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CASE REPORT
Coronary heart disease

Inferior ST-elevation myocardial infarction managed with a pharmacoinvasive strategy and conservative management of delayed atrioventricular block: classical case report

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Background The 2017 ESC guideline on patients with ST-segment elevation myocardial infarction (STEMI) provides guidance regarding the optimal management of these patients. Transient atrioventricular (AV) block is a relatively common complication of inferior STEMI and its management is also addressed in the guidelines.

Case summary A 64-year-old gentleman with multiple cardiovascular risk factors presented to the emergency department with a history of ischaemic type chest pain and evidence of inferior ST-segment elevation on his electrocardiogram (ECG). First-degree AV block was noted on his initial ECG. He was given thrombolytic therapy as part of a pharmacoinvasive strategy of reperfusion. He, however, failed fibrinolytic therapy, and emergency angiography revealed critical disease of the right coronary artery which was successfully stented. Subsequent to reperfusion, he developed complete AV block without evidence of re-infarction, which was managed conservatively with successful resolution of the block after 7 days of expectant management with temporary transvenous pacing.

Discussion We highlight some of the important management principles from the ESC guideline of STEMI including timing and the management of AV block in these patients. In addition, we highlight the role of a pharmacoinvasive strategy for reperfusion where timeous primary percutaneous coronary intervention cannot be performed. The usefulness of such a strategy within the COVID-19 era is also emphasized.

Keywords ST-elevation myocardial infarction • STEMI • Pharmacoinvasive strategy • AV block • Inferior STEMI • Temporary pacing • ESC guidelines • Classical case report

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Learning points

- The mechanism of atrioventricular (AV) block in ST-segment elevation myocardial infarction (STEMI) is on the basis of AV nodal ischaemia, heightened parasympathetic tone or myocardial necrosis with ischaemia of the bundle branches (usually in the setting of anterior STEMI)
- Earlier reperfusion is associated with earlier recovery of AV block.
- AV block in the setting of STEMI usually recovers with reperfusion and an expectant approach is preferred.
- AV block associated with inferior myocardial infarction (MI) is usually supra-Hisian in origin and more likely to recover than AV block associated with anterior MI which is usually infra-Hisian in origin.

Introduction

The 2017 ESC guidelines for the management of patients with ST-segment elevation myocardial infarction (STEMI) provides guidance regarding the optimal management of these patients.[1] Early reperfusion using percutaneous coronary intervention (PCI) or fibrinolysis as part of a pharmacoinvasive strategy to reperfusion is paramount in improving outcomes in these patients. Primary PCI is the preferred reperfusion method if it can be performed in a timely manner. Atrioventricular (AV) block in the setting of STEMI is not uncommon although its development in STEMI has decreased significantly in the era of PCI with early reperfusion to <5%.[2] We use a case of inferior STEMI complicated by AV block post-reperfusion to highlight some of the management principles highlighted in the ESC guidelines. The role of adopting a pharmacoinvasive strategy to reperfusion is also highlighted. The management of AV block in the setting of STEMI is also discussed.

Timeline

8 h prior	The patient developed stuttering angina
2 h prior	The patient developed maximal, ischaemic type chest pain
First medical contact (FMC)	The patient was diagnosed with an inferior ST-segment elevation myocardial infarction and fibrinolytic therapy initiated at the point of FMC. First-degree atrioventricular (AV) block was noted on his initial electrocardiogram (ECG)
60 min after fibrinolytic	The patient had ongoing chest pain and persistent ST-elevation on ECG and a decision was made to perform rescue percutaneous coronary intervention (PCI). Angiography revealed a critical lesion in the right coronary artery (Thrombolysis in Myocardial Infarction II flow) which was successfully reperfused
Post-angiography	AV block of varying degrees was noted
Day 7 post-PCI	There was resolution of AV block

