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Intraoperative sudden arrhythmias in cervical spine surgery adjacent to the stellate ganglion: A case report

Stellate ganglion stimulation causes sudden arrhythmia

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Abstract

BACKGROUND

Atrial arrhythmias such as paroxysmal supraventricular tachycardia (PSVT) and atrial flutter (AF) are relatively common in the perioperative setting. They commonly resolve spontaneously. However, occasionally, they may continually progress to fatal arrhythmias or cause complications. Therefore, prompt and appropriate management is important.

CASE SUMMARY

A 46-year-old female patient diagnosed with cervical C6–7 radiculopathy characterized by decreased sensation in the right 3, 4, and 5 fingers underwent C6–7 anterior cervical disc fusion surgery. During surgery, electrocardiogram showed PSVT and ventricular tachycardia during C6–7 disc retraction. However, the patient remained stable. Initial treatment with esmolol and lidocaine for ventricular tachycardia was ineffective. Carotid massage and Valsalva maneuver were attempted. Nevertheless, PSVT did not resolve. The surgery was paused, and the patient's fraction of inspired oxygen was set to 100%. Next, adenosine was administered for pharmacologic PSVT management. The

arrhythmia temporarily resolved. However, it then transformed into AF. Diltiazem was administered, thereby briefly decreasing blood pressure, which immediately recovered. Surgery resumed while the patient was in normal sinus rhythm. She was discharged safely on the 6th postoperative day without complications and abnormalities. Currently, she is living a healthy life without arrhythmia recurrence.

CONCLUSION

Ganglia associated with cardiac arrhythmias in the surgical site should be identified during cervical spine surgery. Differentiating the characteristics of PSVT from those of AF on electrocardiogram is important to provide an appropriate management. If arrhythmia persists despite initial treatment, overlapping arrhythmias must be suspected.

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Core Tip: PSVT and AF can occur without structural heart disease and are present at any age. In the current case, the arrhythmia was caused by the surgical stimulation of the stellate ganglion in a patient without a significant medical history. Moreover, the electrocardiogram results were similar, thereby making it difficult to identify the type of arrhythmia. Hence, another arrhythmia was observed even after appropriate treatment. In such a case, if an arrhythmia occurs in the context of stimulation of the right stellate ganglion during cervical spine surgery, identification of triggers to consider, correction of the appropriate triggers, and prevention of migration to fatal arrhythmias should be considered.

INTRODUCTION

The incidence of intraoperative arrhythmias is quite high, and some arrhythmias require clinical attention. [1]. Paroxysmal supraventricular tachycardia (PSVT) is a common arrhythmia that can occur at any age in patients without structural heart disease. atrial flutter (AF) is a type of heart rhythm disorder caused by issues in the heart's electrical system. That is, the upper chambers of the heart (atria) beat extremely rapidly but with a regular rhythm. AF is similar to atrial fibrillation, a common heart disorder. [2]

The risk factors of intraoperative arrhythmia include various patient factors, such as age, sex, comorbidities and medications, and surgical factors, such as the type and duration of surgery and the use of anesthetics. In flutter and PSVT, measures to lower blood pressure and the use of medications or other procedures may be required to restore normal heart rhythm. [1]. In cervical spine surgery, particularly cervical C6–7 Level disc surgery, the proximity of the surgical site to the phrenic nerve and stellate ganglion and the potential for nerve irritation may increase the risk of arrhythmia.

This case shows the importance of assessing potentially arrhythmogenic anatomy at the level of the C6–7 spine while recognizing arrhythmia risk factors in patients undergoing cervical spine surgery, and the need to appropriately differentiate and manage potentially life-threatening arrhythmias such as atrial and ventricular arrhythmias.

CASE PRESENTATION

Chief complaints

A 46-year-old female patient was diagnosed with C6–7 radiculopathy characterized by decreased sensation in the right 3, 4, and 5 fingers.

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History of present illness

Nonspecific findings were found on preoperative examination and electrocardiogram (ECG) (Figure 1A).

History of past illness

The patient's medical history was unremarkable.

Personal and family history

She had no family or genetic history of the disease.

Physical examination

Upon departure from the operating room, the patient's vital signs were within normal limits: BP, 110/80 mmHg; HR, 62 beats/min; SpO$_2$, 99%; and temperature, 36.3°C. ECG revealed a normal sinus rhythm (Figure 3A). After anesthesia induction, the patient's vital signs were as follows: BP, 128/71 mmHg; HR, 65 beats/min; and SpO2, 99%. Arrhythmias associated with nonspecific findings were observed on neurological examination.

