DEVICE ROUNDS

Tachycardia during Coronary Computed Tomography Angiography

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Case Presentation

A 76-year-old woman underwent a coronary artery computed tomography angiography (CTA) to exclude coronary occlusive disease after a segmental inferoseptal hypokinesia was discovered at echocardiography in the workup for atypical chest pain. The patient has a body mass index of 40 kg/m^2, a history of arterial hypertension, hyperlipidemia, hypothyroidism, and type 2 diabetes. Her drug regimen consists of acenocoumarol, bisoprolol, potassium canrenoate, simvastatin, levothyroxine, and metformin.

She also has a history of a second-degree type I atrioventricular block found 18 years earlier during an episode of atrial fibrillation (AF) that has led to several syncopes. A Topaz II pacemaker (Vitatron, Maastricht, the Netherlands) was implanted 10 years ago, replaced 1 year ago, with preservation of the lead, by a Zephyr SR 5620 pacemaker (St. Jude Medical, St. Paul, MN, USA) in the ventricular rate modulated pacing (VVIR mode). The pacemaker control examinations 10 days prior and 6 months after CTA showed a normally functioning stimulator with a virtually permanent ventricular pacing and no alert between both examinations. Stimulation (0.75 then 0.75 V) and sensing (9.6 then 9.3 mV) thresholds and lead impedance (827 then 848 ohms) were within normal ranges and relatively unchanged between both examinations. The activity sensor was turned "on" and the maximum sensor rate was set at 130 beats per minute (bpm). Reaction time was tuned to "fast" and recovery time to "medium." Stimulation was programmed in the unipolar mode and sensing in the bipolar mode. Battery voltage was 2.79 V with an estimated remaining life of more than 7 years.

The 64-row multidetector LightSpeed VCT XT scanner (General Electric, Fairfield, CT, USA) parameters were 600 mA, 120 kV, 0.35 s/tube rotation, starting at peak aortic enhancement after the beginning of a biphasic intravenous injection of 90 mL of iomeprol (Iomeron®, Bracco, Milan, Italy), 400-mg nonionic iodinated contrast, and 50 mL of saline both at 6 mL/s. The image acquisition time (x-ray tube "on") was 6 seconds.

The CTA was not evaluable because of the kinetic artifacts, and the patient underwent further workup with catheter coronary angiography within a month that failed to reveal any significant coronary artery disease, but during which similar transient episodes of tachycardia were observed.

Fig. 1 shows the range of the CTA between the two horizontal lines, and Fig. 2 the electrocardiogram (ECG) trace obtained during image acquisition.

Which of the pacemaker components is responsible for the ECG findings?

Comment

The baseline ECG displays a permanent AF and ventricular pacing at 60 bpm.

At the beginning of CTA, the heart rate (HR) rose from 60 bpm to a peak rate of 123 bpm in the span of 16 seconds. At all times, QRS complexes were preceded by a pacing spike.

The patient was hemodynamically stable throughout and after the CTA and the tachycardia rapidly subsided after the end of the examination.

Differential Diagnosis

Possible physical agents that may act on a pacemaker during a CT scan include the electromagnetic field of the rotating gantry tube and the high energy x-ray photons. Since similar ECG alterations were observed with a (nonrotating) fluoroscopic tube during catheter examination, the electromagnetic field interference is unlikely.

The fact that pacing spikes remained prior to each QRS complex suggests that the tachycardia arose from a pacemaker dysfunction. In such a case, the occurrence of a rare but potentially life-threatening runaway pacemaker is *a posteriori* excluded, since our patient's peak rate was below the set maximum pacing rate (123 vs 130 bpm), tachycardia self-subsided, and pacemaker control

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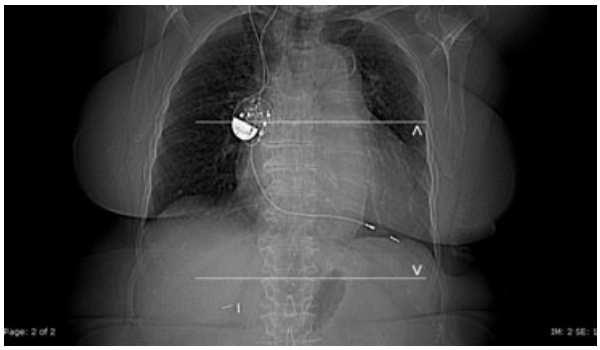

