, indicating a ventricular origin of the escape rhythm and severe hemodynamic compromise. ST-segment changes in leads V2–V4 are consistent with anterior myocardial injury.

Emergency temporary transvenous pacing was initiated, improving hemodynamics transiently. Coronary angiography was done which showed proximal LAD occlusion with significant thrombus burden. Primary PCI was performed, but the patient subsequently developed refractory cardiogenic shock and required inotropic support.

Case 4

A 51-year-old female was admitted following a 3-hour history of ischemic chest pain that had partially settled with rest. During initial assessment in the

emergency department, she developed sudden-onset palpitations and light-headedness.

At the time of arrhythmia onset, blood pressure was 88/58 mmHg and pulse was 236 bpm, regular. The patient was anxious but remained conscious. Heart sounds were rapid but no murmurs were audible. Lung fields were clear.

Troponin I was elevated at 3.2 ng/mL. The pre-arrhythmia 12-lead ECG had shown ST-segment depression in leads V3–V5. At the time of the palpitations, the 12-lead ECG demonstrated a regular narrow-complex tachycardia at a ventricular rate of approximately 236 bpm. No distinct P waves were visible preceding the QRS complexes; retrograde P waves were buried within or immediately followed the QRS, consistent with typical (slow–fast) AVNRT. [Figure 4]

The ECG demonstrating AVNRT at approximately 236 bpm in a patient with myocardial infarction is shown below.

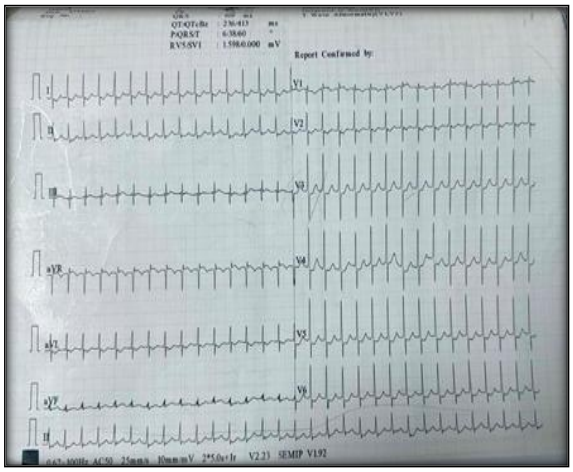

Figure 4: Twelve-lead ECG demonstrating atrioventricular nodal re-entrant tachycardia (AVNRT) at a ventricular rate of approximately 236 beats per minute in a patient with myocardial infarction. The rhythm is a regular narrow-complex tachycardia with no discernible P waves preceding the QRS complexes, consistent with typical (slow–fast) AVNRT in which retrograde P waves are buried within or immediately follow the QRS.

Vagal maneuvers were unsuccessful. Intravenous adenosine 6 mg successfully terminated the tachycardia, restoring sinus rhythm. The underlying MI was managed with dual antiplatelet therapy and urgent coronary angiography, which revealed a critical mid-LAD stenosis treated with PCI.

Case 5

A 62-year-old male presented with a 2-hour history of central chest pain and palpitations. He had a background of essential hypertension and was a current smoker. He had no prior diagnosis of atrial fibrillation.

On examination, blood pressure was 112/72 mmHg and pulse was 140 bpm, irregularly irregular. Heart sounds were variable in intensity. No signs of acute pulmonary congestion were present. Peripheral pulses were palpable but rapid.

Troponin I was elevated at 8.9 ng/mL. The 12-lead ECG revealed an irregularly irregular rhythm with an absence of distinct P waves, replaced by fibrillatory baseline undulations. The ventricular rate exceeded 120 bpm. ST-segment depression was noted in leads V3–V6, consistent with concurrent subendocardial ischemia. An abnormal Q wave was present in lead V1, suggesting anterior infarct involvement. [Figure 5]

The ECG demonstrating atrial fibrillation with fast ventricular response in a patient with myocardial infarction is shown below.

