Paroxysmal supraventricular tachycardia of unusual etiology: A case report

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Paroxysmal supraventricular tachycardia (PSVT) is a serious cardiac disturbance that can arise spontaneously or be initiated by noncardiac mechanisms. An occurrence of this dysrhythmia in a 55-year-old male undergoing monitored anesthesia care for excision of a subxiphoid subcutaneous mass is reported. The unusual cause of this patient's ectopy is discussed. The nature and treatment of PSVT are reviewed, as are the clinical considerations for anesthetists who encounter a comparable clinical problem.

Key words: Anesthesia complications, cardiac arrhythmias, monitored anesthesia care, pacemakers, paroxysmal supraventricular tachycardia.

Introduction

A 55-year-old white male presented for elective removal of a subxiphoid subcutaneous mass using local anesthesia, intravenous (IV) sedation, and monitored anesthesia care (MAC). Surgery was performed on an outpatient basis in a major (600-bed) urban teaching hospital affiliated with the University of Connecticut and Yale University. The hospital averaged 14,000 surgical procedures annually.

At admission, significant findings included diet-controlled diabetes mellitus, chronic anemia, and hypertension. The patient's previous medical history was significant for excision of a pituitary adenoma in 1975 and coronary artery bypass grafting (CABG) in 1986, after which he was free of chest pain. He experienced rare intermittent episodes of atrial fibrillation. His medications included digoxin, aspirin, captopril, atenolol, and prednisone. Unfortunately, a digoxin level was not obtained prior to surgery. Preoperative testing documented a hematocrit of 38.2%, a hemoglobin level of 11.6 g/dL, and sodium and potassium blood levels of 145 mEq/L and 4.3 mEq/L, respectively.

Case summary

In the operating room, noninvasive blood pressure, precordial stethoscope, lead II electrocardiogram, and pulse oximeter monitoring were initiated. Baseline measurements were temperature, 99°F; 18 respirations per minute; pulse rate, 88/min; and initial blood pressure, 160/90 mmHg. The electrocardiogram, which was not of a type that provides ST segment analysis, displayed normal sinus rhythm. Oxygen was administered by nasal cannula at a rate of 3 L/min. Oximetry showed a 100% hemoglobin saturation both before and after starting the oxygen.

The patient received an IV dose of midazolam, 2 mg, and fentanyl, 100 μg, for sedation. The surgeon's initial difficulty in palpating the subxiphoid mass was overcome by having the patient raise his head enough to cause a tightening of his abdominal muscles. The mass could then be easily felt midway between the xiphoid process and an old mediastinal chest tube scar located under the right nipple line at the diaphragmatic margin.

The surgeon infiltrated 1% lidocaine with epinephrine 1:200,000 over the site of the mass. The dissection proceeded uneventfully, until a globular mass of fat and scar material was freed from the...
surrounding tissues. As the mass was removed, the patient experienced two short paroxysms of supraventricular tachycardia (SVT). Both episodes spontaneously resolved each time the surgeon stopped working on the mass. These episodes were not accompanied by discernible changes in blood pressure or level of consciousness.

As the surgeon freed and removed the mass, he discovered that two, 10-cm wires were embedded in and protruding from the tissue. The patient experienced no further ectopy after removal of the mass. The wound was closed, and the patient was transferred to the postanesthesia care unit in stable condition. There were no further episodes of PSVT nor changes in the ST segments of the electrocardiogram (ECG), as read in lead II. Since the patient failed to demonstrate objective evidence of myocardial damage and had no complaints in the immediate postoperative period, no isoenzyme or 12-lead ECG studies were obtained. He was later discharged without further incident.

Discussion

It appears that the wires were lead remnants of a temporary transthoracic epicardial pacemaker that was placed at the time the patient had his CABG. Since epicardial pacemaker leads are often sutured in place, it is not an unusual postoperative practice for surgeons to cut off excess wires at the skin line rather than risk damage to the myocardium or bleeding from excessive traction. Although this is a common practice, references to it are more often found in journal articles than in standard texts on cardiothoracic surgery.

Del Nido and Goldman, in an article dealing with complications of epicardial pacemakers, describe the technique. In it they explain how their residents are “instructed to apply only gentle traction for removal of the atrial and ventricular wires on the day prior to discharge, especially since nearly all patients are on either anticoagulants or antiplatelet therapy; the patients are then instructed to stay in bed and are observed closely for the hour following removal. If there is undue resistance, the wires are cleansed with alcohol, cut short, and allowed to retract.”[Emphasis added.]

