Treatment of Hepatitis C Virus with Peginterferon and Ribavirin May Trigger Atrioventricular Nodal Reentrant Tachycardia in An Intensive Care Unit Patient

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INTRODUCTION

Hepatitis C virus (HCV) infection is often asymptomatic, but chronic infections can lead to liver scarring and ultimately cirrhosis that is generally apparent after many years. The current treatment of HCV is a combination of peginterferon α2b (Peg-IFN) and ribavirin for 24 or 48 weeks⁴. The treatment of HCV with Peg-IFN and ribavirin is more effective than with conventional interferon and ribavirin². Despite the important effects of Peg-IFN and ribavirin therapy, many side effects, such as fever, fatigue, anxiety, irritability, neutropenia and thrombocytopenia, have been reported⁵. In addition, cardiac arrhythmias and syncope are rare consequences of Peg-IFN therapy⁶. In this study, we report an intensive care unit patient who developed atrioventricular nodal reentrant tachycardia (AVNRT), which is the most common type of reentrant supraventricular tachycardia, and presyncopal attack during Peg-IFN and ribavirin therapy.

CASE REPORT

A 46-year-old man developed palpitation and presyncopal attack within 10 weeks of starting Peg-IFN (150 μg/week) and ribavirin (1200 mg/day) therapy for the treatment of chronic HCV. He had developed palpitation approximately 20 years ago and had not taken any medications.
In the intensive care unit, surface electrocardiogram (ECG) and 24-h Holter ECG revealed atrial premature tachycardia (AVNRT) (Figure 1A). Tachycardia stopped after intravenously administering 5 mg metoprolol (Figure 1B). Transthoracic echocardiography and thyroid hormone and other routine laboratory tests were normal. We offered electrophysiology study and catheter ablation therapy to the patient, but he refused them. After the AVNRT attack, the Peg-IFN dose was decreased (120 μg/week) and 50 mg/day metoprolol was initiated. After 10 weeks, AVNRT occurred again during infusion. The patient refused the ablation therapy again. Therefore, the metoprolol dose was increased to 100 mg/day. Close follow-up revealed neither palpitation nor presyncope, and HCV-RNA was undetectable at 24 and 48 weeks of treatment.

**DISCUSSION**

The side effects of Peg-IFN and ribavirin combination therapy for HCV have been documented; however, cardiac arrhythmias are rarely reported\(^2\). Moreover, AVNRT is not associated with Peg-IFN therapy in the literature.

AVNRT is the most common regular supraventricular tachycardia observed in intensive care unit, originating above the His bundle\(^4\). It occurs when a reentry circuit forms within or just next to the AV node. The circuit usually involves two anatomical pathways (dual AV nodal physiology): the fast and slow pathways, which are usually targeted for ablation\(^5\). The slow pathway is located inferior and slightly posterior to the AV node, often following the anterior margin of the coronary sinus. The fast pathway is usually located just superior and posterior to the AV node. The AVNRT therapy can be used for AV nodal slowing drugs, such as adenosine, beta-blockers, as in our case, or non-dihydropyridine calcium channel blockers. Furthermore, for cure, patients can undergo an electrophysiology study via a generally slow pathway ablation.

In this case, these drugs may cause atrial premature complexes during infusion, and the atrial premature complexes may in turn affect the dual pathway physiology. Finally, AVNRT may be triggered. To cure this rhythm, we offered the electrophysiology study and catheter ablation therapy to the patient, but he refused them. Therefore, we had to continue the medical therapy with beta-blockers.

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**Figure 1.** (A) The surface electrocardiogram revealed AVNRT. (B) Tachycardia stopped after an intravenous administration of 5 mg metoprolol.
REFERENCES