Case presentation

A 64-year-old gentleman of mixed ancestry, with poorly controlled type 2 diabetes mellitus (HbA1C—12.7%/115 mmol/mol), hypertension, dyslipidaemia, and 30-pack-year smoking history with no previous history of ischaemic events or angina, presented to a non-PCI centre within our referral network with ischaemic chest pain. He reported intermittent episodes of short-lived (<10 min) pain which initially started 8 h prior to presentation, with subsequent development of non-remitting, central crushing chest pain which reached maximal intensity 2 h before presentation. Initial electrocardiogram (ECG), done within 10 min of presentation, showed evidence of an inferior STEMI (*Figure 1*) with V4R ST-elevation confirming right ventricular (RV) involvement. First-degree AV block was also noted, with no prior history of pre-syncope or syncope. On presentation, he had a blood pressure (BP) of 128/88 mmHg and pulse of 90 b.p.m. with SPO_2 breathing ambient air of 97%. He had grade 1 hypertensive retinopathy and an otherwise normal clinical examination with no clinical evidence of heart failure. Patient was given 1.5 million units streptokinase as part of a pharmacoinvasive strategy (due to unavailability of an after-hours primary PCI service) for reperfusion which was infused over 30 min. In addition, he was given a loading dose of 300 mg aspirin, 300 mg clopidogrel, 30 mg intravenous enoxaparin (further 80 mg subcutaneously after 15 min), 50 mg atenolol, 40 mg simvastatin, and 10 mg enalapril. Cardiac biomarkers were initially not available. Echocardiography was suggestive of right coronary artery (RCA) occlusion with hypokinesia of the infero-postero left ventricular (LV) walls and reduced RV function. LV-systolic function was calculated at 39%. The hypokinetic regions were neither thin nor echobright, suggesting acute ischaemia as the cause for the myocardial dysfunction.

The patient remained stable, but was deemed to have failed fibrinolytic therapy with <50% reduction in ST-elevation and ongoing chest pain at 60 min post-fibrinolysis (*Figure 2*). He was transferred urgently to our PCI centre, where angiography confirmed critical, proximal RCA disease (Thrombolysis in Myocardial Infarction II flow) which was successfully stented with resolution of the patient's chest pain and resolution of ST-elevation (*Figure 3A and B* and *Video 1*). The other vessels showed mild disease without prognostic flow-limiting lesions (*Figure 3C and D and Video 2*). A high-sensitivity Troponin T level, subsequent to the PCI was measured at 29 196 ng/L.

An hour after angiography, the patient developed first-degree AV block progressing to Mobitz I and then to high-degree 2:1 AV block (*Figure 4*). This was in the absence of new chest pain or ECG changes to suggest acute stent thrombosis. He remained haemodynamically stable throughout, albeit symptomatic with episodes of