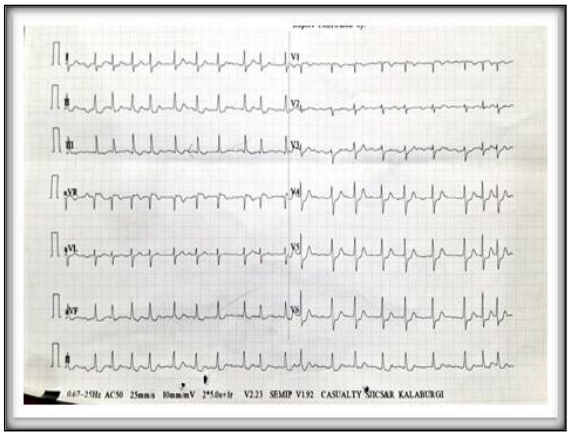

Figure 5: Twelve-lead ECG (paper speed 25 mm/s, gain 10 mm/mV) showing atrial fibrillation (AF) with fast ventricular response (FVR) in a patient with myocardial infarction. The rhythm is irregularly irregular with an absence of distinct P waves, replaced by fibrillatory baseline undulations, at a ventricular rate exceeding 120 beats per minute. ST-segment depression in leads V3–V6 is consistent with concurrent subendocardial ischemia. An abnormal Q wave in lead V1 suggested evolving or prior anterior infarction.

Rate control was achieved with intravenous metoprolol 5 mg administered in incremental doses. Anticoagulation with low-molecular-weight heparin was commenced. Coronary angiography the following day revealed significant LAD disease, and PCI was performed to the culprit lesion. The AF converted spontaneously to sinus rhythm within 24 hours following revascularization.

Case 6

A 58-year-old female was referred from a peripheral facility with a 6-hour history of worsening exertional chest pain and dyspnea. She had a background of type 2 diabetes mellitus and a family history of premature coronary artery disease. She was on metformin and a statin.

On examination, blood pressure was 120/78 mmHg and pulse was 158 bpm, regular. Heart sounds were rapid. Mild bi-basal crepitations were audible. There was no peripheral oedema.

Troponin I was elevated at 6.1 ng/mL. The 12-lead ECG showed a regular narrow-complex tachycardia, which was suggestive of SVT, with widespread ST-

segment depression in, leads I, II, aVF, and V4–V6, and reciprocal ST elevation in leads aVR and V1. All these changes suggested pattern of diffuse subendocardial ischemia. An abnormal Q wave was also noted in lead V1, and T-wave inversions were present in the inferolateral leads. These additional ECG findings further substantiated the diagnosis of NSTEMI. [Figure 6]

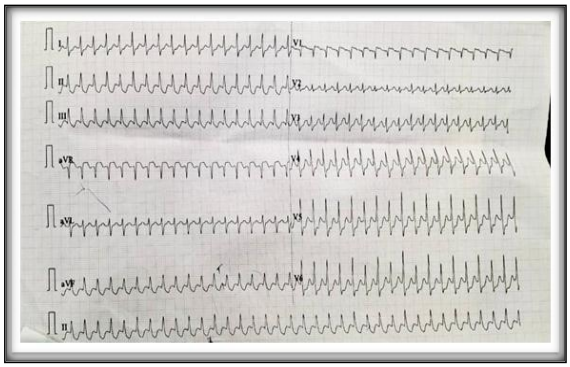

Figure 6: 12-lead ECG showing supraventricular tachycardia (SVT) in the context of NSTEMI. Widespread ST-segment depression is also evident in leads II, aVF, V4–V6, and I. reciprocal ST elevation in leads aVR and V1, forming the electrocardiographic pattern suggestive of diffuse subendocardial ischemia. An abnormal Q wave in lead V1 and T-wave inversions across the inferolateral leads further confirmed the diagnosis of NSTEMI.

The SVT was terminated by intravenous adenosine 6 mg and sinus rhythm was restored. The ST elevation in aVR with widespread depression raised strong clinical suspicion for left main stem (LMS) disease. A coronary angiography was immediately performed which confirmed severe LMS stenosis. The patient was eventually referred for emergency coronary artery bypass grafting (CABG) and had an uneventful post-operative course.

Case 7

A 53-year-old male presented to the emergency department with a 90-minute history of crushing anterior chest pain and sweating. He was a current smoker with no prior cardiac diagnosis. His only regular medication was aspirin taken intermittently.

Blood pressure was 116/74 mmHg and pulse was 52 bpm, irregular. The patient was diaphoretic. Heart sounds were normal. No crepitations or murmurs were identified.

Troponin I was elevated at 10.2 ng/mL, consistent with acute myocardial infarction. The 12-lead ECG demonstrated second-degree atrioventricular block, Mobitz type I (Wenckebach), with progressive PR-interval prolongation terminating in a non-conducted P wave. A QS complex was present in lead V1 with poor R-wave progression across the precordial leads. [Figure 7]

In view of the clinical presentation and biomarker elevation, the patient underwent urgent coronary angiography, which demonstrated a significant stenosis of the mid–left anterior descending (LAD) artery. Percutaneous coronary intervention with stent placement was performed successfully, with restoration of flow. The Mobitz type I block was monitored throughout and resolved following reperfusion without requiring temporary pacing.

