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Adenosine for the treatment of sustained sinus nodal reentrant tachycardia during general anesthesia

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Introduction

Sinus tachycardia is common in the perioperative period, and treatment is often dictated by the cause of the increased heart rate [1]. Intraoperative sinus tachycardia can be associated with pain, sepsis, hypo- or hypervolemia, light anesthesia, transfusion reactions, pediatric age group, preexisting sinus node disease, or electrolyte abnormalities [2,3]. Most of these etiologies are benign in nature or are readily amenable to treatment. It is infrequent to see sinus tachycardia triggered by central venous catheter placement, and even more rare to see it persist after the cause is removed [4,5].

We present a case of sudden persistent sinus tachycardia induced by central vascular access catheter (Broviac catheter, C.R. Bard Inc., Salt Lake City, USA) placement. A discussion of the treatment of nodal reentry tachycardias follows.

Case report

A 62-year-old man with a history of colon carcinoma that metastasized to the cervicothoracic vertebrae underwent a cervicothoracic laminectomy after which complete wound dehiscence and esophagocutaneous fistula occurred secondary to infection. He presented for cervical restabilization, resection of the esophagocutaneous fistula, and central vascular access catheter placement for chemotherapy. One month prior to this procedure, the patient had undergone cervicothoracic hardware removal and wound debridement under an uneventful general anesthetic, and his infection had been treated with ceftazadine and vancomycin. He had no history of cardiopulmonary disease. Pre-operative EKG showed a normal sinus rhythm (Fig. 1a). The patient had a small oral opening and had required fiberoptic intubations previously. Glycopyrrolate 0.2 mg, i.m. was administered as premedication. He was brought to the operating room where standard monitors were placed. A 1-mg defasciculation dose of pancuronium was given and general anesthesia was induced with fentanyl 250 μg and thiopental 375 mg. After 100 mg of succinylcholine were administered, an oral flexible fiberoptic intubation proceeded uneventfully. Anesthesia was maintained with 60% NO_2, 40% O_2, and 0.4% enflurane. Muscle relaxation was maintained with continuous pancuronium administration. Over the next 8 h, the patient received 1500 μg of fentanyl. His blood pressure was approximately 110/60 to 140/80 mmHg with a heart rate of 70–80 beats/min. Sinus rhythm was maintained, and the femoral central venous pressure ranged from 8 to 11 mmHg. He tolerated the cervicothoracic and esophageal part of the procedure well, but as the vascular access catheter was inserted into the innominate vein under direct vision, the ECG showed occasional premature ventricular contraction (PVCs) followed by several seconds of ventricular tachycardia. Lidocaine 100 mg i.v. was given, and the catheter was withdrawn. At this point, the patient's rhythm changed to tachycardia at a rate of 160 beats/min with normal P wave morphology and narrow QRS complexes (Fig. 1b). Even after the catheter was completely withdrawn, tachycardia persisted. Blood pressure remained constant and central venous pressure (CVP) stayed at 11 mmHg. Over the next 20 min, the possible etiology of this apparent sinus tachycardia was sought as treatments were instituted. The patient was given fentanyl 750 μg to increase the depth of anesthesia and provide

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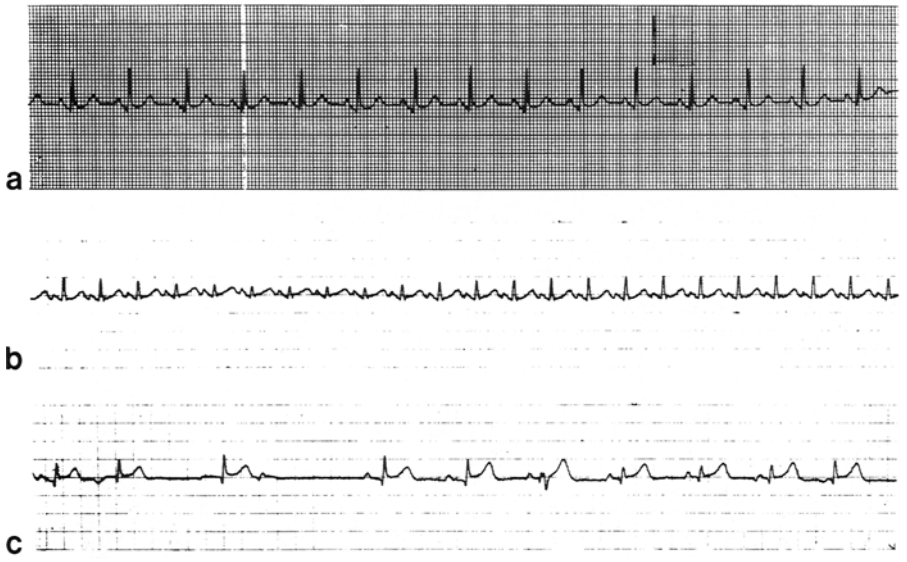