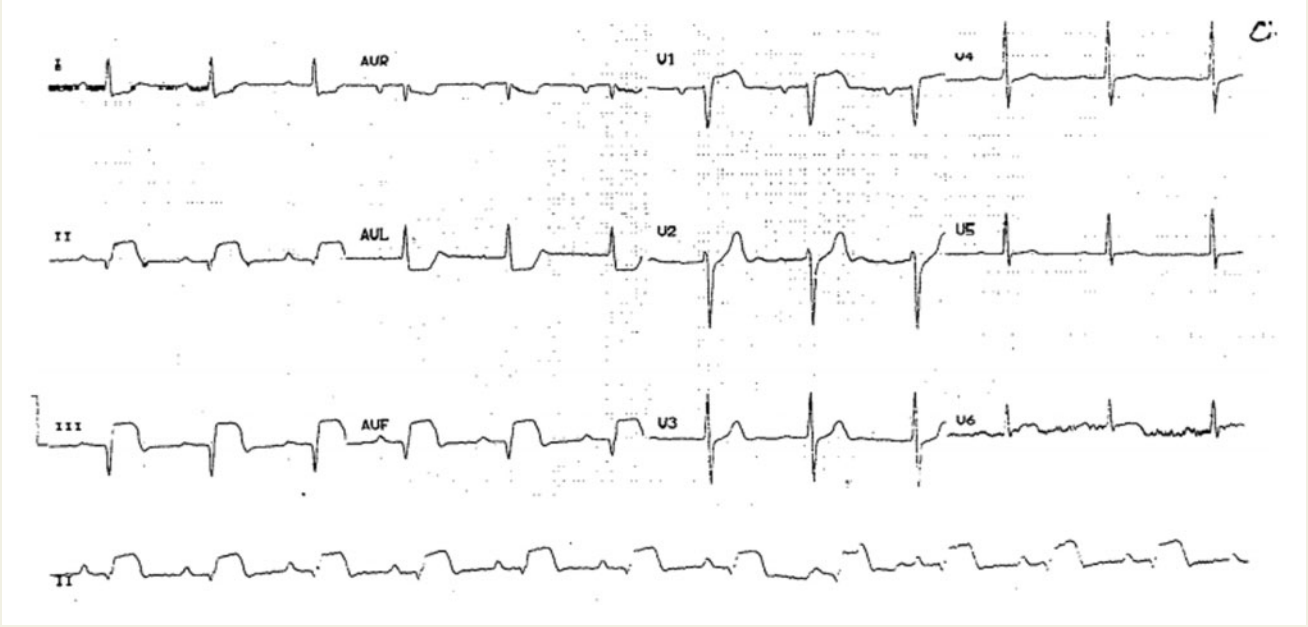


Figure 1 Standard 12-lead electrocardiogram demonstrating inferior ST-elevation.

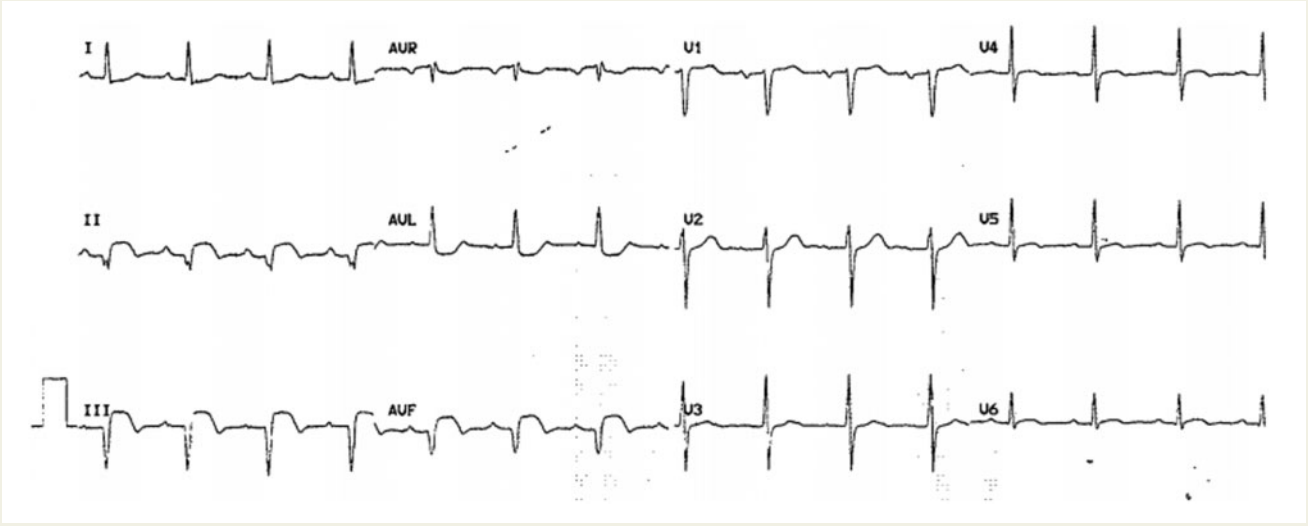


Figure 2 Standard 12-lead electrocardiogram, 60-min post-completion of fibrinolytic therapy with <50% reduction in ST height.

pre-syncope, with a minimum pulse rate of 34 b.p.m. and with no evidence to suggest re-infarction. In light of his symptomatic bradycardia, a temporary, transvenous pacing lead was inserted via the right femoral vein and left at a backup rate of 40 b.p.m. His beta-blockade was stopped and managed conservatively with the temporary pacing wire being replaced on Day 3 and Day 6. He regained sinus rhythm on Day 7 post-infarction with no evidence of AV block with successful reintroduction of beta-blockade prior to discharge.