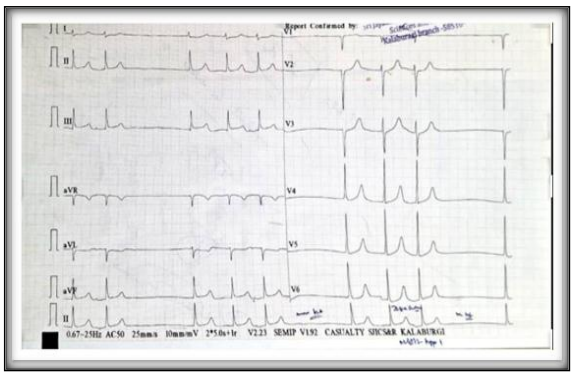

Figure 7: Twelve-lead ECG demonstrating second-degree atrioventricular block, Mobitz type I (Wenckebach phenomenon). The lead II rhythm strip shows progressive beat-to-beat prolongation of the PR interval culminating in a non-conducted P wave (dropped QRS)

Case 8

A 47-year-old male presented with a 3-hour history of chest pain followed by acute-onset palpitations and hemodynamic deterioration. He had a background of hypertension with no prior cardiac events. He was on no regular medication at the time of presentation.

On examination, blood pressure was 94/62 mmHg and pulse was 162 bpm, regular. The patient was pale with signs of reduced peripheral perfusion. Lung fields were clear. No murmurs were audible.

Troponin I was elevated at 5.7 ng/mL. The 12-lead ECG demonstrated a regular narrow-complex tachycardia at approximately 162 bpm consistent with SVT. The tracing shown captures the tachyarrhythmia; the ischemic changes were documented on the presenting and post-adenosine 12-lead ECGs. [Figure 8]

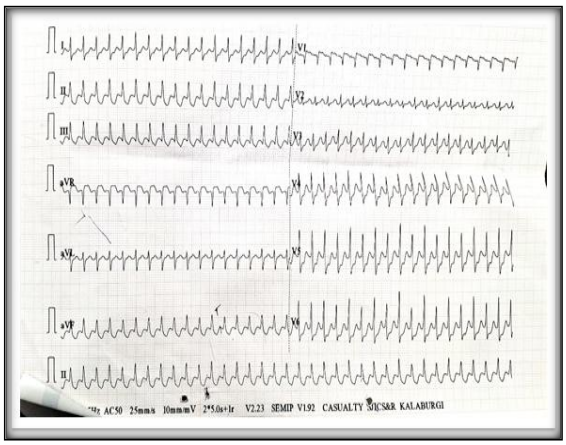

Figure 8: Twelve-lead ECG demonstrating supraventricular tachycardia (SVT) in a patient with acute myocardial infarction. The tracing shows a regular narrow-complex tachycardia at approximately 162 beats per minute.

Intravenous adenosine 6 mg was administered; the tachycardia terminated, revealing sinus rhythm with anterior ST-elevation changes confirming STEMI. The patient was taken for emergency primary PCI to the LAD. Flow was restored and the patient's hemodynamics improved. He was discharged in stable condition after 5 days.

DISCUSSION

Cardiac arrhythmias are well-recognised complications of AMI that significantly affect both short- and long-term patient outcomes. This case series documents the electrocardiographic spectrum of these arrhythmias across 8 patients, illustrating the diversity of rhythm disturbances that can arise in the acute ischaemic setting and the diagnostic value of the 12-lead ECG in characterising each arrhythmia type.

The two cases of second-degree AV block Mobitz Type I (Cases 1 and 7) both demonstrated classic Wenckebach conduction, with progressive PR prolongation culminating in a dropped beat. This pattern reflects AV nodal ischaemia, arising most commonly in inferior MI through reduced flow in the AV nodal artery. Behar et al,[6] reported complete AV block in 11% of 2,273 patients with inferior MI, with in-hospital mortality of 37% in those with block versus 11% in those without, underscoring the prognostic weight of conduction disturbances even in AV-nodal disease. The Wenckebach block in Case 7, which complicated an anterior STEMI rather than an inferior one, is less typical and may reflect transient ischaemia of the proximal conduction system; it was nonetheless transient and self-resolving post-reperfusion.

