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Delayed reversible atypical type I second degree atrio-ventricular block in a patient undergone slow pathway radiofrequency ablation: A case report and a short review of the literature

We here describe a case of a 59-years-old man, who underwent an electrophysiology study (EPS) because of recurrent episodes of palpitations and documented narrow QRS complex tachycardia. Supraventricular tachycardia with a cycle length of 460 ms was induced and a diagnosis of a slow-fast atrioventricular nodal reentry tachycardia (AVNRT) was done. Two RF pulses were delivered for a total duration of 80 s. Fig. 1 shows the 3D mapping and the electrogram on the ablation catheter at the time of the first radiofrequency application. Post-ablation there was no change in AH and HV intervals, compared to baseline. No further tachycardia was also induced.

The patient was admitted to the telemetry ward for a night, as per standard of care in our Department. The hospitalization was unremarkable. Since no arrhythmia was detected, the telemetry was suspended right before performing a transthoracic echocardiogram, which confirmed pre-ablation findings and absence of pericardial effusion. Right after the echocardiogram, patient felt palpitations and dizziness for a few seconds while standing up. Twelve lead ECG was recorded initially while the patient was lying flat and showed sinus rhythm with a heart rate of 78/min and a PR interval of 176 ms. Since the symptoms were triggered by orthostatism, another ECG was recorded after standing up and showed sinus tachycardia at a rate of 140/min in association with a 2nd degree atrio-ventricular (AV) block Mobitz I (Fig. 2A), which was expected since his anterograde Wenckebach in the electrophysiology lab was of 460 msec. Blood pressure didn't show any significant drop during the change in position. After a few seconds, heart rate slowed down and II degree AV block disappeared as well. Since the symptoms were reliably reproduced by standing up from a reclining position and relieved by lying back down, we thought the patient was affected by postural orthostatic tachycardia syndrome (POTS). Patient was therefore re-started on bisoprolol 5 mg once daily (half of the original dose due to the Wenckebach at 460 ms). He remained monitored for the following 24 h, with no evidence of recurrence of further AV conduction abnormalities. ECG was recorded both in a reclining position and standing up (Fig. 2B), with evidence of normal sinus rhythm and no signs of atrio-ventricular conduction impairment. Patient was therefore discharged home. Unfortunately, 5 days after discharge (7 days after ablation), the patient presented himself at the emergency room of our hospital, complaining of irregular beats, fatigue and dyspnea. ECG showed atypical type I second degree AV block with an average heart rate of 43/min (Fig. 2C). Bisoprolol was immediately withdrawn and the patient was admitted for electrocardiographic monitoring. An echocardiogram was performed and ruled out pericardial effusion, valvulopathies or change in left ventricular systolic function. Since there was no change in AV conduction in the 48 h following the re-admission, a systemic corticosteroid treatment was started (methylprednisolone 1 mg/Kg). Four days after having started methylprednisolone, ECG progressively normalized (Fig. 2D and 2E). The treatment with corticosteroids was tapered off and stopped within a week from discharge. A 24-hour Holter ECG performed 30 days after the ablation showed finally evidence of constant sinus rhythm with normal AV conduction and normal chronotropic competence (the maximum heart rate recorded on the Holter ECG was 133 bpm with 1:1 conduction between the atria to the ventricles).

Digging in the scientific literature in order to find similar cases, we have noted that the publications on the topic (i.e. AV conduction disturbances after slow pathway ablation) are old and spare. There is furthermore a lack of evidenced-based data on the possible treatments of this rare complication, not considering permanent pacing of course. We have therefore decided to write a mini-review on the topic, trying to focus on the causes and on the treatment for this undesirable but fortunately rare complication.

RF catheter ablation is a well-established treatment option for AVNRT, with a very high success rate [1,2]. One of the most dreadful complications of this procedure is complete AV block (CAVB). Fast pathway ablation is indeed known to be linked to a rate of post interventional CAVB up to 5–10% [3–5]. Slow pathway RF ablation has demonstrated instead to be a safer target for ablation of these arrhythmias when compared to fast pathway [5,2,6,7].