Laboratory examinations

On perioperative laboratory examinations, the cardiac marker, CK-MB, Procalcitonin, and Tropinon-I levels were within the normal range.

Imaging examinations

The radiologic finding was nonspecific without the C6–7 foraminal stenosis with disc bulging on magnetic resonance imaging (Figure 2A).

FINAL DIAGNOSIS

The patient was diagnosed with AF or PSVT r/o VT. However, the final diagnosis was AF.

TREATMENT

The electrode pads were immediately applied in preparation for DC cardioversion. If the patient was hemodynamically stable, esmolol 10 mg and lidocaine 60 mg were administered for ventricular tachycardia based on the ECG results. However, they were ineffective. PSVT or AF was suspected. Thus, carotid massage and Valsalva maneuver were attempted to relieve r/o PSVT. However, arrhythmia persisted. According to the surgeon's discretion, the surgery was discontinued temporarily. The fraction of inspired oxygen was set at 100%, and adenosine 6 mg was administered for the pharmacological management of PSVT, with 200 mL of crystalloid to maintain hemodynamic stability. There was a transient decrease in BP (from 78/46–67/35 mmHg). Nevertheless, it normalized within 2 min. The ECG findings subsequently changed and appeared to show a temporary disappearance of the arrhythmia, and then changed to AF with an atrial rate of 287 beats/min, with only the p-wave again being prominent (Figure 3D). Diltiazem 10 mg was then added gradually. The flutter persisted. However, cardioversion was not performed as the patient was hemodynamically stable. There was a transient decrease in BP. Nevertheless, the ECG began to normalize. We requested the surgeon to continue the surgery. During the operation, two episodes of premature venous complex were observed. Nonetheless, the arrhythmia disappeared with treatment with lidocaine 60 mg. To prevent arrhythmia recurrence, lidocaine 60 mg was further administered. The BP then increased to 109/66 mmHg 3 min after the administration of diltiazem. The ECG showed a normal rhythm (Figure 3E).

OUTCOME AND FOLLOW-UP

At the end of the surgery, the HR changed from 50 to 58 beats/min and the BP from 105/62 to 126/76 mmHg, and the sPO2 was maintained at 100%. The NMT confirmed a neuromuscular blockade level of 0, sugammadex 200 mg was administered. The patient was fully alert and had spontaneous breathing. Hence, she was extubated. Just before leaving the operating room, the patient's vital signs were as follows: HR, 71 beats/min; BP, 135/73 mmHg; and the SpO2, 100%. The total anesthesia time was 140 min. Upon arrival in the recovery room, the patient's vital signs were as follows: HR, 78 beats/min;

the BP, 133/82 mmHg; and the SpO2, 100%. The patient did not complain of any symptoms other than pain at the surgical site. No arrhythmias such as AF were detected on ECG performed immediately after arrival in the recovery room (Figure 1B).

After discharge from the recovery room, various tests were conducted for further evaluation. The levels of cardiac markers, including creatine kinase-MB (1.72 ng/mL), N-terminal pro B-type natriuretic peptide (38.8 pg/mL), procalcitonin (0.01 ng/mL), and troponin-I (<0.001ng/mL), were within normal limits. Further, the ECG Holter monitoring result was normal. Echocardiography showed mild tricuspid regurgitation (peak velocity at 2.29 m/s) without pulmonary arterial hypertension (30 mmHg), which was believed to be within normal limits. Subsequent ECGs (Figure 1C) revealed no abnormalities, and both room BP and HR during hospitalization were within normal range. The patient was discharged on postoperative day 6. Currently, she is living a healthy life without arrhythmia recurrence.

DISCUSSION

Atrial fibrillation is the most common atrial arrhythmia that can occur during surgery, and AF and supraventricular tachycardia are also frequently observed, particularly among arrhythmias with a constant QRS rhythm. PSVT is a typical arrhythmia that can develop at any age in patients without structural heart disease, most commonly between the ages of 12 and 30 years. Its incidence is 2.5 per 1000 adults, and it is more common in women. PSVT can be caused by atrioventricular nodal reentrant tachycardia or atrioventricular reentrant tachycardia. AF is a type of heart rhythm disorder caused by issues in the heart's electrical system. That is, the heart's upper chambers (atria) beat extremely rapidly but with a regular rhythm. AF is similar to atrial fibrillation, a common disorder. [2] The initial arrhythmia is similar to a wide QRS tachycardia, and several conditions should be distinguished. First, it is important to differentiate true wide QRS tachycardia from tachycardias such as PSVT, which can occasionally mimic a wide QRS. In particular, ventricular tachycardia can be life-threatening and can require proper diagnosis and treatment.

