The curious case of missing heartbeats

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SUMMARY

Paroxysmal atrioventricular block (AVB) is a poorly defined and easily missed bradyarrhythmia, which can potentially lead to sudden cardiac death. It is under-recognised due to its abrupt onset and unpredictability. We describe a case that had paroxysmal AVB post-coronary angioplasty and highlight the mechanism as well as the management of this rare condition.

CASE REPORT

An 83-year-old man with hypertension and hyperlipidaemia, who underwent coronary artery bypass surgery 8 years ago, presented with a 1-day history of chest pain. He was treated for non-ST elevation myocardial infarction (NSTEMI) with a peak serum troponin I value of 1437 ng/dL. His 12-lead electrocardiogram (ECG) on admission showed sinus rhythm with evidence of ST-depressions on leads II, III, and aVF (Figure 1a). He was started on continuous cardiac monitoring in the ward. He received aspirin and clopidogrel prior to procedure. He then underwent coronary angiogram, which showed native left main and triple vessel disease with a patent left internal mammary artery graft to the left anterior descending artery and occluded saphenous venous graft (SVG) to the right coronary artery. His SVG to the first obtuse marginal branch (OM1) was patent with evidence of severe stenosis beyond anastomosis. He received balloon angioplasty to the OM1 beyond the SVG anastomosis with reduction in stenosis from 99% to less than 40% and normal coronary flow. There was resolution of ST-depression on his 12-lead ECG post coronary intervention (Figure 1b). He was continued on telemetry post-angioplasty. On day 2 post-angioplasty, his telemetry strips showed an initial sinus rhythm with wide QRS complexes followed by an episode of ventricular standstill lasting 5.6 seconds (Figure 1c). The patient was not on any beta blockers or calcium channel blockers.

The patient was haemodynamically stable and asymptomatic. He was transferred to the high dependency ward for closer monitoring. A temporary transvenous pacing wire was inserted in view of potential recurrent serious bradyarrhythmia, and baseline pacing rate was set at 60 beats per min. There were no further ventricular standstill episodes. His transthoracic echocardiogram showed left ventricular ejection fraction of 35% with regional wall motion abnormalities consistent with multivessel disease and mild-to-moderate mitral regurgitation. A transvenous dual chamber implantable cardioverter-defibrillator (ICD) was implanted for management of bradyarrhythmia in view of the potential need for pacing (bifascicular block and paroxysmal AVB) and for primary prevention against sudden cardiac death on day 5 of admission. He was discharged well 3 weeks later after rehabilitation.

DISCUSSION

Paroxysmal AVB is an uncommon cause of syncope and is often missed to be diagnosed due to its acute onset and unpredictability. It was first described by Sachs and Traynor in 1933,[1] subsequently by Coumel et al.[2] in 1972, demonstrating two cases of paroxysmal AVB precipitated by a premature atrial complex. It is defined by Rosenbaum et al.[3] as an unexpected complete AVB in a patient with delayed ventricular escape but otherwise with normal 1:1 AV-conduction. However, there is no clear consensus on the definition of paroxysmal AVB at present. Patients often present with syncope or presyncope, unlike in our patient, where it was incidentally noticed via continuous cardiac monitoring. It is an ominous cardiac rhythm that may lead to sudden cardiac death as there may be no suggestion of underlying AV conduction disease between culprit episodes.

The prevalence of paroxysmal AVB is not well established in the medical literature and is likely underreported due its unpredictability and abrupt onset. In a prospective study of 52 patients with incomplete or complete RBBB (right bundle branch block) who had syncope and a negative electrophysiology study (EPS), implantable loop recorders showed development of complete heart block in 13 (25%), but only 5 (10%) had complete heart block triggered by a premature beat and were attributable to paroxysmal AVB.[4] Evidence of distal conduction disease is often present and includes underlying RBBB, LBBB (left bundle branch block), and intra-ventricular conduction defect in descending order. However, in up to one-third of the patients, the baseline ECG may be normal.[5]

There are a few mechanisms that can explain paroxysmal AVB, namely vagally mediated, intrinsic, and idiopathic causes. An intrinsic paroxysmal AVB occurs when there is a phase 4 bradycardia-dependent block, which results when a supraventricular or ventricular impulse reaches a diseased His-Purkinje system (HPS) during phase 4 of the action potential when sodium channels are inactive. This leads to ventricular asystole as subsequent impulses are unable to depolarise the diseased tissue. This AVB persists until another premature atrial or ventricular beat captures the HPS prior to phase 4 depolarisation to restore normal conduction. Our patient developed a RBBB and LAFB post-NSTEMI likely due