Figure 1. *The range of the CTA between the two horizontal lines.*

examinations showed a sufficient remaining battery life and no evidence of circuit damage.[1–2] Differential diagnoses for the component causing the x-ray-induced malfunction are discussed below and include:

- Lead alterations and induced currents.
- Damage to the complementary metal-oxide-semiconductor (CMOS) components of the pacemaker.
- Transient alteration in crystal oscillator function.
- Accelerometer sensor spurious signal.

Lead alteration is unlikely, as its integrity was demonstrated by normal impedance values. Moreover, various studies have shown that x-rays failed to interfere with pacemaker functions when only the lead(s) are in the scanning range.[3–5]

High energy x-rays photons entering a CMOS system may generate electron movement and positive charge "holes" in the circuit by a local photoelectric effect. Modern cardiac rhythm management devices use CMOS technology for construction of integrated circuits. Complementary circuits are more sensitive to lower levels of radiation than were discrete components. Two effects are most commonly studied. First, the total ionizing dose (TID) effect occurs when charge becomes trapped in the gate oxide of a transistor changing the characteristics of the transistor. It requires a high level of irradiation such as radiation therapy, for example,[3,6] and accounts for permanent, cumulative damages that were not found at pacemaker control. Second, the single event effect (SEE) is caused by a single energetic particle-generating electrons and holes in the silicon that drift to nodes in the circuit. Clinical translation of this effect in the pacemaker likely translate as pacing inhibition,[4] not tachycardia.

The crystal oscillator contained in the pacemaker pulse generator is a piezoelectric material acting as the timing circuit. Ionizing radiations may interfere with this device by direct interference with the crystal structures and shift in the vibration frequency, thus potentially changing the output of the device.[7] With recent technology crystals that are build resistant to ionizing radiations, occurrence of this interaction is however very unlikely.[8]

Therefore, the x-ray-induced tachycardias in this patient most likely originate from an accelerometer sensor spurious signal, mimicking an important activity increase. This translated as a progressive tachycardia, which peaked after the termination of x-ray output and gradually subsided without any intervention. Indeed, the rate-responsive sensor of the patient's pacemaker comprises a piezoelectric crystal and surrounding CMOS. The same argumentation as for the pulse generator piezoelectric crystal resistance to x-ray interference makes the dysfunction of this component of the accelerometer unlikely. However, occurrence of a SEE in the coupled CMOS would have been responsible for the particular kinetics of our patient's tachycardia (i.e., the rapidly but gradually accelerating heart rate) that are compatible with the rate-response "fast" reaction settings. This has been acknowledged by the manufacturer as the most likely cause of our pacemaker dysfunction.[9]

Discussion

A small but growing number of implantable cardiac rhythm management device (ICRMD) malfunctions during routine CT scan have been

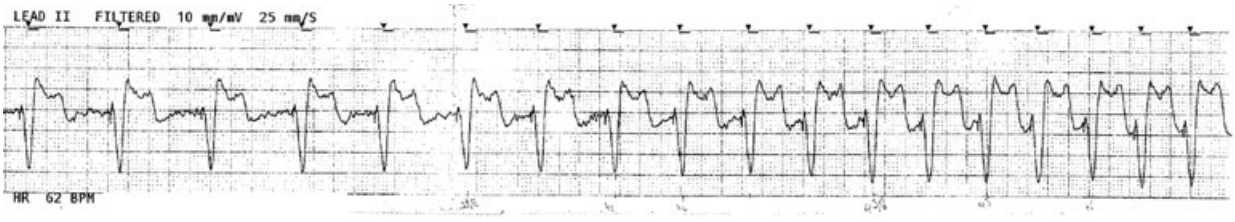

Figure 2. *The ECG trace obtained during image acquisition.*

reported in the last few years.[10] Although most of these dysfunctions have no clinical impact, some x-ray examinations sensitive to kinetic artifacts, such as CTA, could benefit from temporarily switching off the accelerometer of rate-responsive pacemaker-implanted patients.

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