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SPOTLIGHT

Unmasking of infra-Hisian conduction abnormality by intravenous isoproterenol during electrophysiology study for syncope

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A 48-year-old gentleman had one episode of dizziness followed by unresponsiveness for 10–20 s. His wife noted an abnormal groaning sound when she rushed to attend him. His recovery was uneventful. There was no post-ictal confusion or bowel/bladder incontinence. The same day ECG revealed a complete left bundle branch block (LBBB) and left axis deviation (LAD) (Figure 1A). Echocardiography revealed a structurally normal heart. A 24-hour Holter showed complete LBBB throughout recording but no high-grade AV block. There was no family history of AV block, early pacemaker implantation, sudden cardiac death, or premature coronary artery disease. Subsequently, two ECGs done in the next 7 days were within normal limits with a narrow QRS complex (Figure 1B). The PR interval was also normal. Another Holter monitoring was done which revealed narrow QRS even during sinus tachycardia up to @130 bpm. At this point, he presented to our outpatient clinic on the 14th day of illness. In view of his initial LBBB, an electrophysiology study (EPS) was planned. A coronary angiogram was performed during the same procedure which was normal. During EPS, his AV Wenckebach during decremental atrial pacing was consistently normal (320–330 ms, Supra- His). During atrial pacing or atrial extrastimuli (AES) he never developed any LBBB. The HV remained normal (42–44 ms) throughout (Figure 2A). At this point, repeat testing after isoproterenol infusion was planned. A slow IV push of 4 mcg isoproterenol was administered. As his sinus rate went >110 bpm decremental atrial pacing was repeated. This time the Wenckebach point was lower and he developed complete LBBB and LAD (similar to his first clinical ECG). Moreover, his HV jumped from 42 to 76 ms (Figure 2B) as he developed LBBB (Figure 3A). This phenomenon was reproducible even on single AES (S1-S2-450/270 ms). In view of his abrupt and significant HV prolongation during the appearance of LBBB, a diagnosis of infra-Hisian disease was made. After discussion with patient relatives a shared decision of permanent pacemaker implantation was taken. Considering his age, a cardiac MRI was performed to rule out any underlying cardiac sarcoidosis and cardiomyopathy. The subsequent pacemaker implantation was uneventful (Abbott Medical, DDDR, Endurity MRI).

In fact, his ECG on follow-up revealed incomplete RBBB (Figure 3B) suggesting a tri-fascicular block/delay which was dynamic and intermittent. Furthermore, his device interrogation revealed 12% ventricular pacing requirement despite long AV delay [sensed AVD- 200 ms, paced AVD- 250 ms] and ventricular intrinsic preference (VIP)-"ON" (extra 100 ms). This retrospectively supported our decision of pacemaker implantation.

The decision of permanent pacemaker implantation (PPI) based on a single episode of syncope in the background of fascicular block is often problematic. The conclusion is more controversial when the fascicular block is intermittent. EP study can help in decision-making but the sensitivity and specificity are limited.[1] To improve