The specific arrhythmias experienced by a patient during surgery are based on various factors and should be managed immediately. Anti-arrhythmic medications can be used to manage QRS tachycardia only if the tachycardia is regular and the patient is hemodynamically stable, as in low-risk patients with stable ECG and hemodynamic values before and after the arrhythmia, those with normal caffeine intake, and those without a history of suspicious medical conditions, excessive exercise, alcohol abuse, and smoking. [3] However, misdiagnosing a ventricular tachycardia for supraventricular tachycardia can pose a serious risk. Thus, the condition should be treated as ventricular tachycardia when it cannot be appropriately identified. In this case, wide QRS tachycardia was inhibited after the administration of adenosine, and the p-wave was tapped more than the QRS on ECG, thereby indicating AF. [4] The intentional atrioventricular block is extremely useful in differentiating narrow QRS tachycardias, and tachycardia termination is achieved with the use of Valsalva or adenosine. In this case, the tachycardia was inhibited. However, only the p-wave morphology changed to that of AF, which was observed evidently. New-onset AF during general anesthesia induction for noncardiac surgery in a patient without a history of cardiac arrhythmias is rare, and similar cases are challenging to identify. [5] AF is caused by a macro-reentry between the interatrial septum and the free wall of the right atrium, including the isthmus between the tricuspid annulus and the inferior vena cava. [6] The atrial rate is >240 beats/min. The atrioventricular node has an atrioventricular conduction pattern that varies from 1:1–8:1. [7] In this case, the atrioventricular conduction varied from 1:1–7:1. The ventricular rhythm was relatively constant. Nevertheless, the atrioventricular rate differed, probably due to the effects of adenosine, which limits AV conduction. This atrial rhythm was probably attributed to the interaction of the drug with any issues in atrial autonomic control. While the exact cause of atrial tachyarrhythmias is often unknown, several factors have been associated with its development. Age more than 60 years, structural heart diseases such as heart valve disorders, history of heart surgery or congenital heart defects, coronary heart disease. Hypertension can lead to changes in the structure and function of the heart.

Thyroid disorders such as hyper or hypothyroidism can disrupt the normal electrical activity of the heart. Sympathetic stimulation, use of stimulants such as nasal decongestants, hypovolemia or anemia, hypoxemia, hypercarbia, fever, sepsis, and malignant hyperthermia also can lead to it. In addition to these factors, there are possible aggravating factors specific to this tachyarrhythmia, which include direct surgical damage to autonomic nerve fibers innervating the atria and sinoatrial node, high perioperative heart rate due to sympathetic nervous system excitation, inflammatory changes in the myocardium with age, and premature atrial contraction. [8] In this case, tachycardia was not observed before surgery, and there was no evidence of premature atrial contraction. A preoperative echocardiogram was not performed because the patient had no history of cardiac disease. However, a trivial tricuspid regurgitation was observed on the postoperative echocardiogram. There are some speculations as to the effect of these findings on the development of arrhythmia. The other actions of the autonomic nervous system have also been implicated in AF development, and AF has been reported in a patient without cardiovascular risk factors for arrhythmia and with a normal preoperative ECG who developed AF due to increased abdominal pressure during laparoscopic gastrectomy. [5] This indicates that even in the absence of an underlying medical condition, AF can be caused by autonomic nervous system activity.

 The stellate ganglion is the fusion of the inferior cervical sympathetic ganglion, comprising the sixth and seventh cervical ganglia, and the first thoracic sympathetic ganglion in a space bounded anteriorly by the subclavian and the vertebral artery and posteriorly by the neck of the first rib and the seventh cervical transverse process. [9] The stellate ganglion is the efferent component of the sympathetic nervous system, and the postcardiac ganglionic sympathetic nerves located in the stellate ganglion are distributed throughout the heart, releasing neurotransmitters (e.g., norepinephrine [NE]) bind with beta-adrenergic receptors to affect cardiac electrophysiological and contractile functions, including heart rate, cardiac conduction, and myocardial contraction. It is widely recognized that cardiac postganglionic sympathetic nerve terminals innervate the sinoatrial node, atrioventricular node, His bundle, and contractile myocardium.