Discussion

Reduction of total ischaemic time and reperfusion method

Total ischaemic time is a function of patient as well as system delays until successful reperfusion occurs.[1] Early reperfusion is essential in improving outcome in patients with STEMI.[1] Patient delay is reduced by effective health education. Once first medical contact (FMC) occurs, an ECG should be obtained within 10 min to assess for ST-

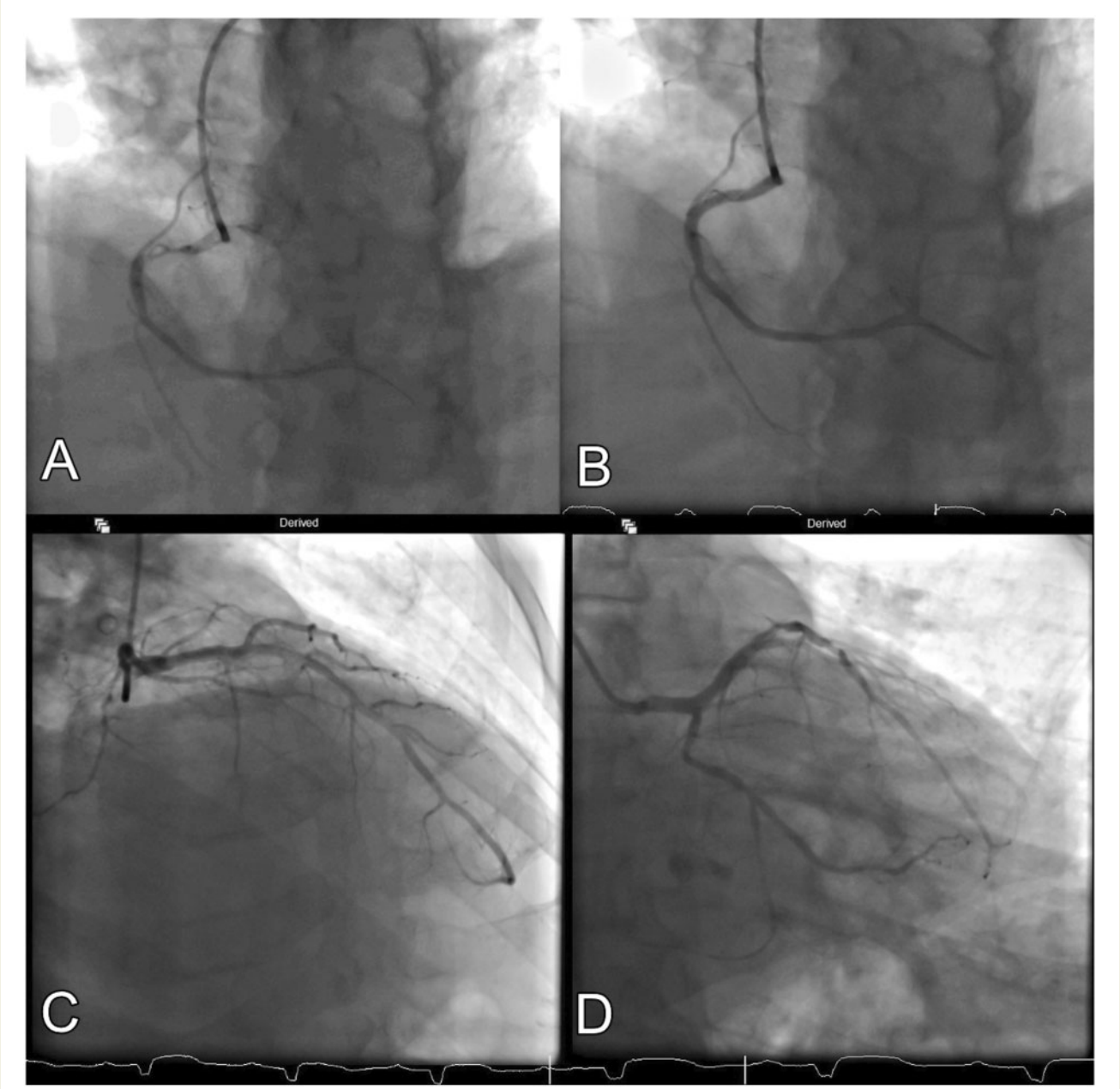
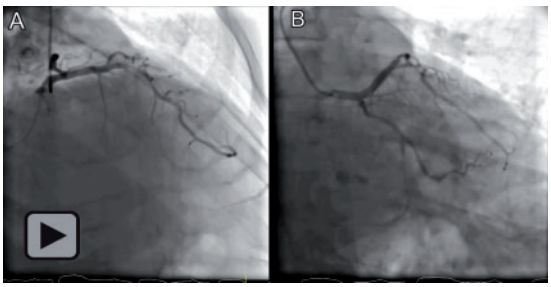


