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CASE REPORT

Third-degree AV block sensitive to prednisolone 72 hours post AVNRT ablation

Abdul S. Parwani, Anna I. Schröder, Daniela Blaschke, Florian Blaschke, Martin Huemer, Philipp Attanasio, Burkert Pieske, Leif-Hendrik Boldt & Wilhelm Haverkamp

Department of Cardiology, Charité – Universitätsmedizin Berlin, Campus Virchow-Klinikum, Berlin, Germany

Correspondence
Abdul S. Parwani, Department of Cardiology, Charité – Campus Virchow Klinikum, Augustenburger Platz 1, 13553 Berlin, Germany. Tel: +49-30-450-665410; Fax: +49-30-4507665410; E-mail: abdul.parwani@charite.de

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Key Clinical Message

A patient developed a transient first-degree AV block during a radiofrequency ablation of an atrioventricular nodal reentrant tachycardia. Three days later the patient presented with a third-degree AV block. It resolved within 24 h under antiphlogistic therapy. Patient was asymptomatic without necessity for pacemaker implantation at 12 months follow-up.

Keywords

Ablation, atrioventricular node reentry, emergency medicine, third-degree AV block.

Case Report

A 45-year old male patient with recurrent symptomatic documented supraventricular tachycardia for the last 8 years was referred to our department for an electrophysiological (EP) study. His symptoms consisted of fast palpitations and dyspnea. No history of syncope or angina pectoris was present. The ECG during the symptoms showed a narrow QRS complex tachycardia at 180 beats/min (bpm). The patient had no structural heart disease and no resting ECG abnormalities. An arterial hypertension and a diabetes mellitus type II had been well treated for the last couple of years.

The EP study was performed under a fasting state and under sedation with midazolam. At the time of the procedure the patient was in sinus rhythm (SR) (heart rate (HR): 76 bpm; PQ-interval: 156 msec; QRS-interval: 82 msec).

One diagnostic catheter was placed in the higher right atrium (HRA), one in the apex of the right ventricle and one in the coronary sinus (CS). The AV node conduction time (AH-interval) and conduction time through His-Purkinje system (HV-interval) were normal, with 115 msec and 48 msec, respectively. Using progressive atrial extrastimulus testing and under administration of orciprenaline, a typical atriovenricular node reentry (AVNRT) tachycardia with a cycle length (CL) of 330 msec could be induced. Conventional mapping of the inferior AV-node was performed. First, the inferoposterior site of the septal annulus of the tricuspid valve at CS ostium was targeted. The location was confirmed by fluoroscopy, and on typical electrophysiological signals for slow pathway, 10 RF impulses (one RF impulse = 60 sec) were delivered at and around this site but with a transient effect. For stable catheter position and a better result a long sheath was insert and six RF impulses were applied at a more medial site, during which junctional rhythm occurred. The mean temperature that was achieved was 45°C and the mean energy that was delivered was 35 watts. In all attempts, the longest possible distance from the AV node was kept. Following the last energy delivery, a first-degree AV block occurred for approximately 30 sec. The ECG showed a SR with a HR of 76 bpm, PQ-interval of 210 msec, and a QRS-interval of 85 msec. The AH- and

HV-interval did not show any changes. Incremental atrial pacing hereafter and at the end of the study showed no dropped beats. During the observational time of 20 min, AVNRT was not inducible anymore, also not under administration of orciprenaline (a cumulative dose of 0.5 mg over 15 min). At the end of the study, the AV node effective refractory period (AVERP) was 370 msec and the retrograde AVERP was 430 msec. The ECG at the end of the study showed a normal SR (HR 78 bpm; PQ-interval 185 msec; QRS-interval 80 msec).

Before discharge the patient was monitored by telemetry for 24 h on our ward. The ECG did not reveal any ventricular or supraventricular disturbances throughout the time. The ECG at discharge showed a normal SR (HR 85 bpm; PQ-interval: 176 msec; QRS-interval 82 msec). Pericardial effusion before discharge was ruled out. The patient was discharged without any complains. The initial symptoms had totally resolved at discharge.

