Weakness and Near Syncope in a 79-Year-Old Woman

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A 79-year-old woman with a past medical history of systemic arterial hypertension, paroxysmal atrial fibrillation, and chronic kidney disease (stage 3) was admitted for evaluation of weakness, malaise, and near syncope. An electrocardiogram was recorded and is shown in the figure below.

Figure. Electrocardiogram recorded on admission.

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DIAGNOSIS: Sinus bradycardia and arrhythmia, high-grade second degree atrioventricular (AV) block with only one conducted P wave, a slow junctional escape rate, left atrial enlargement, and nonspecific T-wave changes.

The P waves are upright in all of the limb leads, except AVR where they are inverted. Therefore, the rhythm is sinus, but the rate of the sinus node is only 37/min, and its rhythm is slightly irregular. Of the 6 R-R intervals, five are regular, and all of the P-R intervals are different; thus, there is AV dissociation with a junctional escape rhythm at a rate of 39/min. The first five P waves occur when the AV conduction system should not be refractory, but only the fourth P wave is conducted to the ventricles as indicated by the shorter fourth R-R interval and by the slightly different configuration of the fifth QRS complex compared to the others. Accordingly, the incomplete AV dissociation is due to high-grade second degree AV block, and the overall ventricular rate of only 41/min is the cause of the patient’s weakness and presyncope. The narrowness of the QRS complexes and the long P-R interval (0.32s) of the one conducted complex indicate that the site of the AV block is almost certainly in the AV node. The P waves are broad (0.12s) with a +/- configuration in the inferior leads and a bifid contour, with >0.04s between the two peaks, in the mid and lateral precordial leads. All of these are features of left atrial enlargement or intratratrial block.1

The patient had felt weak for several days. She admitted to having mistakenly taken an extra dose of her medications, which included extended-release diltiazem 360 mg daily, clonidine 0.3 mg tid, and digoxin 0.125 mg daily. All of these were taken by mouth. This last drug had been discontinued by her primary care physician, but the patient failed to stop it.

Clonidine is a centrally acting agonist of alpha2-adrenoceptors in the brain stem, and thereby decreases sympathetic outflow, which slows heart rate and lowers blood pressure.2-4 Diltiazem is a nondihydropyridine calcium channel blocker that inhibits calcium ions from entering the “slow channels” during phase 2, the plateau phase, of the action potential, prolongs the AV nodal refractory period, slows the sinus node, and causes arterial vasodilatation.5,6 Digoxin is a cardiac glycoside that inhibits Na+/K-ATPase, thus causing a transient increase in intracellular sodium and then an increase in calcium by way of the sodium-calcium exchange mechanism. Increased intracellular calcium lengthens phase 4 of the action potential, which leads to a decrease in heart rate. Digoxin also activates the parasympathetic nervous system, which slows the sinus node and inhibits AV conduction.7,9

This patient was on three drugs which can slow the sinus node, and diltiazem and digoxin can also impair AV conduction.2-9 In addition, clonidine and diltiazem also reduce blood pressure. Thus, her symptomatic bradycardia was no surprise, and similar cases of severe bradyarrhythmias have been described in patients on similar drug combinations.10,11 Furthermore, clonidine, digoxin, and active metabolites of diltiazem depend on the kidneys for elimination,4,5,9 and her 79 years and stage 3 kidney disease set the stage for toxic accumulation of the drugs. Because the patient was in no acute distress, withdrawal of the drugs was the only treatment needed.

REFERENCES

5. Ibid:240-244.

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