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A left lateral accessory pathway unmasked by rivastigmine ☆

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Abstract A 75-year-old woman was referred for advice regarding surface electrocardiographic modifications after the initiation of rivastigmine. In our patient, the baseline ECGs appeared perfectly normal. However, the initiation of a cholinesterase inhibitor unmasked a left lateral accessory pathway that had never been diagnosed before. Although cholinesterase inhibitors are known to increase vagal tone, the PR interval was shortened after rivastigmine administration, thus excluding this hypothesis to explain the appearance of the accessory pathway. Therefore, we hypothesized that cholinesterase inhibitors may have increased conduction velocity in the accessory pathway or in the atria.

Keywords: Accessory pathway; Cholinesterase inhibitors; Electrocardiogram

Clinical case

A 75-year-old woman was referred to our institution for advice regarding surface electrocardiographic modifications after the initiation of rivastigmine. This patient had a history of high blood pressure treated with propranolol 40 mg and no other significant cardiovascular symptoms. She had recently been diagnosed with mixed dementia, manifesting as a rapid and severe decrease in cognitive functions associated with temporal and spatial disorientation, for which rivastigmine (4.5 mg patch) was prescribed. Baseline ECGs were considered normal before and during the first 48 h of treatment (Fig. 1). The first ECG showed a regular sinus rhythm at 80 bpm, with a normal atrioventricular delay as indicated by the normal PR interval of 180–190 ms. The QRS axis was normal.

Two months later, the patient was seen by the geriatrician to assess both clinical tolerance to the treatment and cognitive status. No medical issues came to light during the clinical exam. Surprisingly, the ECG recorded at that time was markedly different from the previous one (Fig. 2). On the second ECG recording two months later, the sinus rhythm was slightly slower at 75 bpm. However, the PR interval was shorter than on the previous ECG (80–100 ms) and there was a delta wave responsible for an enlarged QRS complex with a right bundle branch block pattern. The positive pre-excitation in the anterior leads suggested left localization of the accessory pathway (AP). The presence of a QR pattern in lead I, and positive polarity of the QRS in lead III suggested a left lateral position of the AP around the mitral annulus (M2) [1,2].

By precaution, rivastigmine was interrupted and a new consultation was planned for 2 weeks later. Two weeks after the interruption of rivastigmine, ECGs were identical to baseline ECGs recorded before the medication. The patient remained asymptomatic. After the Valsalva maneuver (in order to slow conduction through the atrioventricular (AV) node and thus to help unmask conduction through the accessory pathway), we were able to temporarily record some pre-excited QRS complexes (Fig. 3), confirming the presence of a concealed anterograde left lateral AP.

Discussion

Rivastigmine is a cholinesterase inhibitor, which is commonly prescribed to treat dementia. The cardiovascular effects of cholinesterase inhibitors are complex, but they generally increase vagal tone in the heart conduction network and may promote bradycardia [3] One Canadian study, which analyzed 1.4 million patients >67 years, reported that the recent intake of acetyl-cholinesterase inhibitors increased the risk of hospitalization for bradycardia [4]. Another study also demonstrated that the use of cholinesterase inhibitors was associated with increased rates of syncope, bradycardia, pacemaker insertion, and hip fracture in older adults with dementia [5].

In our case, the initiation of rivastigmine unmasked a left lateral accessory pathway that had never been diagnosed before. Due to faster conduction through the AV node than

☆ Conflicts of interest: none.

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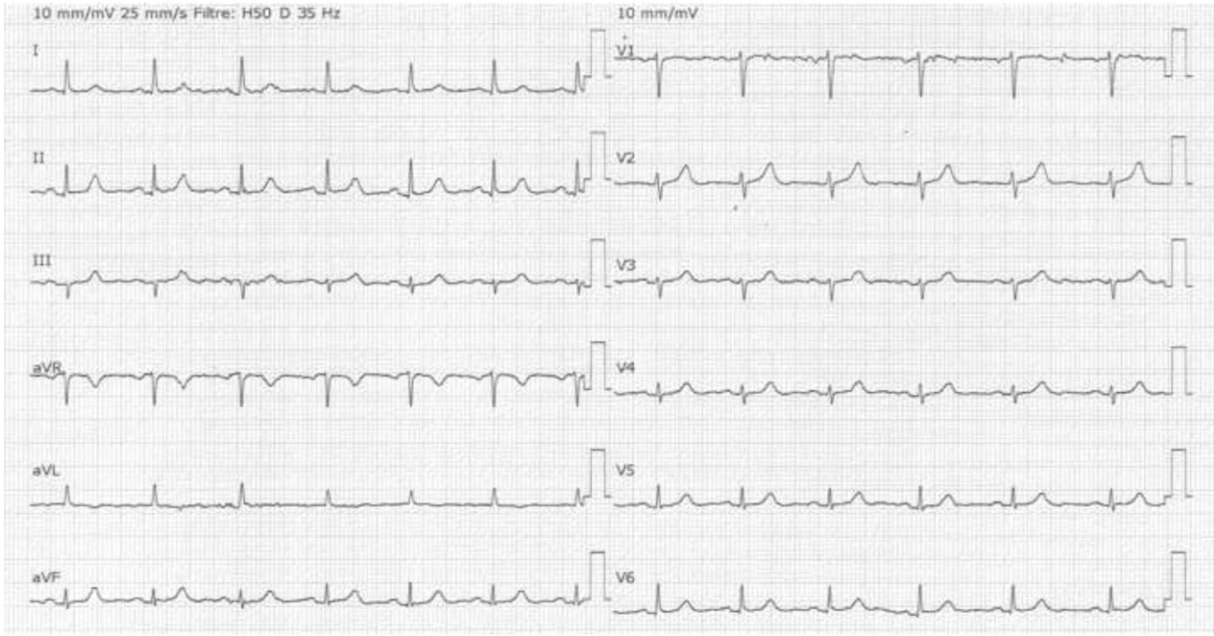