Although the number of neurotransmitters, including NE, is primarily determined by the intensity of the cardiac postganglionic sympathetic nerve activity, the number of NE molecules that bind to cardiac adrenergic receptors and induce biological effects. [10-12] Increased sympathetic tone and abnormal responsiveness of the parasympathetic nervous system is associated with heart failure and elevated levels of plasma NE correlate with worse outcomes. [13] Based on the abovementioned mechanisms, clinical studies, and animal experiments have shown that the remodeling of postcardiac sympathetic neurons in the stellate ganglion can cause cardiac sympathetic overactivation and ventricular arrhythmias. [14] Therefore, physical stimulation of the cervical spine adjacent to the stellate ganglion site by the iatrogenic situation made mass effect or such as deformities or damage, may trigger the arrhythmia. Although stellate ganglion block (SGB) is often performed in pain clinics without proper monitoring, the site of the block may directly trigger the ganglion. In fact, the location of the surgical approach is almost identical to the approach for SGB, especially the right stellate ganglion block, which may require more careful monitoring for adverse effects. It has also been observed in females that the cricoid cartilage is located relatively cranially, especially when the patient is in a position similar to the present case with neck extension. As the cricoid cartilage is relatively unobstructed and relatively easily exposed to the outside environment, palpation of the transverse process of C6 may increase the likelihood of arrhythmias if force is used. [15] The stellate ganglia are commonly bilateral and there is little evidence of dominant bilateral innervation. [16] But the sinoatrial node is primarily innervated by cardiac postganglionic sympathetic nerve terminals from the right stellate ganglion. [17] Therefore, physical cervical sympathetic stimulation especially right side, such as deformities or damage, may trigger the arrhythmia by sinoatrial node sympathetic tone change. There is only one study showing relatively good results with the left stellate ganglion block in a group of patients who were not effectively managed with predominantly right stellate ganglion block. [18] In addition, animal studies have shown that asymmetric electrical stimulation of the supraventricular node induces transient acute AF due to sympathetic overactivation. [19] In a study of patients with a history of

atrial fibrillation, the inducibility of atrial fibrillation was significantly reduced after stellate ganglion blockade. Even in patients with induced atrial fibrillation, the arrhythmia duration was reduced. [20] In addition, some studies have shown that patients undergoing bilateral stellate ganglionectomy experienced a significant reduction in the incidence of electrically induced atrial fibrillation and the preservation of sinus rhythm. [21] In this case, it might have been necessary to identify which side of the cervical spine was retracted and affected the stellate ganglion during surgery. Although there was a change in the size and rate of the p-wave with drug administration, the arrhythmia resolved safely after a few minutes, and the operation could be safely continued.

As the ECG remained normal in the postoperative period without any recurrence, the occurrence of AF in this patient was not caused by the patient's underlying disease but by the presence of some stimulation related to the heart rhythm in the surrounding area caused by the stimulation of the surgical site. In particular, based on the fact that the surgical site was C6–7, the area was stimulated with a retractor to expose C6–7 during surgery at the time of ECG changes. Further, the area was the site of a procedure involving the stellate ganglion. Thus, the arrhythmia was temporarily induced by the overactivation of the autonomic nervous system caused by physical stimulation of the stellate ganglion.

For the acute control of hemodynamically stable AF, beta-blockers such as diltiazem and verapamil are useful. Diltiazem is the preferred calcium channel blocker as it is safe and effective. Verapamil and diltiazem should not be used in patients with advanced heart failure with heart block or dyssynchrony or in patients with preexcitation. The heart-rate-lowering effect of beta-blockers is related to the reduced sympathetic nervous system activity. Esmolol is generally preferred because of its rapid onset of action., which is useful in patients with PSVT and atrial fibrillation. In patients with PSVT, these drugs can be administered intravenously to inhibit tachycardia or when urgent rate control is required in conditions including atrial fibrillation and flutter. [4] Unlike adenosine, these drugs can occasionally cause bradycardia and hypotension. Therefore, they are safe to infuse slowly over 2–3 min. Maintenance doses can be continued for several days if