Fig. 1a–c. The *upper strip* (**a**) represents the patient's normal sinus rhythm as seen in lead 2 of preoperative ECG. The *middle strip* (**b**) represents the intraoperative tachycardia seen in lead 2. The *lower strip* (**c**) demonstrates the conversion of tachycardia to sinus rhythm in lead 2. The *lower strip* also shows usual changes in rhythm as adenosine is administered. At the time of conversion to sinus rhythm, a variety of new rhythms are known to appear on the electrocardiogram. The *lower strip* shows few P wave inversions before and after the sinus bradycardia. The patient remained in sinus rhythm afterwards

increased vagotonia. Additional volume was administered which increased the CVP to 15 mmHg. Arterial blood gas analysis showed a pH of 7.44, $Paco_2$ of 38 mmHg, and Pao_2 of 370 mmHg. He was normothermic and electrolyte evaluation was within normal limits. The hematocrit was 33%. No recent transfusion had been given, and he had remained asymptomatic after the completion of earlier transfusions. At this point, carotid massage was performed without success. Three separate boluses of phyenlephrine 40 μg raised the blood pressure but did not slow the heart rate. Three 20-mg, two 40-mg, and one 60-mg bolus doses of esmolol were given over a 10-min period which decreased blood pressure slightly but did not slow the heart rate.

When traditional sinus tachycardia therapy was not successful and other etiologies of sinus tachycardia could not be found, it was suspected that this was an atypical form of paroxysmal supraventricular tachycardia, and adenosine therapy was instituted through the femoral CVP catheter. Initially 6 mg did not change the heart rate; 30 s later, a second 6-mg dose slowed the rate to 75 beats/min after an initial bradycardia and other variety of new rhythms (Fig. 1c). The rhythm reverted to tachycardia within 15 s. An additional bolus dose of adenosine 6 mg slowed the rate to 75 beats/min where it remained in sinus rhythm. These rate changes were abrupt and were not associated with hemodynamic changes. Because the patient required postoperative chemotherapy and parenteral nutrition, a central vascular catheter was placed again, taking care to locate it above the right atrium. Its position was checked by contrast dye using fluoroscopy. The operation was completed 30 min later without further rhythm changes, and tachycardia did not return postoperatively. The patient underwent subsequent operations requiring general anesthesia at a later date without reappearance of tachycardia.

Discussion

Sinus tachycardia is the most commonly occurring arrhythmia in the perioperative period. Treating its etiology generally eliminates the problems [3]. Sustained sinus tachycardia at a rate of 150 beats/min for more than 30 min can cause decreased diastolic filling and increase myocardial oxygen demand, both of which may lead to ischemia [1,3].

The perpetuation of sinus tachycardia was unusual in this patient. He clearly received adequate amounts of anesthesia (fentanyl 1500 μg over 8 h and a 750 μg bolus) as determined by normotension and the absence of sympathetic responses. Analysis of temperature, blood gases, and intravascular volume were all within normal ranges. No significant bleeding occurred at this point as documented by a hematocrit of 33%. No signs of pre-existing sinus node disease or endocrine problems were found in his medical history or with previous anesthetics. Although pancuronium's vagolytic effects can predispose a patient to tachyarrhythmias, the dose prior to this event was 3 mg/h given by continuous infusion. Therefore, vagolysis from pancuronium would not be an expected etiology of the tachycardia. Drug error was unlikely, and no drug was administered other than lidocaine at the time of the arrhythmia's initiation. Tachycardia was initiated by placement of the central venous access catheter but did not resolve with complete withdrawal of the catheter from the central vein.

Catheter-induced arrhythmias have been reported to be as high as 48% during right-sided heart catheterizations but rhythms were mostly confined to

premature ventricular contractions and ventricular tachycardia [4]. They are usually self-limited but may require therapeutic intervention. Occasionally premature atrial contractions have been noted, but they usually disappear spontaneously following withdrawal of the catheter or treatment with lidocaine [4,5]. There are no reported cases of persistent sinus tachycardia caused by catheter placement. We excluded hypotension, hypovolemia, hypoxia, hypercarbia, vagolysis, fever, transfusion reaction, light anesthesia, myocardial infarction, and pulmonary embolism as etiologies of this patient's arrhythmia and deduced that this was an unusual presentation of paroxysmal supraventricular tachycardia.

Paroxysmal supraventricular tachycardia is a group of arrhythmias with unusual aberrant conduction pathways. There are seven different forms of paroxysmal supraventricular tachycardia. Of these, two are most common. They are atrioventricular (AV) nodal reentrant tachycardia, and orthodromic AV reentrant tachycardia. Two other types, intraatrial tachycardia and sinus node reentrant tachycardia are rare but more closely resemble our rhythm [6]. Descriptions of these four types follow.

First, AV nodal reentrant tachycardia is the most common form of paroxysmal supraventricular tachycardia (PSVT). Two types exist of which the slow-fast sequence is the most common. In this form, electrical impulses are conducted from atrium to ventricle via a slow pathway within the AV node and returned to atrium via a fast pathway. It is characterized by narrow QRS complexes and inverted P waves which are usually not identifiable.

Second, orthodromic AV reentrant tachycardia involves an accessory pathway. Impulses depolarize the ventricle in the usual fashion, return to atria via accessory bypass pathways, and then go back to the AV node causing tachycardia. It is characterized by narrow QRS complexes and inverted P waves which follow the QRS complex.