It is interesting that the authors note: “Considering the meticulous attention to detail that is inherent to open heart procedures, it is distressing how casual and often inappropriate is the deployment of temporary epicardial pacing wires at the completion of such surgery.”[1] Allowing the retained wires to retract subcutaneously is considered a normal and prudent course of action when on attempted extraction significant pulling traction is encountered.

Another article, which deals with transvenous rather than epicardial pacemakers, emphasizes the problems inherent in forcibly removing pacemaker wires once the need for pacing has ended. The authors note: “Continuous forceful traction can produce chest pain, bradycardia, ventricular tachycardia and fibrillation, invagination of the right ventricular wall or of the tricuspid valve, hemopericardium, and cardiac tamponade.”[2] The same article also recommends that uninfected pacemaker leads be left in the subcutaneous tissue if more than “gentle traction” is required for their removal. Long-term experience has documented the safety of this approach.[2] This case appears to be an exception.

Evidently, in the intervening 7 years since his CABG surgery, the patient's remnant pacemaker leads served as a base for the tissue thickening and scarring that became the subxiphoid mass. As the surgeon manipulated the mass, the remaining implanted epicardial ends of the wires generated enough activity to disrupt myocardial conduction. This seems to have initiated the two episodes of SVT, which spontaneously resolved when the manipulation stopped.

It can only be speculated as to why the patient did not experience more arrhythmias or myocardial tearing and bleeding as the remaining wires were freed. Perhaps enough scarring or hypertrophy of surrounding tissues had developed to toughen the myocardium at the wire insertion points so that such problems did not occur. Another possibility is that the wires were more easily manipulated due to their degeneration with age and merely “snapped off” at the myocardial margin when they were pulled. The patient suffered no significant sequelae from the unplanned removal of the leads.

Causes and treatment of PSVT

Most cardiac tachyarrhythmias (ventricular response > 100 beats per minute) arise in the supraventricular area above the level of the atrioventricular (AV) node.[3] They include atrial flutter and fibrillation, multifocal atrial tachycardia, and PSVT. Ventricular tachyarrhythmias typically are diagnosed by electrocardiographic changes, including a QRS complex width greater than 140 ms, left axis deviation, AV dissociation, and the presence of capture/fusion beats.[4]

Although some SVTs are considered to be of little clinical consequence, two classes of supraventricular arrhythmias can require emergency treatment. One is atrial flutter or fibrillation, with a corresponding rapid ventricular response rate that compromises hemodynamics. Although this pa-
tient had a history of intermittent bouts of atrial flutter/fibrillation, they were not accompanied by altered hemodynamics. The other is PSVT, which is accompanied by severe hypotension that compromises hemodynamics. When this occurs during anesthesia, a decision must be made whether the disturbance is immediately life threatening. The disturbance can either be a precursor to a life-threatening arrhythmia or indicative of acute myocardial ischemia. Therefore, whenever a patient exhibits both PSVT and hypotension, the anesthetist should suspect underlying coronary artery disease (CAD) or some other form of acute ischemia.

Tachydysrhythmias are especially dangerous in the presence of CAD, and the anesthetist must take appropriate measures to restore normal rhythm and support the circulation. Algorithms for recognizing and treating tachydysrhythmias have been incorporated in the American Heart Association (AHA) standards for advanced cardiac life support (ACLS).

Paroxysmal supraventricular tachycardia can be the result of reentry phenomena. Other causes include enhanced automaticity of normal or ectopic pacemaker cells. In PSVT caused by reentry, the normal activity of the heart is disrupted so that signals leave and then reenter the conducting system, producing excessive increases in heart rate than can compromise hemodynamics.

In this case, SVT was not accompanied by hypotension, and oxygen saturation remained high. Obviously, this lessened but did not fully eliminate the likelihood that the patient suffered myocardial ischemia. Given the lack of evidence to the contrary, it would appear that myocardial function was maintained, that the patient remained well oxygenated, and that these conditions facilitated spontaneous remission of the ectopy. It is probable that the PSVT episodes were generated by reentry phenomena. Manipulation of the remaining embedded pacemaker wires could have stimulated the ectopic pacemaker cells to fire and thus initiated reentry.

By definition, PSVT can start and stop quickly and unpredictably, just as it did in this case. It may also resemble or be associated with Wolff-Parkinson-White (WPW) syndrome, which is the most common of the preexcitation syndromes and is found in approximately 0.3% of the population. Transmission of cardiac impulses down the Kent fibers (an alternative atrial pathway) causes early activation of the ventricle. Wolff-Parkinson-White syndrome is seldom associated with paroxysmal atrial tachycardia. Treatment of WPW includes vagal stimulation followed by various IV medications, such as diltiazem, propranolol, and procainamide. If life-threatening hypotension is present along with atrial fibrillation, cardioversion is indicated.