Two days later the patient presented to our emergency department complaining of palpitations and lightheadedness. The resting ECG showed a third-degree AV block with a high junctional rhythm (HR: 65 bpm; QRS-interval 85 msec) (Figure 1). The patient was admitted to our telemetric monitoring ward and permanent pacemaker implantation was discussed.

An echocardiography was also performed during the second admission. There was no sign of pericardial effusion or any other changes compared to the echocardiography done during the first admission.

As the AV block was interpreted as a consequence of the prior RF ablation, we immediately initiated an antiphlogistic therapy with an intravenous bolus (iv) of 250 mg prednisolone, which was continued orally for another 5 days with 50 mg/day. Additionally, ibuprofen 600 mg twice daily was administered.

Under this therapy a regression of the third-degree AV block was observed in the telemetric monitoring. Shortly after the initial administration of the prednisolone iv bolus second-degree AV block was documented (Figure 2). The following 2 days, there were only a few dropped beats, predominantly at night. By the end of day 5 of antiphlogistic treatment the telemetric monitoring showed a regular SR without recurrence of the patient's symptoms. The ECG at resolution of complete AV block, showed a SR with a HR of 69 bpm (PQ-interval 224 msec and QRS-interval 86 msec) (Figure 3). The Holter ECG after therapy administration and before discharge revealed a normal SR during the recording. Minimal HR was 54 bpm and maximum 115 bpm. The mean HR was 75 bpm. Five episodes of isolated *P*-wave block were observed during the night. The patient was discharged without the need of a permanent pacemaker implantation.

After a 1 year follow-up period the patient presented to our outpatient clinic without any symptoms. The ECG at one-year follow-up showed a normal SR with a HR of 78 bpm (Figure 4). The PQ-interval was 180 msec and the QRS-interval 92 msec. The Holter ECG (24 h) at one-year follow-up revealed a normal SR with a minimal HR of 54 bpm and maximal HR of 120 bpm. The mean HR was 74 bpm. One asymptomatic dropped beat occurred during the night while patient was sleeping.

Discussion

We report a case of a third-degree AV block sensitive to prednisolone occurring 72 h after a RF ablation for the treatment of an AVNRT.

AVNRT is one of the most common supraventricular tachycardia, affecting most commonly patients of young age. RF ablation is, even though an invasive therapy, currently the first-line treatment for symptomatic AVNRT patients with success rates exceeding 90% after the first attempt. AV block during or after a RF ablation is a rare but feared complication, occurring in approximately 5–10% of cases of fast pathway versus 1–2% of slow pathway ablations [1]. Thus, modification of the slow pathway is the preferred type of procedure. In most cases, an AV block appears within the first 24 h after the procedure. Pathophysiological reasons are either direct damage to the AV node or increased vagal tone caused by pain during the ablation procedure or by direct stimulation of parasympathetic nerve fibers of the atrium [2].

Risk factors associated with the development of AV block after successful slow pathway modification are a prolonged PR-interval prior to the procedure, which seems to have a higher incidence in older patients and in those with structural heart disease [3]. Furthermore ablation at the near side of AV node and the appearance of a transient AV block during the procedure both have a strong positive predictive value of development of a high-degree AV block thereafter [4, 5]. All of these criteria were not present in our case.

Nevertheless, in clinical practice most patients with an AVNRT, do not have any structural heart disease or other serious comorbidities. The presence of a high-degree AV block as a complication of an RF ablation would inflict the need of a permanent pacemaker implantation that might not be well accepted by the patient regarding his long expected lifetime and the overall benign nature of AVNRT.

The rapid recovery of the AV node conduction in the presented case after administration of an antiphlogistic medication is opposed to the common notion that high-degree AV block after RF ablation is a consequence of irreversible damage to the AV node with the necessity of permanent pacemaker implantation.