Immediate appearance of AV conduction disturbances or CAVB after RF delivery is usually related to a direct injury of the fast pathway. Not infrequently though, the damage of the fast pathway is collateral. Transient episodes of AV conduction disturbances or CAVB could also be related to bumping of the AV node, due to inadvertent catheter dislodgment during ablation due to tachycardia or patient movement. Even if transient CAVB developed within the ablation procedure seems to be associated with an overall good prognosis [8], the risk of occurrence of late permanent CAVB is not negligible.

If it is relatively simple to imagine the cause of AV conduction disturbances occurring right after RF delivery, it is more challenging to explain the pathogenesis of delayed or late CAVBs. They are

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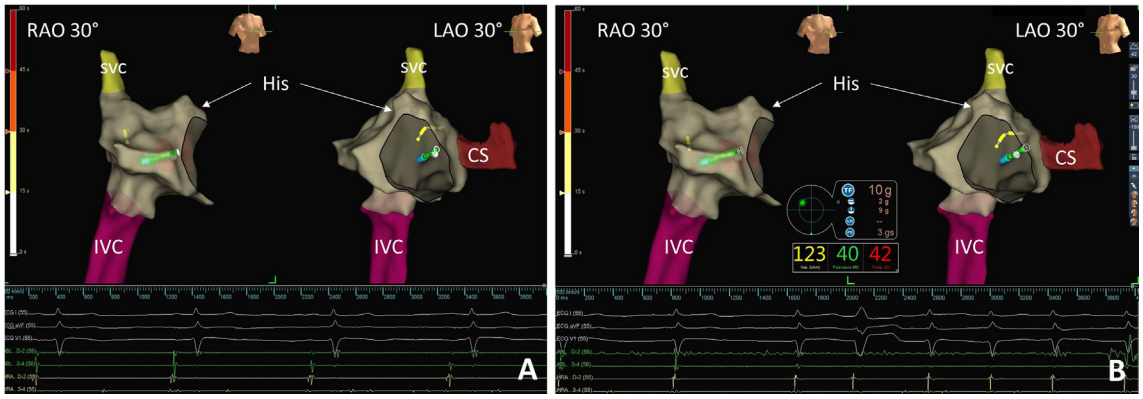

Fig. 1. Two projections (Right Anterior Oblique – RAO 30° and Left Anterior Oblique – LAO 30°) of the 3D geometry of the right atrium and correspondent electrograms before (Panel A) and during ablation (Panel B).