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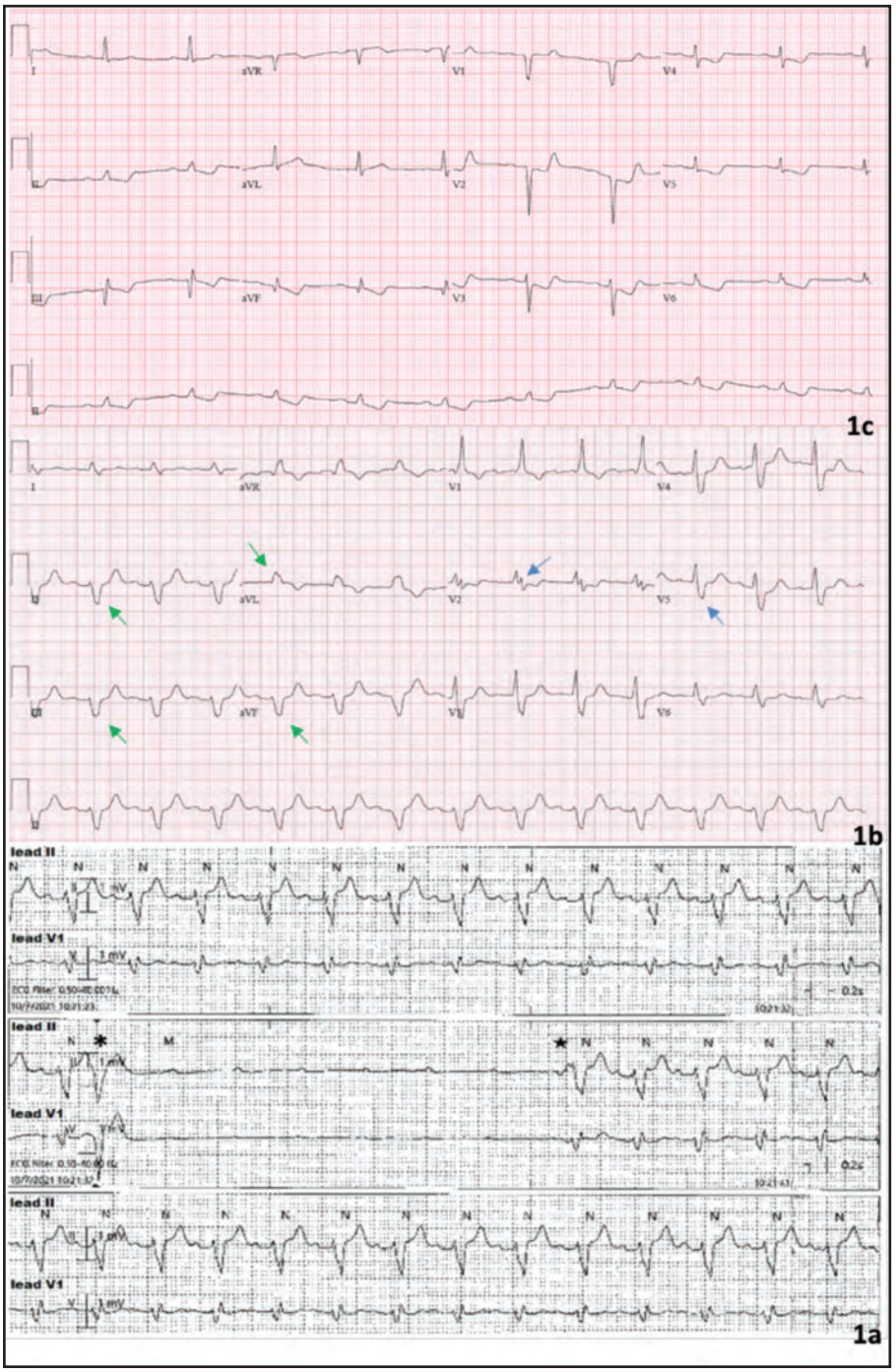

Fig. 1a: A 12-lead ECG on admission, showing sinus rhythm with down-sloping ST-depressions in leads V2–V6 and II, III, and aVF.

Fig. 1b: A 12-lead ECG post-angioplasty, showing baseline sinus rhythm with right bundle branch block (RBBB) (blue arrows) as evidenced by the following features:
1) QRS duration >120 ms
2) RSR' pattern in leads V1 and V2
3) S wave in leads I and V5–V6
The figure also shows left anterior fascicular block (LAFB) (green arrows) as demonstrated by
1) Left axis deviation
2) rS complexes in leads II, III, and aVF
3) Prolonged R wave peak time in aVL > 45 ms
ST-depression resolution was also found in the patient's ECG.

Fig. 1c: Telemetry strip post-angioplasty captured in the ward
The figure shows an initial sinus rhythm with widened QRS complexes. This was followed by a ventricular premature beat (*) with a left bundle branch block (LBBB) morphology that led to an episode of ventricular standstill lasting 5.6 seconds. A non-sinus premature atrial impulse with a negative P wave in lead II (star) restored subsequent AV conduction. This is highly suggestive of paroxysmal atrioventricular block (AVB).

