The Practitioner
Le praticien

Country cardiograms case 58: Answer

The ventricular rate is 110 beats/min and regular. P waves cannot be seen in lead II, but lead VI clearly shows 2 P waves for each ventricular complex (Fig. 1, on page 20). Although it looks like an atrial flutter with a 2:1 block, the atrial rate is about 220 beats/min and hence more properly diagnosed as a supraventricular tachycardia (SVT) with a 2:1 block (an atrial flutter has a rate of 250–350 beats/min). The PR interval is 120 ms, which is borderline short. The QRS interval is normal at 80 ms, and the QT interval is wide. The P wave morphology is difficult to assess given its fast rate. There are no pathologic Q waves and there is normal R wave progression. There are no acute changes to the T wave or ST segment. In summary, the abnormal changes on this electrocardiogram (ECG) include an SVT with a 2:1 atrial ventricular block rhythm.

An SVT is defined by the American College of Cardiology, American Heart Association and the Heart Rhythm Society as being an atrial and/or ventricular rate greater than 100 beats/min at rest due to an arrhythmia caused by heart tissue involving the bundle of His, or anywhere above it.1 An SVT includes atrial flutter, atrial fibrillation, focal and multifocal tachycardia, junctional tachycardia, atrioventricular nodal reentrant tachycardia and accessory pathway reentrant tachycardia.1 An atrial flutter is a rapid and regular atrial rhythm with a rate of 250–350 beats/min and is caused by reentry of the rhythm within the right atria.2

The most common causes of an SVT include coronary ischemia, heart failure, respiratory failure, shock, anemia, infection, pain or anxiety.3 Patients with an SVT may present with the following symptoms: palpitations, syncope, lightheadedness or dizziness, chest pain, dyspnea and decreased level of consciousness.5

The treatment of an SVT depends on the stability of the patient and the cause of the SVT. If the patient is hemodynamically unstable with a known SVT cause, an attempt should be made to reverse the cause of the SVT.1,3 Synchronized cardioversion is the first-line recommendation for patients with hemodynamic instability and rapid decompensation with an unknown SVT cause, and an intravenous adenosine normal saline push is the most commonly used method for patients who are hemodynamically stable.1,4 Second-line treatments for patients with acute SVT include intravenous calcium channel blockers, such as verapamil or diltiazem, or intravenous β blockers, such as metoprolol.1

Because this patient was hemodynamically unstable, we would normally chosen to do synchronized cardioversion. However, given her goals of care, that was not an option. The ideal option for acute SVT treatment would have been an intravenous adenosine normal saline push. However, because this ECG was inappropriately diagnosed as atrial flutter, a rate-control treatment was considered. The choice between intravenous calcium channel blockers and intravenous β blockers for rate control is largely based on physician and patient preference. β blockers tend to be better for rate control and conversion to sinus rhythm, whereas calcium channel blockers tend to be better for patients with chronic obstructive pulmonary disease or asthma.5 In this case, the patient
was given intravenous metoprolol because she had previously taken oral metoprolol for atrial fibrillation control. Once the metoprolol was administered, the patient’s heart rate slowed and returned to sinus rhythm. We were already treating the cause of the SVT with empiric antibiotics. The patient’s condition improved over the next 12 hours. Her blood and urine cultures surprisingly showed no bacterial growth, but because the clinical picture was clearly that of septic shock, we continued to treat with intravenous ceftriaxone.

REFERENCES

For the question, see page 20.

Competing interests: None declared.