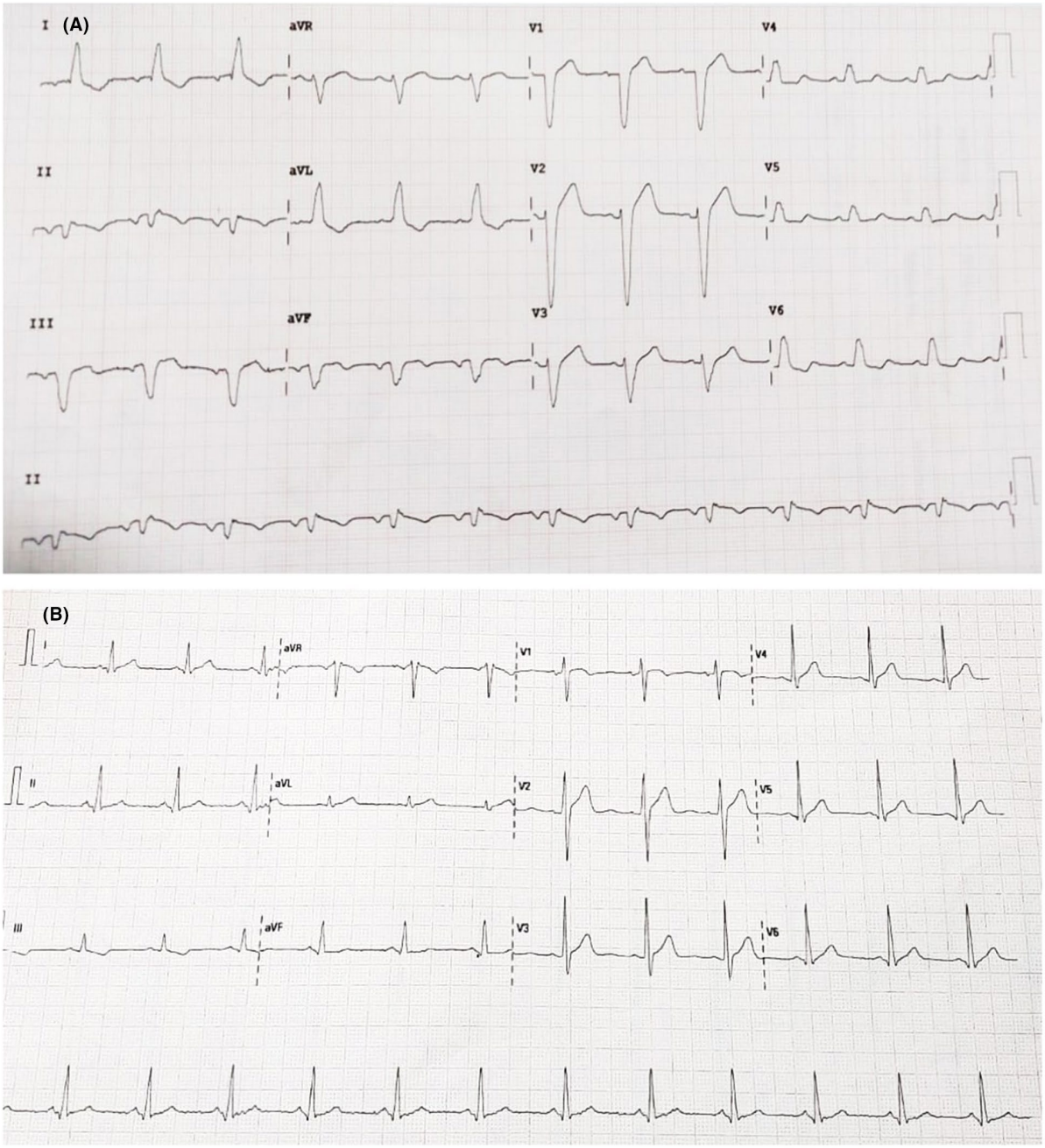

FIGURE 1 (A) ECG on the day of the episode showing complete LBBB. (B) Subsequent ECGs showing normal sinus rhythm with normal QRS complex. The PR interval is also normal making the possibility of equal delay in the left and right bundle unlikely

the sensitivity, some investigators have suggested use of intravenous (IV) ajmaline (when HV is normal / borderline),[1] however, it is unavailable in India. As a substitute, oral flecainide and propafenone are sometimes recommended for off label use but the sensitivity and specificity are poor.[1,2] Moreover, in busy cath labs the use may not be feasible as they are often slow acting.

Atropine and isoproterenol are sometimes useful as they can paradoxically worsen the conduction and reduce the ventricular rate by producing AV block (2:1 or high grade).[1,3,4] These can be useful in determining the level of block noninvasively when Wenckebach or 2:1 are present at baseline. An elegant study by Halpern et al.[5] had studied the effect of isoproterenol on a similar cohort of intermittent bundle branch blocks like the index case. They found that isoproterenol tends to improve conduction related to tachycardia-dependent blocks, but may impair bradycardia-dependent conduction blocks.

Our case was unique as all other maneuvers were negative including atrial pacing which revealed normal supra-Hisian Wenckebach.