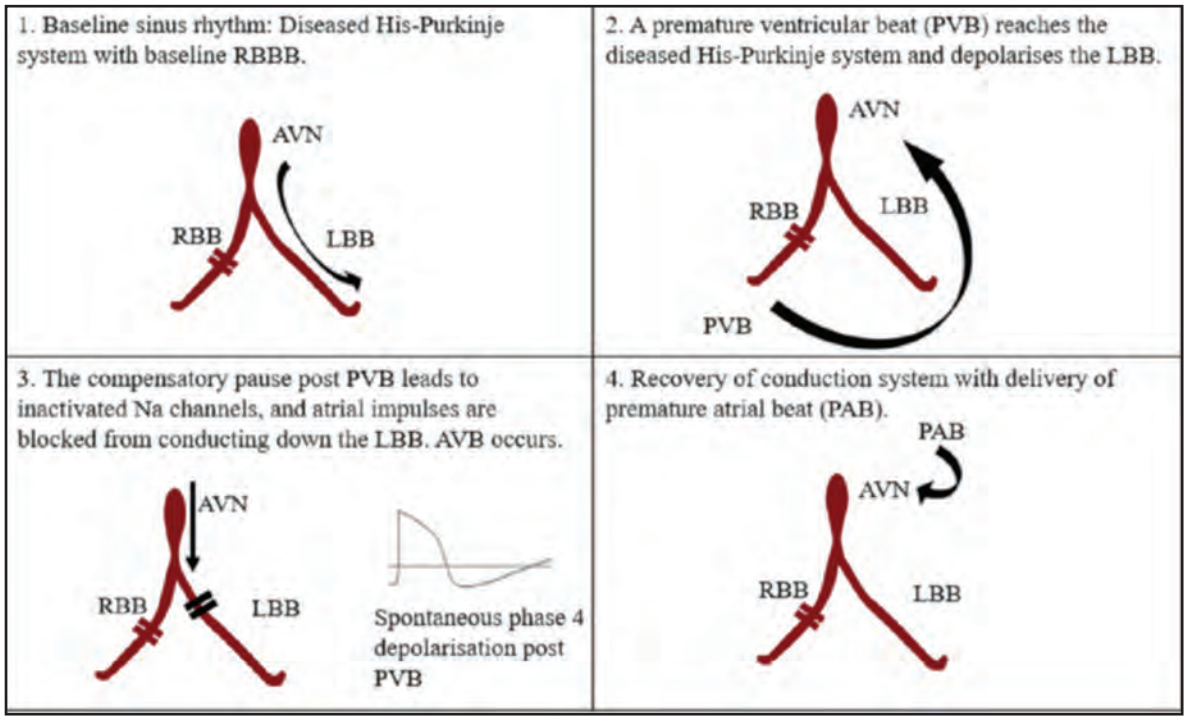

Fig. 2: A schematic diagram showing mechanism of paroxysmal AVB in our patient with baseline RBBB ECG, initiated by a premature ventricular beat leading to spontaneous phase 4 depolarisation of left bundle branch with period of ventricular asystole ensues until restoration of conduction system by a premature atrial beat. RBBB: right bundle branch block; RBB: right bundle branch; LBB: left bundle branch; AVN: atrioventricular node; PVB: premature ventricular beat; PAB: premature atrial beat.

to ischaemia and infarct, with evidence of phase 4 bradycardia-dependent block occurring over the left bundle branch (LBB), leading to a period of ventricular asystole as subsequent atrial impulses are unable to conduct down the LBB (Figure 2). The conduction system was subsequently restored by a premature atrial beat.

It is important to differentiate between paroxysmal AVB and vagally mediated AVB because the latter shows a benign prognosis with no benefit in prophylactic pacemaker implantation.[6] It may not always be possible to differentiate the two entities, but there are some clues on the ECG that allow us to lean towards either diagnosis. Paroxysmal AVB is usually initiated by a premature atrial or ventricular beat, followed by a pause or tachycardia leading to suppression of AV conduction. In vagally mediated AVB, there is gradual slowing with P-P and P-R prolongation prior to AVB or sinus arrest. One can also look at the clinical history to see if there is any suggestion of high vagal tone, which would be more suggestive of vagally mediated AVB. In paroxysmal AVB, there is usually sudden development of AVB and symptoms.

Prompt recognition of paroxysmal AVB is essential because of its potential to lead to sudden cardiac death, which can be prevented by permanent pacemaker implantation. A possible exception to a pacemaker implantation is when paroxysmal AVB occurs in the acute setting with a reversible process such as ischaemia in acute coronary syndrome.[7] However, there are no studies to provide evidence that paroxysmal AVB related to ischaemic events will not recur. In our patient, the episode of paroxysmal AVB occurred 3 days post-myocardial infarction and after revascularisation. Although he was asymptomatic during that episode, decision was made for implantation of dual chamber ICD for prevention of future arrhythmic events after shared decision-making between the patient and medical team. Decision was made for ICD implantation rather than a cardiac resynchronisation therapy device as the patient was unlikely to be pacemaker dependent.

CONCLUSION

Paroxysmal AVB is a rare bradyarrhythmia, whose diagnosis may be easily missed due to its unpredictability and occurrence even in patients with baseline normal 1:1 AV conduction. Prolonged continuous cardiac monitoring may be useful to detect paroxysmal AVB in patients with syncope, especially in those with underlying bundle branch blocks or structural heart disease.

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