Some authors, such as Stoelting, recommended verapamil as the drug of choice for treating PSVT. However, verapamil should not be used to treat PSVT in WPW syndrome associated with atrial flutter or fibrillation, because the drug may induce excessive ventricular response rates that lead to pronounced hemodynamic deterioration. In such a setting, cardioversion is the preferred treatment.

If the patient is hemodynamically stable and the PSVT persists, the initial treatment consists of maneuvers to stimulate the vagus nerve, including carotid sinus massage and repeated Valsalva's maneuvers. Pneumatic antishock trousers have also been successfully used, although they are not recommended in current AHA ACLS guidelines.

Under current AHA ACLS guidelines, adenosine is the recommended initial therapy for PSVT. It acts on the AV node to slow conduction and inhibit reentry pathways. If, after adenosine is administered, the ECG QRS complex remains narrow and the blood pressure is stable, verapamil can be administered. If verapamil is unavailable, edrophonium can be substituted, although its use is not presently included in the AHA ACLS guidelines.

After verapamil has been administered, it is recommended that digoxin, beta-blockers, and diltiazem be considered. If the patient's QRS complex is wide and the blood pressure is stable, lidocaine can be administered in an attempt to stabilize impulse transmission within the heart's conducting system. Procainamide can be administered as a follow-up drug if the lidocaine does not fully stabilize conduction. In this case, spontaneous remission of the PSVT obviated the need for such measures.

If the patient remains hemodynamically unstable after adenosine, the recommended treatment, regardless of the width of the QRS complex, consists of synchronized cardioversion with 100 J of power, proceeding in 100-J increments to a maximum of 360 J.

Clinical considerations

Three considerations regarding this case are clinically important. First, the problems encountered with this patient might have been avoided by a preoperative chest x-ray. The wire leads embedded in the mass would have been clearly revealed by radiography. However, because preanesthetic chest x-rays have been shown to produce only a "low yield" of positive previously unknown find-
ings in surgical patients, they are not considered to be cost-effective for routine use.  

As a result, many hospitals have abandoned routine chest x-rays as a screening measure. This is especially likely to occur with patients who have a negative pulmonary history, those who are asymptomatic, and those who are considered to be healthy and are undergoing relatively short, uncomplicated surgical procedures on an outpatient basis.

A similar policy is in effect at the hospital in which this surgery was performed, so the patient had no preoperative screening x-ray. A preanesthetic chest x-ray in this patient might have alerted the surgeon and the anesthetist that pacemaker lead wires remained embedded in cardiac tissue.

A second danger to be considered in a case like this is that posed by electricity. Had the surgeon used an electrocautery unit to coagulate bleeding vessels at the operative site or had there been a malfunction of the operating room's other electrical equipment, the patient could have been subjected to serious injury, even death. Electrocautery or malfunctioning electronic equipment can generate high levels of electrical current. If that current passes through the patient's entire body, it results in macroshock, which can cause burns or other injury. Potentially more lethal is the danger posed by microshock, where an electrical current that is generated at or near the heart passes through it with a density great enough to completely disrupt its normal conduction system. Under such conditions as few as 20 μA have been known to induce ventricular fibrillation and death.

In this patient, cautery contact at one end of the residual pacemaker wires would have conducted such a density of current directly to the other end of the wires, which remained embedded in heart muscle. Such an occurrence in this case would almost certainly have had a catastrophic end. Since the surgeon did not use electrocautery, an electrical injury was avoided. Had the presence of the wires been known before surgery, avoidance of the use of a cautery would have been a deliberate and prudent decision.

This case reemphasizes the adage that no case is "routine." Even the healthiest patient who undergoes the simplest surgery under monitored anesthesia care and local anesthesia is never totally immune from danger.

Summary

Paroxysmal supraventricular tachycardia can arise from either cardiac or noncardiac causes. In this case, two episodes of PSVT occurred in a 55-year-old male who was undergoing minor surgery for excision of an abdominal mass. These episodes appear to have been caused by reentry phenomena associated with the surgical manipulation of two residual pacemaker wires from CABG surgery 7 years earlier.

Both the surgeon and anesthetist were unaware of the presence of the wires. Fortunately no electrocautery devices were used that could have placed the patient in danger. The two PSVT episodes spontaneously resolved, and the patient's surgery was completed without further difficulties.

REFERENCES


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