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ANAESTHESIA
Case Report

Intra-Operative Carotid Sinus Hypersensitivity Secondary to Surgical Stimulation Resulting in Cardiac Arrest

Osman Salaria[1], Omar Viswanath[1]*, Orlando Santana[2], Hector Davila[1]

[1]Miami Beach Anesthesiology Associates, Inc., Department of Anesthesiology, Mount Sinai Medical Center, Miami Beach, Florida, U.S.A

[2]Columbia University Division of Cardiology at the Mount Sinai Heart Institute, Miami Beach, Florid, U.S.A

***Corresponding Author:** Omar Viswanath, Miami Beach Anesthesiology Associates, Inc., Department of Anesthesiology, Mount Sinai Medical Center, Miami Beach, Florida, U.S.A.

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Abstract

We present a case of a 79 year old male who presented to the hospital for an elective anterior cervical discectomy and fusion. Intraoperatively, during the initial surgical incision, the patient developed pauses and progressive bradycardia followed by profound hypotension and ventricular fibrillation. The procedure was aborted and the patient was successfully resuscitated and cardioverted to sinus rhythm. A cardiovascular workup revealed a diagnosis of carotid sinus hypersensitivity secondary to surgical stimulation resulting in cardiac arrest.

Keywords: *Carotid Sinus; Fibrillation; Parasympathetic; Electrophysiologist*

Introduction

The carotid sinus is a dilatation of the internal carotid artery in proximity to its base near the common carotid artery. It forms the major component of the baroreceptor reflex mechanism. The baroreceptor mechanism monitors changes in blood pressure and trasmits these signals through the afferent glossopharyngeal and vagus nerves to the nucleus tractus solitarius. Efferent signals are then sent through the sympathetic and parasympathetic portions of the autonomic nervous system. This homeostatic mechanism helps prevent frequent fluctuations in blood pressure.

Carotid sinus hypersensitivity (CSH) is a rare condition associated with an exaggerated response of this system in response to stimulation in the form of manual pressure, tight collars, or neck movements. This condition primarily manifests in elderly males with a history of coronary artery disease, hypertension, or arrhythmias. There are three subtypes of CSH:

a. cardioinhibitory (producing 3 second pauses)
b. vasodepressor (a reduction in blood pressure > 50 mm Hg)
c. a mixed component comprising of both subtypes. [1]

The present case report illustrates the mixed component type CSH.

Case Description

A 79 year old African American male with a past medical history of hypertension presented to the hospital after a syncopal episode and fall from his bed. His daily medications included tamsulosin, lisinopril, hydralazine, hydrochlorothiazide. He also complained of worsening bilateral lower extremity weakness and fluctuating numbness from the neck down. Prior to the fall, the patient stated that he would ambulate favoring more his right side. Magnetic resonance imaging of his cervical spine revealed severe disc herniations in the C5-6 and C6-7 area of the spinal cord and he was advised to undergo an anterior discectomy and fusion of this area.

Pre-operative evaluation included a negative anesthesia history, well controlled hypertension, an electrocardiogram (ECG) showing normal sinus rhythm with a ventricular rate of 76 bpm, and an echocardiogram showing an ejection fraction of 65-70%. On the day of surgery, in the pre-anesthesia holding unit, the patient was monitored according to our hospital's standard protocol including 3-lead ECG, non-invasive blood pressure cuff, and pulse oximeter. Pre-operative vitals included a blood pressure (BP) of 164/83 mm Hg, heart rate (HR) 80/min, and a 100% pulse oxygen saturation. Pre-operative preparation for the procedure consisted of a peripheral intravenous line, a radial arterial line, and a right subclavian vein double lumen antibiotic coated catheter. In the operating room successful intubation was achieved with a glidescope using a 7.5 mm endotracheal tube after sucessful bag mask ventilation. Proper patient positioning and surgical field preparation was then obtained with both the anesthesiologist and neurosurgeon present. Prior to incision, the patient had stable vital signs that included a BP of 146/78, HR 86, and an oxygen saturation of 100%.