Figure 3 (*A*) Right coronary artery injection confirming critical right coronary artery disease and (*B*) right coronary artery injection after successful stent deployment. (*C*) Right anterior oblique cranial and (*D*) Anterior-posterior caudal injections of the left main coronary artery.

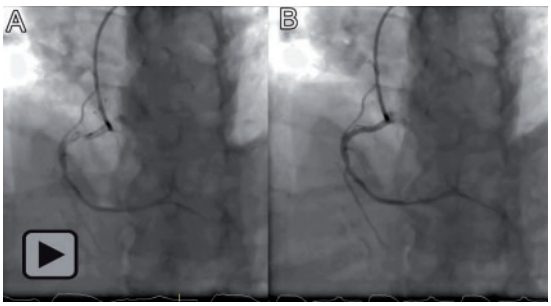
elevation. If ST-elevation is identified, further management is guided by the ability to perform primary PCI in a timeous manner (<120 min). If not achievable, a fibrinolytic strategy for reperfusion is preferred and should be initiated within 10 min of the diagnosis of STEMI being made.[1] Despite initial patient delays, after FMC, our patient was efficiently triaged, ECG performed, and a fibrinolytic strategy initiated.

Primary PCI is the preferred reperfusion strategy for STEMI and has been shown to reduce mortality, re-infarction and stroke if performed expeditiously (<120 min).[1,3,4] Where primary PCI cannot be offered timeously, fibrinolysis remains an alternative option especially when patients are treated early after symptom onset (<3 h).[1,5,6] This should be coupled with early angiography (within 2–24 h) as part of a pharmacoinvasive strategy for reperfusion as this reduces the rates of re-infarction and recurrent ischaemia without an increased risk of adverse events.[1,7,8] A fibrin-specific agent such as tenecteplase is the preferred agent.[1] Due to cost-related issues, streptokinase is the only fibrinolytic agent available in our referral network and was given to our patient.

Due to resource constraints, a primary PCI strategy cannot be offered within our referral network unless the patient presents directly to our PCI centre. We, however, have very good experience



Video 1 (*A*) Right coronary artery injection demonstrating critical proximal right coronary artery stenosis. (*B*) Right coronary artery injection after successful stent deployment.



Video 2 (*A*) Right anterior oblique cranial projection and (*B*) Anterior-posterior caudal projection of the left main coronary artery.

