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Adverse Effects of Spinal Anesthesia in a Patient with Idiopathic Hypertrophic Subaortic Stenosis

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Idiopathic hypertrophic subaortic stenosis (IHSS) is a cardiomyopathy characterized by asymmetrical septal hypertrophy (ASH) and obstruction to the left ventricular outflow tract. The anesthetic management of this condition should focus on avoiding measures that increase left ventricular outflow resistance.

REPORT OF A CASE

An 85-year-old woman was admitted to the hospital after falling from a bed that evening. A subcapital fracture of the left femur was found. She had a long history of moderate dyspnea on exertion associated with palpitations and intermittent episodes of exertional and nonexertional chest pain relieved by sublingually administered nitroglycerin. A harsh grade III/VI systolic murmur and a high-pitched grade III/VI systolic murmur were audible along the left sternal edge and at the apex, respectively. Lungs were clear to auscultation and percussion. The ECG revealed regular sinus rhythm, Q-waves in leads II, III, AvF, and V4-6. Laboratory investigations were normal except for a mild anemia (Hb, 10.4 g%). Although congestive heart failure or myocardial infarction were not evident, a diagnosis of mild aortic stenosis and mitral incompetence was made. Upon arrival in the operating room, an iv infusion of 500 ml lactated Ringer's solution with 5% dextrose was given slowly, together with 100 μg of fentanyl iv, and a modified V5 lead was placed. Spinal anesthesia with tetracaine 8 mg in dextrose 80 mg was performed in the left lateral position, whereupon the patient acknowledged the sensation of heat and numbness in her lower extremities. Within a minute of being turned to the supine position, the patient became markedly diaphoretic, with a fall in arterial blood pressure from 130/80 to 70/50 mmHg and a rise in heart rate from 90 to 130 beats/min. She complained of severe precordial chest pain accompanied by nausea and vomiting. Auscultation of the chest revealed bilateral basal rales. Following an increased rate of iv crystalloid infusion and the iv injection of ephedrine 15 mg and naloxone 0.4 mg, the vital signs returned to normal. A 12 lead ECG showed marked ST segment depression in leads II, III, AvF, and V4-6. Surgical reduction of the fracture was cancelled and the patient transferred to the cardiac care unit for further evaluation. She was treated with iv nitroglycerin for the next few days and remained stable except for recurring episodes of nonexertional chest pain and dizziness. ECG changes of lateral and inferior wall ischemia persisted. An echocardiogram performed 2 days after the spinal block revealed the classic findings of idiopathic hypertrophic subaortic stenosis, viz. left atrial enlargement, asymmetrical septal hypertrophy (ASH), systolic anterior motion (SAM), and mitral regurgitation. Response to discontinuation of nitroglycerin and institution of propranolol therapy was marked by the disappearance of the episodic chest pains and a return to normal of the ECG changes. One week later the patient was rescheduled for surgery. After premedication with propranolol 20 mg po, flow-directed and radial arterial catheters were inserted. Anesthesia was induced with thiopental and maintained with halothane 0.5–0.7% via an endotracheal tube and incremental iv doses of fentanyl. The anesthetic and operative course were uncomplicated except for the infusion of packed erythrocytes during surgery. The trachea was extubated at the end of the procedure, and the postoperative course was uneventful.

DISCUSSION

Despite the diversity of terminology, intensive investigation has concluded that IHSS is part of hypertrophic cardiomyopathy, a disease with protean manifestations.[1–3] ASH is the common anatomic denominator in hypertrophic cardiomyopathy and is genetically inherited as an autosomal dominant trait.[4,5] Patients with ASH may be asymptomatic, have left ventricular outlet obstruction already in the basal resting state or develop obstruction when hemodynamically provoked. The patients with the obstructive component are commonly referred to as having IHSS. Despite well-documented signs and symptoms, the clinical picture varies considerably and IHSS is rarely considered in the clinical diagnosis of the elderly patient.[6] Furthermore, IHSS often is misdiagnosed as the symptoms suggest more common hypertensive, vascular and valvular diseases, as was the case with the above patient. The mechanism of left ventricular outlet obstruction, as visualized on echocardiogram, is caused by the abnormal forward motion of the mitral valve and the apposition of its anterior leaflet with the greatly hypertrophied septum (called SAM). Associated findings are left atrial enlargement, decreased left ventricular compliance, prolonged left ventricular isovolumic relaxation, impaired left ventricular diastolic filling, and mitral regurgitation.[7] A large pressure difference or obstructive gradient develops across the left ventricular outlet and can be as high as 175 mmHg. This ventriculo-aortic gradient is labile, in contrast to a fixed left ventricular outlet obstruction. Three mechanisms, physiologic or pharmacologic, produce or