The two cases of complete third-degree AV block (Cases 2 and 3) illustrate contrasting degrees of severity and haemodynamic impact. In Case 2, complicating inferior STEMI, the ventricular escape rhythm had a broad left bundle branch block (LBBB) morphology, and its slow rate necessitated emergency temporary pacing to maintain hemodynamic stability while revascularization was arranged. A similar case has also been reported by Liang et al., in which complete heart block complicating an inferior STEMI remained hemodynamically stable and reverted to sinus rhythm following recanalization of an occluded right coronary artery, obviating the need for permanent pacing.[7] In Case 3, complicating anterior STEMI, the escape rate was critically slow at 25 bpm and reflected infranodal block from bilateral bundle branch ischaemia. Alnsasra et al.[8], in a contemporary multicentre survey of 11,487 ACS patients, reported high-grade AV block in 2.7% overall, with the incidence declining from 4.2% in 2000 to 2.1% in 2010, attributable to more widespread use of primary PCI. Co-existing right ventricular involvement is a key determinant of mortality in inferior MI with CHB: Mavrić et al,[9] demonstrated a mortality of 41% when CHB was accompanied by right ventricular infarction, versus 14% without it, confirming that the combination of factors defines the highest risk subgroup.

Atrial fibrillation with fast ventricular response (Case 5) is the most common sustained supraventricular arrhythmia in AMI.[10] Angeli et al.[11], in a systematic meta-analysis, found that AF in AMI patients was associated with a twofold increased risk of in-hospital mortality (pooled OR 2.00, 95% CI 1.93–2.08). The GISSI-3 dataset of 17,944 AMI patients confirmed that new-onset AF during hospitalisation carried significantly worse short- and long-term survival, particularly when it developed after the initial 24 hours.[12] In our case, prompt rate control with beta-blockade, anticoagulation, and early coronary revascularisation were the cornerstones of management, in line with current consensus guidance.[13]

AVNRT at 236 bpm (Case 4) is an unusual but recognised complication in the AMI context. It arises from a re-entrant circuit within or near the AV node, with retrograde atrial activation occurring simultaneously with or just after ventricular depolarisation, explaining the absence of antecedent P waves on the 12-lead ECG. Zoni Berisso et al,[14] studying 209 AMI patients using 24-hour Holter monitoring in the late hospital phase, identified supraventricular tachyarrhythmias in 54% of patients, with complex forms (including paroxysmal tachycardia and AF/flutter) in 13%, and found a significant association between complex SVT and subsequent cardiac events. AVNRT at very high ventricular rates, as illustrated here, causes haemodynamic compromise directly through impaired diastolic filling and indirectly through worsening myocardial oxygen demand in an already ischaemic myocardium. Adenosine remains the first-line treatment and was effective in this case.

SVT in the context of NSTEMI with diffuse subendocardial ischaemia (Cases 6 and 8) presents a diagnostic challenge. The aVR-predominant ST-elevation pattern in Case 6 pointed strongly to LMS or severe proximal LAD disease — findings that require emergency escalation beyond arrhythmia management alone. This combination of concurrent tachyarrhythmia and critical coronary anatomy requires a systematic approach to the 12-lead ECG so that the ischaemic pattern is not overshadowed by the arrhythmia. In both cases, termination of the SVT with adenosine unmasked the underlying ischaemic changes and guided the subsequent management trajectory.[10]

The EHRA/ACCA/EAPCI joint position paper on cardiac arrhythmias in ACS recommends continuous ECG monitoring and 12-lead ECG-guided management for all arrhythmias complicating ACS, with early reperfusion as the most effective arrhythmia treatment.[13] The findings in this series support that recommendation. In all cases where revascularisation was achieved, conduction disturbances and tachyarrhythmias improved or

resolved following restoration of coronary flow, highlighting ischaemia as the common upstream driver.[15]

Limitations of this series include the small number of cases and its descriptive nature, which precludes statistical comparison. Cases were collected from a single tertiary centre and may not represent the full spectrum encountered in primary or community settings. Longer-term follow-up data, including arrhythmia recurrence and mortality, are not available within the scope of this report. Nonetheless the primary value of this case series lies in illustrating the breadth of ECG findings and their clinical context in cases of MI.

CONCLUSION

This case series highlights how varied the ECG findings can be when arrhythmias complicate AMI. Each rhythm disturbance in the series carried its own distinct electrocardiographic features. AV nodal Wenckebach block appeared in inferior wall myocardial infarction. Complete heart block with a critically slow ventricular escape appeared in anterior wall MI. Rapid AVNRT and AF with a fast ventricular response were also seen. In addition, SVT was found superimposed on a pattern of diffuse subendocardial ischemia. These distinct ECG features are what guide both diagnosis and management. Systematic 12-lead ECG analysis therefore remains essential. When combined with rapid coronary reperfusion, it is central to reducing arrhythmia-related morbidity and mortality in AMI.

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