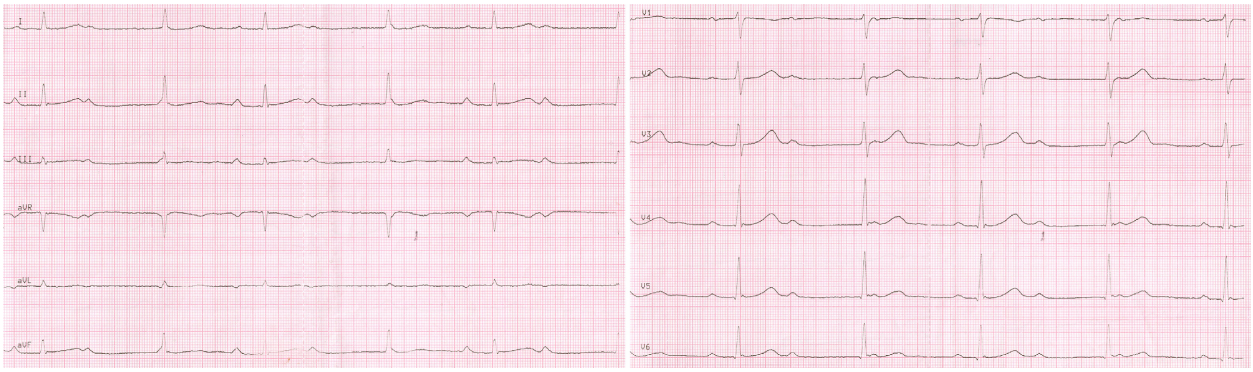

Figure 1. ECG showing complete AV block, 3 days after AVNRT ablation.

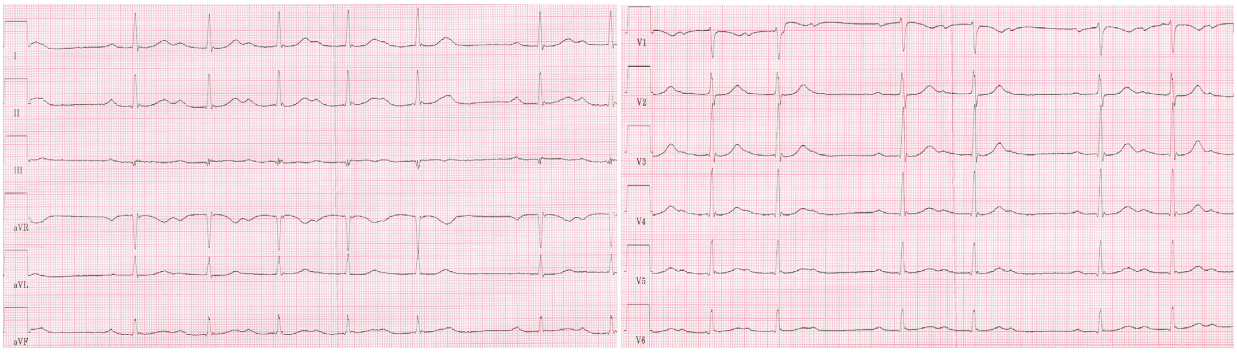

Figure 2. ECG after initiation of antiphlogistic therapy.

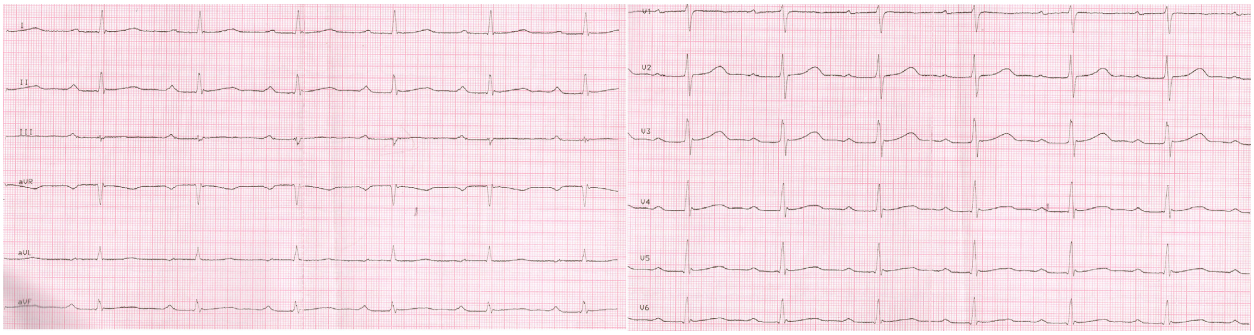

Figure 3. ECG at complete resolution of third degree AV block, showing a sinus rhythm with a first-degree AV block.