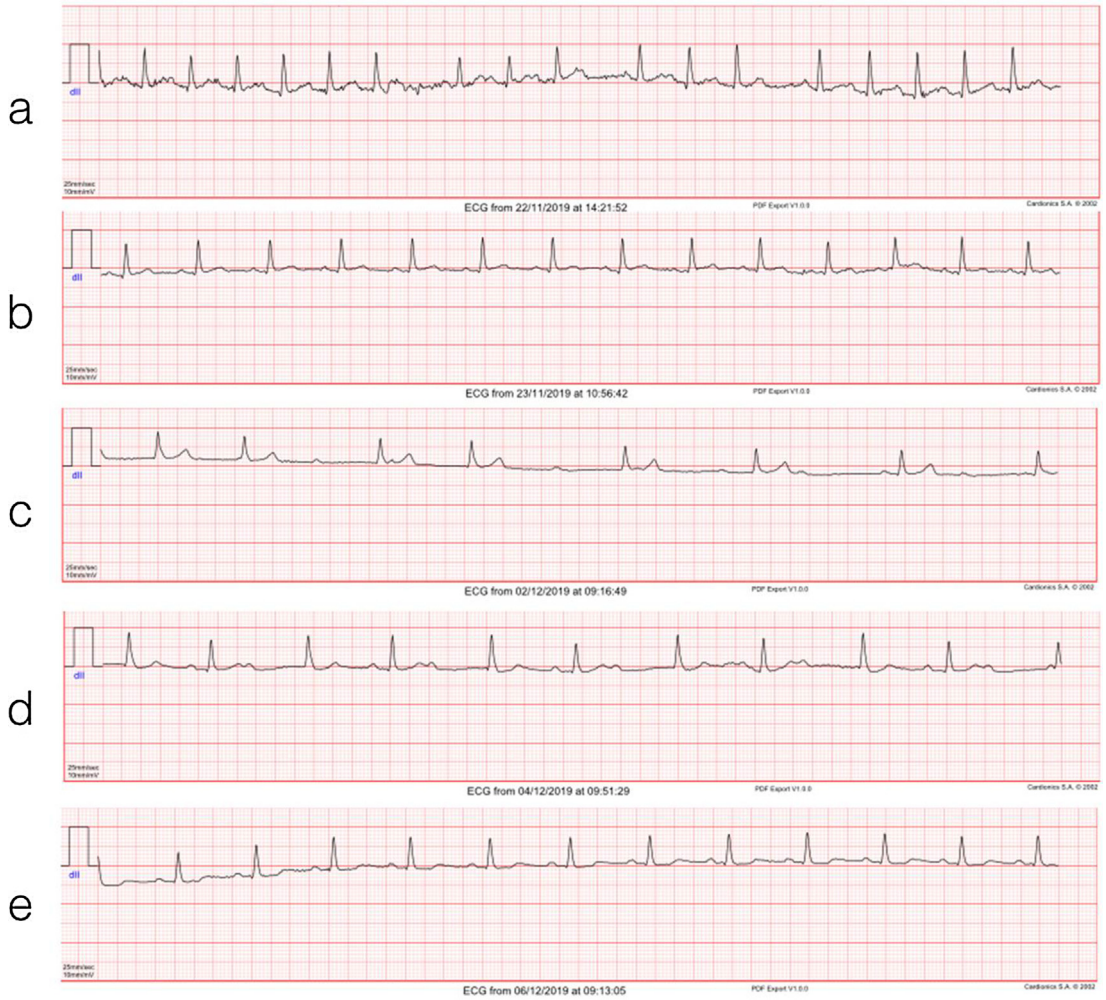

Fig. 2. a) Lead II ECG the day after the ablation standing up, showing typical type I second degree AV block; b) lead II ECG the day of discharge from the hospital, while patient was taking bisoprolol 5 mg once daily; c) lead II ECG, 7 days after the ablation, showing atypical type I second degree AV block; d) lead II ECG, 12 days after the ablation, showing still atypical type I second degree AV block but with less degree of increase of PR interval during the Wenckebach phenomenon; e) lead II ECG , 15 days after the ablation (6 days of corticosteroid therapy), showing sinus rhythm with a complete recovery of the AV conduction.

probably related to the development of inflammation and/or edema around the direct lesion provoked by RF. At present, there is lack of randomized trials or international multicentric studies showing solid data on therapeutic options in patients with RF related AV conduction disorders. There is though a bunch of case reports showing positive data on corticosteroids administrated for those patients with delayed CAVB [9–11].

Our case is interesting for different aspects. The first clinical manifestation of the patient was POTS the day after RF ablation. The reason why our patient developed POTS after the ablation

can be only speculated. The history of previous bariatric surgery and the possible autonomic dysfunction described after this type of surgery could have played a role in favoring this clinical event [12]. Indeed, the second relevant aspect deriving from our case report is a confirmation that delayed AV conduction disturbances are possibly sensible to corticosteroids. The effect of corticosteroids is supposed but could not be demonstrated with certainty, because a natural resolution of the inflammation or the withdrawal of betablockers can have also contributed to the restoration of the normal AV conduction.

Finally, with our case report, we liked to underline how important could be to follow a mechanistic approach in medicine: more specifically in our case, we tried to reject all possible reversible causes of AV conduction disturbances and give a chance to anti-inflammatory medications to do their course, before opting for implantation of a permanent pacemaker.

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