Fig. 1. Baseline ECG.

through distant left AP, pre-excitation on standard ECG tracings may sometimes be subtle. In our patient, the baseline ECGs appeared perfectly normal. Our first hypothesis was that rivastigmine may have contributed to the unmasking of a pre-excitation syndrome by increasing vagal tone on AV nodal conduction. However, this hypothesis was not supported by the two ECG recordings. Fig. 1 shows the pre-rivastigmine ECG with normal sinus rhythm without manifest pre-excitation and a PR of around 180–190 ms. This suggests that the time from the beginning of atrial activation to ventricular activation through the accessory pathway was >190 ms before the initiation of rivastigmine.

Fig. 2 shows normal sinus rhythm with manifest pre-excitation and a PR of 80–100 ms two weeks after rivastigmine initiation. This suggests that the time from the beginning of atrial activation to ventricular activation through the accessory pathway had decreased to 80–90 ms. This may have occurred via two possible mechanisms:

- Atrial conduction velocity was increased by rivastigmine (possibly by improved conduction through Bachman's bundle).
- Accessory pathway conduction velocity was increased by rivastigmine.

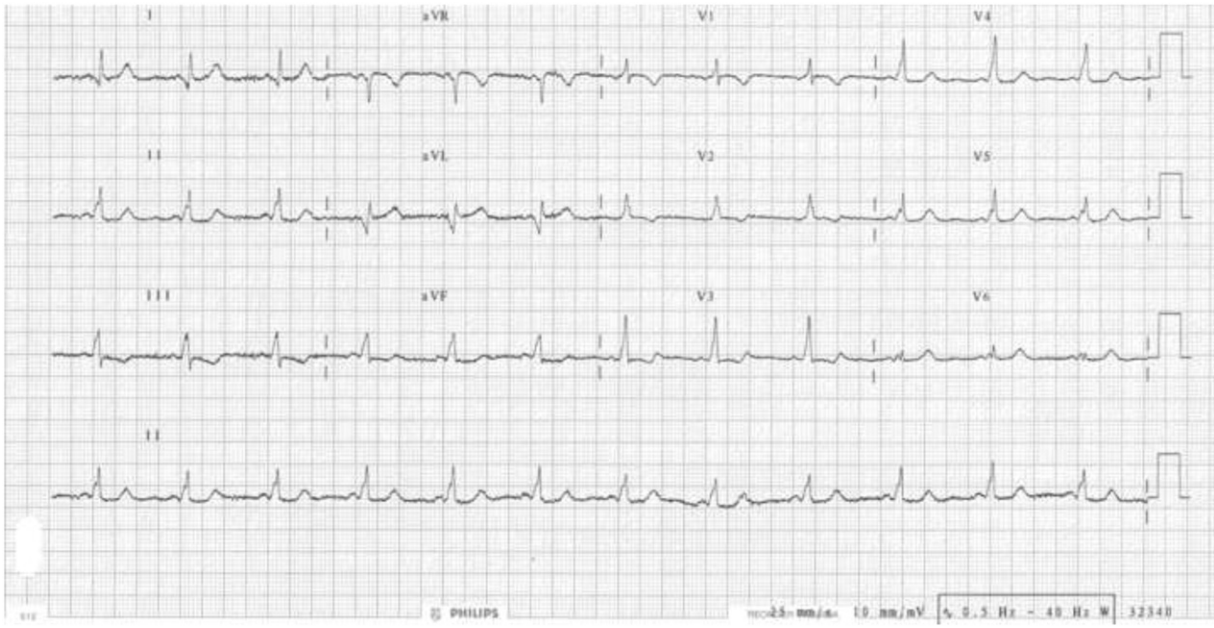

Fig. 2. ECG two weeks after rivastigmine initiation. The PR interval is short with a delta wave responsible for an enlarged QRS complex with right bundle branch block pattern. The positivity of the preexcitation in anterior leads may indicate left localization of the accessory pathway.