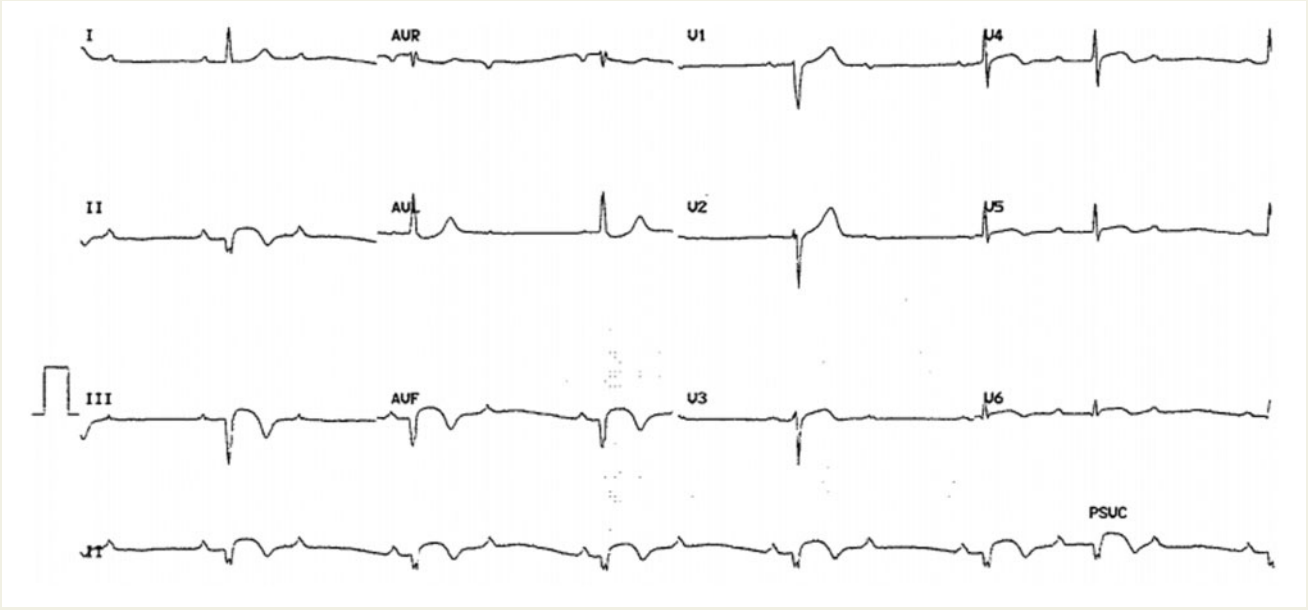


Figure 4 Standard 12-lead electrocardiogram demonstrating high-degree 2:1 atrioventricular block.

with a pharmacoinvasive reperfusion strategy with good rates of initial successful reperfusion (>90%) with fibrinolytic agents. As with our patient, rescue PCI is indicated if fibrinolysis is deemed to have failed (<50% ST-segment resolution at 60–90 min) or have evidence of worsening ischaemia, electrical or haemodynamic instability.[1,9] The role of a pharmacoinvasive approach to reperfusion during the COVID-19 era may be even more relevant where further system delays or concerns regarding infection control may delay time to reperfusion.

Atrioventricular block in ST-segment elevation myocardial infarction

Atrioventricular block in the setting of STEMI is not uncommon although its development in STEMI has decreased significantly in the era of PCI with early reperfusion to <5%. Earlier reperfusion is associated with earlier recovery of AV block.[10] Atrioventricular block in the setting of myocardial infarction (MI) may range from first-degree AV block with inconsequential haemodynamic effects to complete AV block with shock.

Enhanced vagal reflexes (Bezold–Jarisch reflex) due to parasympathetic afferent nerve activation of the infero-posterior LV wall may result in reflex AV block.[11,12] Atrioventricular nodal ischaemia due to compromised blood supply as well as accumulation of local metabolites within the AV node are also thought to be contributory factors. Despite recanalization and patency of epicardial coronary arteries, reperfusion injury in the form of microvascular obstruction may result in ongoing hypoperfusion to myocardial as well as specialized conduction tissue.[13] The blood supply to the AV node typically arises from the RCA, and less commonly the left circumflex artery. In addition, some collateral blood supply to the AV node is provided via septal perforators of the left anterior descending artery. This dual blood supply makes the AV node relatively resistant to transient

ischaemia. However, prolonged ischaemia may result in irreversible AV nodal injury.[11,12] It is most likely a combination of these various mechanisms that results in AV block, with the most dominant mechanism defining the course and clinical consequences thereof. Extensive septal infarction associated with an anterior infarction may also result in extensive bilateral bundle branch infarction and subsequent AV block, and is less likely to recover.[12]