needed. [22] In this case, adenosine was used to differentiate from supraventricular tachycardia. Adenosine activates K+ channels by acting on α1 receptors and increasing the permeability of the cell membrane to K+, shortening the action potential period of the freezing node, atrioventricular node, and atrial cells, and activating the membrane potential. It causes atrioventricular conduction disorder. However, its half-life is extremely short, within approximately 10 s. Thus, it is considered an extremely effective and safe drug for terminating supraventricular tachycardia. In addition, adenosine is used for the diagnosis of AF, ventricular fibrillation, and atrial tachycardia based on the fact that it causes temporary atrioventricular block. Moreover, it is used in the differential diagnosis of arrhythmias with a wide QRS width. [23] However, there are reports of adenosine causing VT and ventricular fibrillation. Therefore, a more cautious approach should be recommended. [24] During an arrhythmia, lidocaine was used. That is, it suppresses electrical activity in depolarized (ischemic, arrhythmogenic) tissues, and it is, therefore, extremely effective in suppressing arrhythmias associated with depolarization (e.g., ischemia, digitalis poisoning). However, it is relatively ineffective against arrhythmias that occur in normally polarized tissues (e.g., AF and fibrillation). In this case, it was effective when used for additional premature ventricular contractions after arrhythmia is controlled, but not when used for atrial arrhythmia.

Atrioventricular block induction is extremely useful in detecting narrow QRS tachycardia. In addition to the Valsalva maneuver, the other methods used to induce atrioventricular block include carotid sinus massage and drugs such as adenosine and verapamil. Atrioventricular node regression tachycardia and atrioventricular regression tachycardia in which the atrioventricular node is essential to the regression circuit are inhibited by this method. AF is easily recognized based on the presence of a distinct flutter wave during the atrioventricular block. [25] In this case, the arrhythmia did not resolve after adenosine tranquilization and the persistence of flutter waves on the ECG waveform indicates AF rather than PSVT. Electrical cardioversion should be performed when a patient with AF is hemodynamically unstable. However, pharmacologic therapy can be attempted if the patient is considered hemodynamically stable. If cardioversion is

performed, acute antithrombotic therapy can be beneficial because the short-term stroke rate after cardioversion for AF was 0%–7% based on a meta-analysis. The rate of thrombosis in patients with persistent AF is high, with an annual average of 3%. [26] In this case, the patient was hemodynamically stable; therefore, pharmacologic treatment could have been prioritized. The long-term risks of AF also include an increased risk of stroke, heart failure, and mortality. [27]

CONCLUSION

In cases as in our patient, where the anatomy that must be accessed for surgery can cause arrhythmias, appropriate monitoring and the clinician's empirical ability to act proactively in case of an arrhythmia are required. If an arrhythmia occurs in the context of stimulation of the right stellate ganglion during cervical spine surgery, identification of triggers to consider, correction of the appropriate triggers, and prevention of migration to fatal arrhythmias should be considered. And the patient with a relatively high risk of AF, preoperative ECG should be performed to assess the presence of tricuspid regurgitation. Moreover, arrhythmias such as AF and PSVT can occur during surgery due to various factors and can have similar electrocardiographic characteristics, thereby making them challenging to differentiate from each other and treat. Hence, clinicians should be knowledgeable about the electrocardiographic features of these arrhythmias.

Figure Legends

Figure 1 Periopreative electrocardiogram of the patient. A: Preoperative electrocardiogram, Sinus rhythm, HR 57 bpm; B: Postoperative electrocardiogram (Post-operative day 0), Accelerated junctional rhythm, HR 83bpm; C: Postoperative electrocardiogram (Post-operative day 2), Sinus rhythm, HR 58bpm. **Figure 2** Images of the patient. A: MRI image: C6-7 disc bulging with marginal spur and neural foraminal stenosis C6-7; B: Intraoperative picture with C-arm image: Tracing with cervical distractor to expose the surgical site & Spinal fusion retraction device used in this operation. Indication device for exposure C6-7 intervertebral disc space.

Figure 3 Hemodynamic data and events with drug administration and electrocardiogram in chronological order. A: First, arrythmia occurred suddenly during approach the cervical spine; B: The flutter like ECG occurred continuously; C: Adenosine was administered, and the ECG pattern was changed to Atrial Flutter pattern; D: Diltiazem injection for management of Atrial Flutter; E: Disappear the flutter like ECG; MBP: mean blood pressure; HR: heart rate; BP: blood pressure; BIS: bispectral index; ETCO2: end-tidal CO2

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