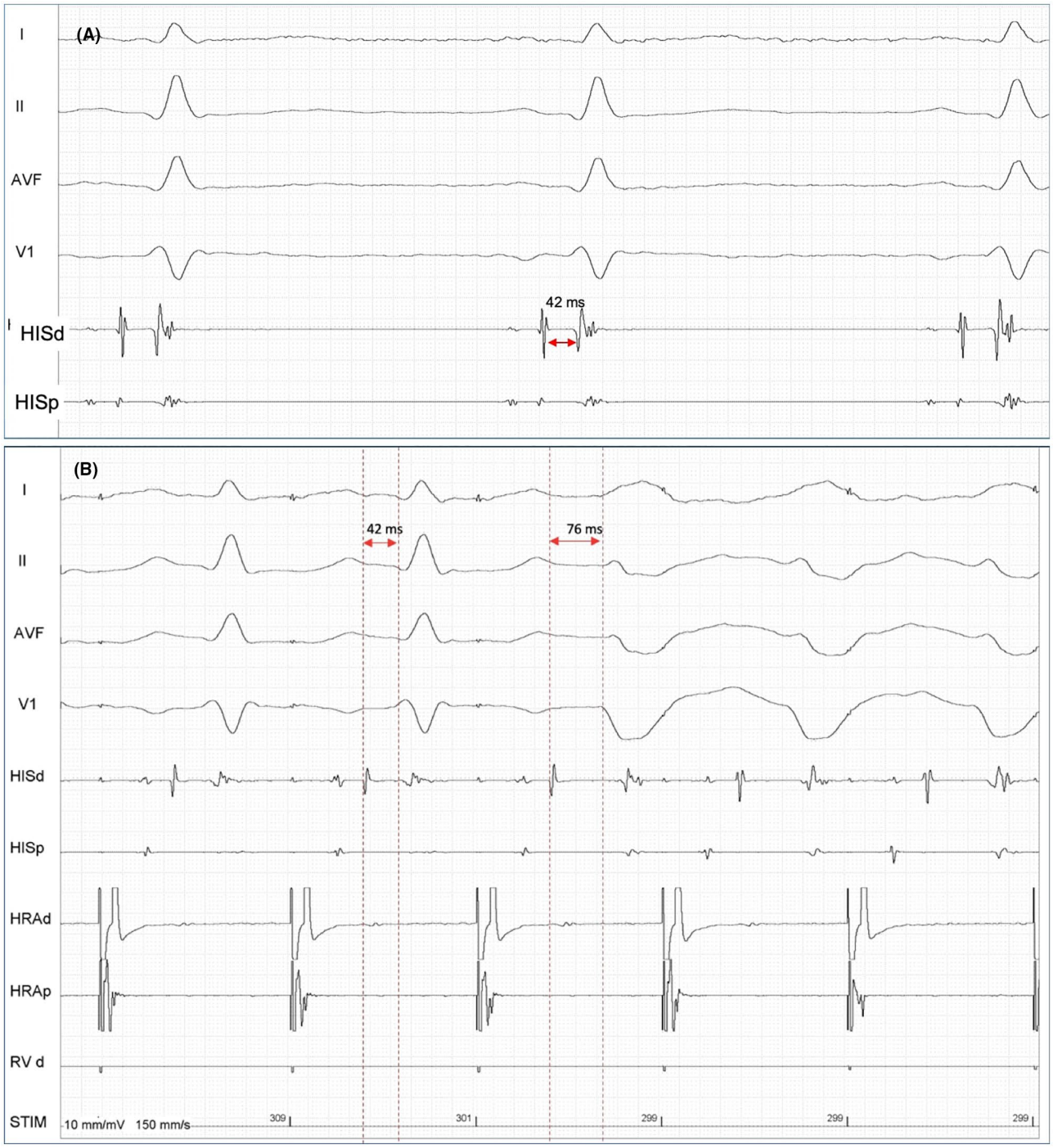

FIGURE 2 (A) Baseline intervals were normal. AH =88 ms, HV =42 ms. During atrial pacing the HV remained same until block (320 ms). (B) Atrial pacing after isoproterenol led to sudden transition into complete LBBB with concomitant prolongation of HV (76 ms) at PCL of 300 ms. [PCL-pacing cycle length]

However, isoproterenol was able to stress the His-Purkinje (HP) system by allowing 1:1 AV nodal conduction up to pacing cycle length of 250 ms. The development of clinical LBBB and concomitant HV prolongation was unmasked which confirmed significant infra-Hisian disease. It appears that isoproterenol should become a part of bradycardia-EP study, especially when the AV Wenckebach at baseline is longer than 300–400 ms. If the AV Wenckebach at baseline itself is <250 ms and AH-HV intervals are normal, isoproterenol might be optional as a fair assessment of the HP system at a faster rate is already made.