Immediately after manipulation of his anterior neck during the surgical incision, the patient developed 3 second pauses and bradycardia with a HR of 34/min followed by a BP 63/35 mmHg. The end tidal carbon dioxide also dropped precipitously from 32 to 24 at this time. Bolus doses of both ephedrine and phenylephrine failed to raise the BP and HR. Atropine and epinephrine were immediately given to treat the bradycardia and hypotension. The patient then developed ventricular fibrillation. Advanced cardiac life support protocol was initiated, cardiopulmonary resuscitation and cardioversion were performed that included six electrical shocks and multiple boluses of chronotropes and pressors. During this time an intraoperative transesophageal echocardiogram was performed and did not reveal any cardiac wall motion abnormalities.The total resuscitation time was 18 minutes resulting in return of spontaneous circulation. Cardiology was consulted, and once the patient's vitals stabilized enough, he was transported directly to the neurosurgical intensive care unit for monitoring and further workup.

On post-operative day one, the patient was extubated sucessfully maintaining an oxygen saturationof 99-100%. All vasopressors were discontinued as the patient remained normotensive. He was responsive to verbal commands and showed no evidence of any neurological deficit compared to baseline pre cardiac arrest status. An acute pulmonary embolus was ruled out by computed tomography angiography. Chest x-ray revealed mild fluid overload with no other abnormalities. Laboratory results revealed serum electrolytes within normal limits, hemoglobin and hematocrit levels at 12/37.6, BUN/Creatinine of 18/1.42, and cardiac troponin levels were negative. Repeat echocardiogram demonstrated a hyperdynamic left ventricle with an ejection fraction > 70%. A Doppler ultrasound of the carotids performed showed no evidence of hemodynamically significant carotid artery stenosis. In view of the patient's history and the preceding events, it was concluded that the patient's intraoperative cardiac arrest was a result of an exaggerated hypersensitive response by the carotid sinus or CSH secondary to surgical stimulation.

After three days in the neurosurgical intensive care unit, the patient was transferred to a telemetry floor. Two weeks after the intraoperative cardiac arrest, the patient agreed to proceed with the original surgical procedure. Pre-operatively he had a temporary transvenous pacemaker inserted electively by an electrophysiologist via the right subclavian vessel and immediately followed with a successful C5-6 and C6-7 anterior discectomy and fusion with no intraoperative adverse events or complications. He did well post-operatively and was referred to the electrophysiologist for follow-up and further evaluation for possible permanent pacemaker implant.

Discussion

CSH is a rare condition with a complex pathophysiology which is poorly understood. [2] Although, it consists of a simple reflex arc with afferent, central, and efferent outputs, the etiology is complex as hypersensitivity can occur anywhere along this arc or at the target organs. [2] Some view a linkage between CSH and susceptibility to reflex syncope supported by exaggerated oculo-vagal bradycardic response and tilt-table induced syncope present in CSH patients. [3] It has also been shown that symptomatic patients with CSH have a lower ability to maintain cerebral blood flow in response to hypotensive episodes. [4]

The incidence of intra-operative CSH has been reported to be at 28% in the elderly and 10% in younger patients. [5] The cardioinhibitory type comprises 70-75% of cases. The predominant manifestation is a decreased heart rate, which results in sinus bradycardia, atrioventricular block. The vasodepressor type comprises 5-10% of cases. The predominant manifestation is a decrease in blood pressure without a change in heart rate. While the mixed type comprises 20-25% of cases and consists of a decrease in heart rate and blood

pressure. When the index of suspicion is high, a proper diagnosis requires ECG and BP measurement of sustained 10 second carotid sinus massage to note for changes in HR and BP respectively. [6]

The management of CSH is based on the frequency and severity of each patient's symptoms. Some patients can be treated with education and lifestyle changes. However, some may have incapacitating and recurrent symptoms which require therapy. Permanent pacemaker implantation is generally considered an effective treatment for cardio-inhibitory CSH and mixed forms of CSH. The current American College of Cardiology/American Heart Association/Heart Rhythm Society clinical practice guidelines consider permanent pacing therapy to be a class I indication in patients with recurrent syncope caused by spontaneous carotid sinus stimulation and carotid sinus pressure that induces ventricular asystole of more than 3 seconds. Permanent pacing is considered a class IIa indication in patients with recurrent syncope without clear, provocative events and with a hypersensitive cardio-inhibitory response of 3 seconds or longer. [7]

In conclusion, carotid sinus hypersensitivity is a rare condition with a vaguely understood pathophysiology. Given the rarity of this diagnosis along with the potentially significant patient morbidity and mortality, CSH needs to be part of the anesthesiologist's differential diagnosis when profound bradycardia and hypotension suddenly present intra-operatively while under surgical stimulation.

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