Cardiac Tamponade Caused by Drug Interaction: A Delayed Complication of Apical Pacemaker Placement

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Cardiac tamponade is a rare but well-known complication of epicardial pacemaker placement, and delayed pericardial tamponade is extremely rare. Amiodarone-warfarin interaction, however, is a common occurrence that is not specifically related to pacemaker placement. This article discusses a case of a 75-year-old woman who develops cardiac tamponade secondary to an elevated international normalized ratio (INR) caused by an amiodarone-warfarin interaction.

CASE PRESENTATION

A 75-year-old woman is brought to the emergency department complaining of malaise, diaphoresis, and chest pain. Her present medications include amiodarone, warfarin sodium, and fluoxetine.

History

The patient’s medical history is significant for paroxysmal atrial fibrillation and sinus node dysfunction. She had a dual chamber permanent pacemaker installed 2 weeks prior to presentation. The patient's prothrombin time and INR had not been monitored. Information gathered from the patient's previous medical records reveals that amiodarone was initiated several days prior to pacemaker placement and warfarin was started after the pacemaker was placed. After an adjustment in her warfarin dose, the patient was discharged with a therapeutic INR of 2.5.

Physical Examination

On physical examination, the patient appears pale, diaphoretic, and in respiratory distress. The patient has a systolic blood pressure of 60 mm Hg; diastolic pressure is not measurable. The patient is febrile, with a heart rate of 110 bpm and a respiratory rate of 24 breaths/ min. Head, eyes, ears, nose, and throat examination demonstrates a raised jugular venous pressure. The patient has muffled heart sounds, diminished pulses, and pulsus paradoxus. Her lungs are clear and no evidence of peripheral edema is demonstrated.

Laboratory Studies

Laboratory studies reveal a leukocyte count of 11,300 mm$^3$, blood hemoglobin of 14.3 g/ dL, and hematocrit of 42.9%. The platelet count is 237,000 mm$^3$ with normal differential. Renal function is normal. Coagulation profile shows a prothrombin time of 48.7%, an INR greater than 8, and a partial thromboplastin time of 50.8 seconds. Fibrinogen is normal.

Electrocardiography demonstrates sinus tachycardia and old left bundle branch block with no new changes. Pacemaker interrogation is unremarkable. Echocardiography reveals cardiac tamponade with right atrial systolic inversion and right ventricular systolic collapse.

Treatment

The patient is given intravenous fluids and supplemental oxygen. Based on the results of echocardiography, the patient undergoes emergency thoracotomy, and 200 to 300 mL of blood is evacuated. A small laceration with no active bleeding is revealed in the right anterior ventricle. The laceration is thought to be secondary to wire perforation that occurred when the pacemaker was placed.

After the procedure, the patient is stabilized. Her recovery is uneventful. Warfarin administration is stopped, and the amiodarone dose is reduced.

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DISCUSSION

The incidence of myocardial perforation during placement of permanent epicardial pacemakers ranges from 3% to 9%.1–3 The majority of cases of perforation occur within 24 hours and, in many instances, patients are asymptomatic. Despite the relative frequency of myocardial perforation, cardiac tamponade occurs rarely, usually within 24 hours secondary to manipulative procedures. Delayed cardiac tamponade, as demonstrated in this case study occurring 2 weeks after pacemaker placement, is extremely rare.

Amiodarone and Warfarin

The well-known interaction between amiodarone and warfarin results in the prolongation of the prothrombin time and introduces a risk of bleeding;4–6 these effects have been attributed in part to interference with the hepatic degradation of warfarin.7 Amiodarone is often prescribed for the treatment of supraventricular, junctional, and ventricular tachycardia, as well as atrial fibrillation and tachycardia associated with Wolff-Parkinson-White syndrome. A high degree of efficacy and a lack of negative inotropism are advantages of amiodarone therapy. However, amiodarone use is complicated by the agent’s long half-life, side effects, and severe interactions with other drugs (eg, warfarin).

Also, amiodarone is extremely lipid soluble with a volume of distribution between 0.1 and 148l/kg, and therefore requires several months to 1 year to achieve a steady state (ie, the target drug concentration in the body in which the dosing rate equals the rate of elimination). This volume of distribution is responsible for gradually evolving drug interactions, such as the amiodarone-warfarin interaction. During established amiodarone-warfarin treatment, the dose of warfarin is reported between 16% and 45% of the typical allotment.6

SUMMARY

Amiodarone-warfarin interactions can have catastrophic consequences, as is clearly demonstrated by this case study. These physicians recommend that the INR be monitored twice as frequently at the beginning of amiodarone-warfarin therapy, and the frequency modified accordingly as the INR values become more stable. HP

REFERENCES