INFORMED CONSENT

Consent has been taken from patient.

CONFLICT OF INTEREST

None.

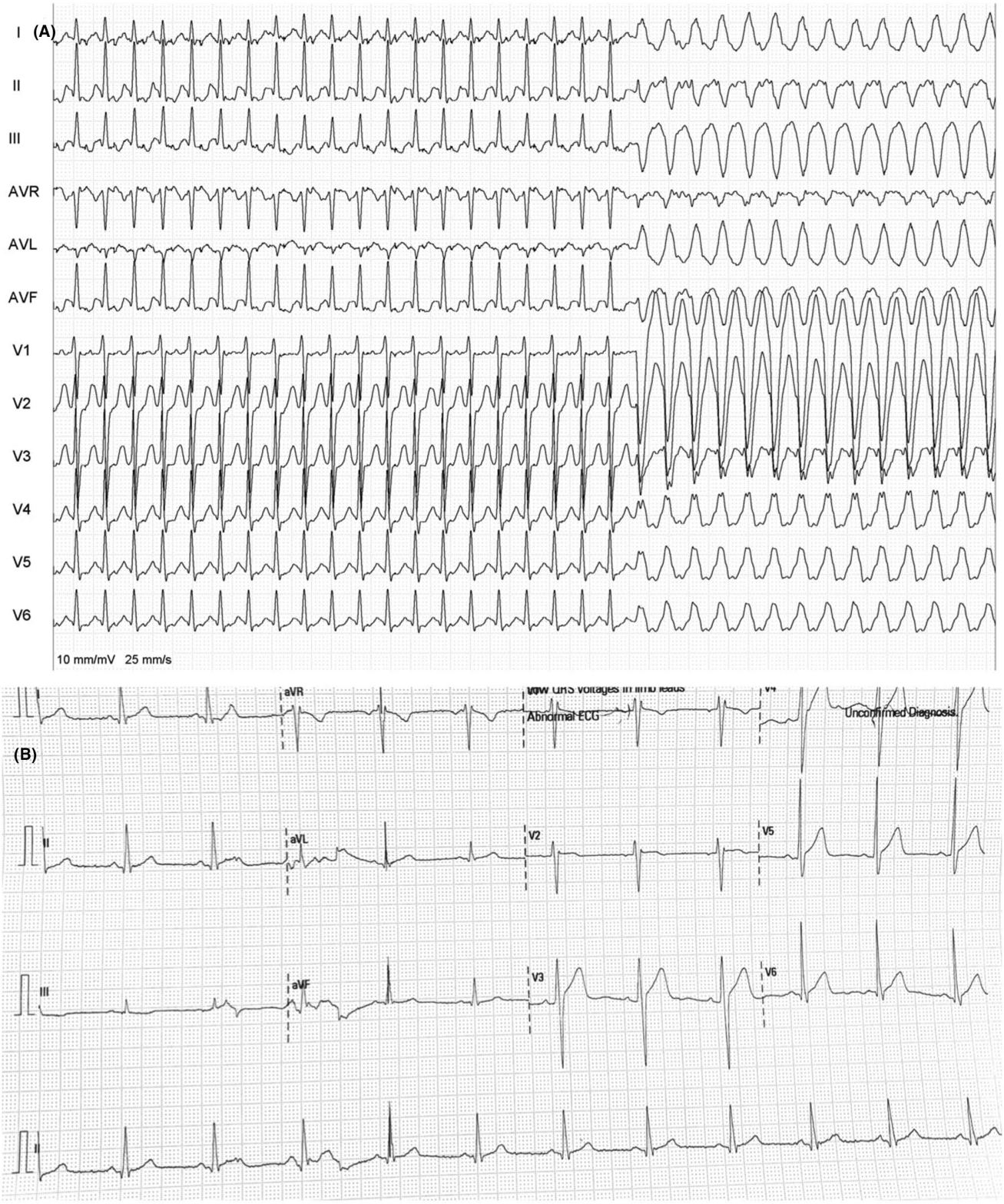

FIGURE 3 (A) Surface ECG during transition into LBBB during atrial pacing after intravenous isoproterenol. (B) Post-pacing ECG on follow-up showing RBBB morphology. Note the presence of S wave in lead 1 along with V1 having rSr'

DATA AVAILABILITY STATEMENT

All raw data and recording during the case are available for review.

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