New first-degree or Mobitz I AV block is more commonly associated with inferior STEMI and is seldom of haemodynamic significance. Higher degrees of AV block associated with inferior MI are usually supra-Hisian in origin and may be related to increased vagal tone or transient AV nodal ischaemia.[1] Conversely, AV block associated with anterior wall MI is usually infra-Hisian in origin and as noted previously may signify extensive myocardial necrosis with damage to normal conduction tissue and is associated with poorer patient outcomes.[1] The increased mortality (up to 80% early mortality) in anterior wall infarction with AV block is driven primarily by the extensive necrosis with secondary LV dysfunction and shock as opposed to underlying conduction abnormalities.[14] Patients who develop AV block in the setting of STEMI, irrespective of the site of infarction, have higher mortality rates in comparison to those without AV block.[11]Short-term (30-day) mortality rates in this group of patients who develop AV block is lower in the setting of inferior MI compared to anterior MI and may be an indication of the more benign pathogenic mechanism of AV block in the setting of inferior MI.[11] After 30 days, the mortality rates of patients with and without AV block are equal.[15]

Atrioventricular block associated with STEMI, especially in the setting of inferior MI, usually resolves with reperfusion with recovery of AV nodal function within 2–7 days.[1] During the recovery period, as with our patient, medically refractory, symptomatic, or haemodynamically significant AV block/bradycardia may require temporary pacing.[14] Given the difficulty in assessing reliable myocardial capture, painful nature of stimulation and inability to alter the sensing threshold of the intrinsic ventricular electrical activity with transcutaneous pacing, transvenous pacing is preferred over transcutaneous pacing.[16] Atrioventricular nodal blocking agents including beta-blockers (which are part of the management of STEMI) should be used with caution in this setting. First-degree AV block and Mobitz I AV block are often managed supportively. Atropine can be used in the initial management of AV block originating at the level of the AV node. Given that the natural course of AV block is associated with recovery of conduction, a decision regarding permanent pacemaker implantation should often be deferred for at least 1 week.[1,16]Infra-Hisian AV block (usually extensive anterior MI related) is less likely to recover and is often associated with more severe LV dysfunction and there may be a role for cardiac resynchronization therapy devices with defibrillator capacity in these patients.[1,16]

The development of high-degree AV block after reperfusion, as with our case, is also not uncommon and has in fact been proposed to suggest successful reperfusion.[17,18] Microvascular obstruction, a form of reperfusion injury, has also been suggested as a mechanism for ongoing ischaemia of specialized conduction tissue despite patent epicardial coronary arteries.[19] Additionally, flow restoration may facilitate leucocyte migration to the infarcted area with stimulation of vagal nerve endings in the infero-posterior LV wall with resultant AV block.[18] Due to increased vagal afferents within the posterior ventricular myocardium, this manifestation of AV block is thought to be more common in inferior MI.[17,18] Interestingly, the delayed resolution of AV block in these patients suggests that there may be more complex or multiple mechanisms that contribute to the development of AV block with reperfusion. As the effects of increased vagal tone are expected to be short-lived, transient ischaemia-related AV nodal dysfunction seems to be the more likely aetiology. The complete resolution of AV block, with normalization of the PR interval in our patient, suggests that irreversible damage to the AV node did not occur.

Conclusion

This case highlights the importance and modes of early reperfusion to improve outcomes in STEMI patients. Early reperfusion may also play a role in the early recovery of AV block which may occur as a complication of MI, more commonly inferior or extensive anterior MI. High-degree AV block in this setting usually responds to reperfusion with temporary pacing being reserved in the recovery phase for patients who are haemodynamically unstable or have symptomatic bradycardia. Decisions regarding the implantation of permanent pacemakers should be delayed and should be based on the likelihood of recovery of normal AV conduction.

Lead author biography

Thadathilankal-Jess John completed his medical studies at the University of Cape Town and went on to complete his Physician training at the University of Stellenbosch. He is currently a Cardiology fellow at the University of Stellenbosch with an interest in coronary interventions and pericardial disease.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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