Third, intraatrial tachycardia might be caused by reentrance somewhere in the supraventricular conduction system [7,8]. It is characterized by narrow QRS complexes and varying P wave morphology. The P waves preceed each QRS complex.

Finally, sinus node reentrant tachycardia is differentiated from sinus tachycardia by its abrupt onset and termination. It has narrow QRS complexes, a rate between 100 and 160 beats/min, and P waves which precede the QRS complex and demonstrate the same morphology as in sinus rhythm [8]. Of all the different PSVTs, our case most closely resembles sinus node reentrant tachycardia (Fig. 1b). Sinus node reentry tachycardia is an extremely rare form of PSVT that accounts for less than 10% of PSVTs in electrophysiologic studies and is seen mostly in electrophysiologic lab settings [9]. There is very little information regarding this rhythm and its treatment [10,11]. Sinus node reentry tachycardia appears to be associated with organic heart disease. Atrial stretch and fibrosis may be factors predisposing to inhomogeneous conduction leading to a small-dimension circus movement within perisinus node tissue [8,11]. Rabbit studies have demonstrated that early impulses may stimulate only part of the SA node. The remaining original impulse may slowly restimulate another part of the SA node and reexcite the atrium, leading to sinus node reentry tachycardia [12].

Esmolol's ultra-short-acting half-life of 2 min and beta selectivity makes it an ideal choice to treat tachycardia [1,2]. In this case, we had administered high doses of esmolol without effect. Although verapamil's success rate with PSVTs is impressive, it was not administered due to a high associated incidence of hypotension and heart failure from vasodilation and decreased contractility [13]. More importantly, verapamil is associated with marked sinus bradycardia and sinus arrest in combination with beta blockers which we had already administered [13]. Vagotonic maneuvers such as administration of fentanyl, phenyleprhrine [3], and carotid massage [3,14] were not helpful. Edrophonium was not administered because of its association with cardiac arrest [15].

Although adenosine had never been reported for the treatment of intraoperative sinus nodal reentrant tachycardia, it was administered to this patient. Adenosine, a naturally occurring nucleoside, has long been known to reduce or block sinoatrial (SA) and atrioventricular (AV) node conduction and automaticity in humans [16]. Adenosine's clinical utility had been focused on treatment of recurrent PSVT involving the AV node; 90% of patients with recurrent PSVT involving the AV node as part of the reentry loop were successfully converted [17].

PSVT is not only associated with reentry tachycardia involving the AV node, but also sinus node reentry and intraatrial reentry tachycardia which are responsible for approximately 10% of PSVTs [7,9]. This is where adenosine's effect on the SA node can be beneficial. Until now, adenosine had never been shown to be effective in converting PSVTs without AV node involvement.

In laboratory settings involving humans and animals, adenosine depresses SA node automaticity and conduction [17]. When given intravenously in animals, it increases the sinus cycle length by 50% within 20 s after administration. Previous clinical treatment studies of PSVT have shown adenosine to be effective at the AV node only [18]. Here, we present the first clinical case where the sinus node-slowing effect of adenosine is

responsible for conversion of sinus node reentrant tachycardia to sinus rhythm.

Adenosine is an endogenous purine nucleoside that is normally found in large amounts throughout all human tissues. Investigators infusing adenosine into humans have not been able to demonstrate increases in circulatory adenosine, thus making pharmacokinetic studies impossible to perform [19]. Adenosine is an essential component of energy production. The quantity administered is small compared to existing amounts of adenosine in the blood. Its half-life is less than 1 s and it has a plasma clearance of less than 30 s. Its short half-life is due to the rapid metabolism by blood elements and vascular endothelium with rapid cellular uptake [9,18]. Adenosine appears to cause depression of the SA and AV nodes by increasing potassium conductance mediated through depression of cyclic adenosine monophosphate [6]. The main effect appears to be prolongation of the nodal-to-His bundle interval, resulting in slowing or blocking of AV node conduction. This explains adenosine's main effect in the treatment of PSVT associated with reentry tachycardia at the AV node [17].

Intravenous adenosine will convert the most common PSVTs to normal sinus rhythm. A variety of new rhythms such as atrial premature contractions, sinus bradycardia, sinus pause, and ventricular and junctional escape beats are seen at the time of conversion to normal sinus rhythm. Such findings were seen in many patients and last only a few seconds [19]. Transient hypotension can be seen also. Adenosine does not lead to tachyphylaxis or rebound hypertension because it does not lead to activation of the renin-angiotensin system [20]. It is also reported to be safe when administered to patients with congestive heart failure and patients on beta blockers [16].

The most commonly reported side effects of adenosine are facial flushing, chest discomfort, and dyspnea, which are short-lived and easily tolerated [18]. Interestingly, more adverse effects are associated with lower doses than higher doses. If overdose occurs, its short half-life elimination will help prevent complications. Theophylline and other methylxanthines block adenosine receptors and thus inhibit its effect. Conversely, its effect is potentiated by dipyridamole, which inhibits its uptake [6].

We describe the first reported case of intraoperative sinus node reentrant tachycardia which was successfully treated with adenosine.

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