A possible explanation for the delayed development of a symptomatic AV block in our patient could be that around the region of RF application inflammation and edema caused impaired myocardial and AV node conduction. Several cases have described late AV block formation after RF ablation reaching from several hours to several months after the ablation with the need of permanent pacemaker implantation. Largely, this had been explained by late tissue retraction and scar formation around the ablated area. Elhag et al. [6] described two cases of high-degree AV block occurring several months after RF ablation. They argued that after successful RF ablation there is a continuous healing process in the RF lesion, marked by inflammation, leading to formation of fibrosis over a

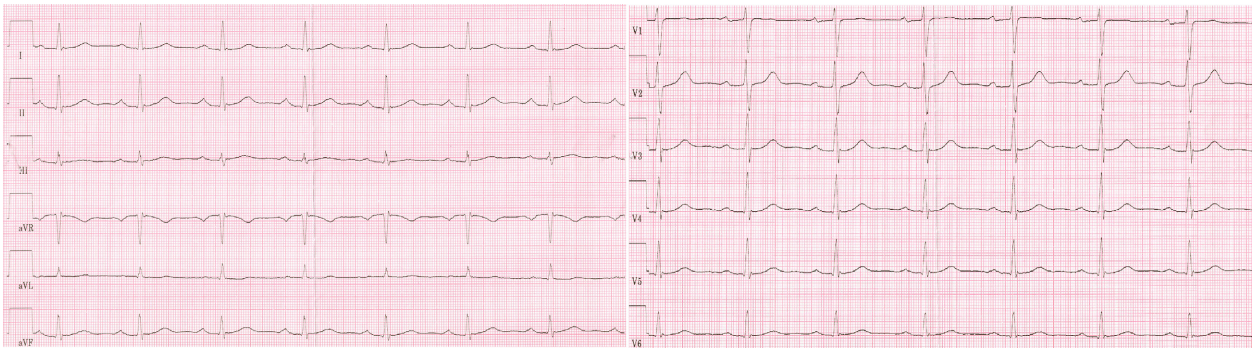

Figure 4. ECG at 1 year follow up, showing a normal sinus rhythm.

period of weeks to months. This fibrosis could lead to extension of the initially ablated area leading to late conduction disturbance [6]. Haissaguere et al. described "a progression of tissue lesion" as a cause of the appearance of prolonged PR intervals or a Wenckebach block following several days after RF ablation [7].

Our results show a progressive decrease in PQ-interval after administration of anti-inflammatory drugs over a few days. These findings suggest that the reversibility of the AV block in the presented case is due to the early administration of antiphologistic medications. We believe that this could have prevented the development of late thermal injury that would have otherwise caused irreversible damage to the conduction properties of the AV node with the necessity of permanent pacemaker implantation.

Conclusion

Our case underlines the progressive and variable tissue response to RF application. We suggest that at least in the case of delayed development of AV block following RF ablation, the initial inflammatory response with edema and tissue swelling with the possibility of a late development of fibrosis and an irreversible damage to the AV node could be successfully treated and prevented with antiphlogistic medication.

In our opinion, this could be of great potential concerning the young age of the patients often undergoing the procedure and the burden of a permanent pacemaker implantation.

Authorship

ASP, WH: conceived the data. ASP, MH: collected ablated data. AS, DB, PA: collected and analyzed ECG. FB, LHB: performed medical therapy and follow-up. ASP, AS, LHB: drafted the article. BP, WH: critically revised the article. ASP, BP, WH: approved the article.

Conflict of Interest

None declared.

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