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increase this dynamic obstruction by intensifying the apposition of the anterior mitral leaflet against the septum: 1) Increased myocardial contractility; 2) decreased preload; and 3) decreased afterload. Conversely, reduction in contractility or increase in the preload or afterload reduces or abolishes the obstruction.[1] The myocardial mass is increased consistently, and angina pectoris results from an imbalance in the myocardial oxygen supply and demand. The coronary vessels, however, are often normal, although obstructive coronary artery disease may develop in patients over the age of 45.[8] Subsequently, factors that serve to increase the left ventricular outlet obstruction also serve to decrease the coronary blood supply. The mainstay of treatment of IHSS has for many years been beta-adrenergic blockade with propranolol. Propranolol effectively decreases the myocardial contractility and oxygen consumption, increases the left ventricular compliance, and prevents marked increases in the heart rate. The clcium entry blocking drugs, verapamil and nifedipine also have demonstrated beneficial clinical effects and appear to improve the left ventricular diastolic filling, without any effects on myocardial contractility.[9-11]

The anesthetic management of this condition has been described.[12,13] Those techniques or agents that increase the obstructive gradient should be avoided. Digitalis glycosides, isoproterenol, nitroglycerin, amyl nitrate, high airway pressure (Vasalva maneuver), and hypovolemia have been demonstrated to increase the obstructive gradient.[1,14-17] Regarding inhalation anesthetics, halothane decreases myocardial contractility and as such reduces the ventriculoaortic gradient. The cardiovascular hemodynamics in these patients have been improved with halothane and fluoroxene.[18,19] In contrast, enflurane and isoflurane might not be suitable because they both decrease the peripheral vascular resistance and increase heart rate, factors that theoretically could increase the obstructive gradient.[20,21] Similarly, spinal block–induced decreases in the preload and afterload secondary to peripheral venous pooling and sympathetic blockade could act to increase the left ventricular outlet obstruction.[22,23]

These effects may preclude the choice of regional anesthesia in patients with IHSS. However, if regional anesthesia is performed and hypotension ensues, an alpha-agonist such as phenylephrine or methoxamine should be given because they increase systemic vascular resistance while being devoid of any positive chronotrophic or inotrophic properties. Methoxamine has been demonstrated to consistently abolish or reduce the obstructive gradient.[1,16]

Patients who develop left ventricular outlet obstruction only when hemodynamically provoked present a special risk. Typically, they are first degree relatives of patients with IHSS or have a family history or hypertrophic cardiomyopathy. Cardiac catheterization provides a good example of this hemodynamic provocation, when amyl nitrate and phentolamine are used to provoke and evaluate the obstructive gradient.[24] Similarly, during anesthesia, a combination of hypovolemia- and catecholamine-induced increases in heart rate and myocardial contractility could provoke left ventricular outflow obstruction. IHSS, although uncommon, should not be forgotten in those patients who suddenly deteriorate after the intraoperative administration of drugs with positive inotropic or venodilator properties. The presence of a new heart murmur or characteristic arterial and venous waveforms may point to the diagnosis.

In the above patient, the administration of a spinal anesthetic, despite a preload of iv crystalloid administration produced severe hypotension, tachycardia, and consequent myocardial ischemia. The decreased preload and afterload secondary to venous pooling and sympathetic blockade, respectively, acted to either provoke or increase left ventricular outflow obstruction and decrease the coronary blood supply. The marked tachycardia also served to decrease the coronary perfusion time during ventricular diastole.[25] It is surprising that sublingual nitroglycerin was consistently effective in relieving the patient's anginal symptoms prior to the hospital admission. Theoretically, the patient's symptoms should have become worse after sublingual nitroglycerin. Perhaps the obstructive component was not present in the basal resting state and that the effects of nitroglycerine directly on the coronary vessels viz. coronary vasodilation and improved collateral flow[26] outweighed its ability to provoke the obstructive component. Braunwald *et al.* did note that, in spite of nitroglycerin-induced increases in the ventriculo-aortic gradient, a clinical history of intolerance to nitroglycerin was not detected in any of his patients with IHSS.[1] The cardiovascular effects of pain and stress due to the fracture may have provoked the obstructive component. Hemodynamic intensification of the obstruction might explain the persistent ECG ischemic changes and episodes of chest pain and dizziness, when the patient was treated with iv nitroglycerin in the cardiac care unit.

In summary, spinal anesthesia was poorly tolerated in this patient with undiagnosed IHSS. The secondary effects of sympathetic blockade contributed to increasing the left ventriculo-aortic gradient with subsequent severe myocardial ischemia.

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Anesthesia for Neodymium–YAG (Nd–YAG) Laser Resection of Major Airway Obstructing Tumors

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The development of technology to permit passage of laser energy through fiberoptic filaments allows ablation of previously nonoperable airway obstructions and improvement in respiratory function.[1–5] Until development of neodymium–yttrium-aluminum-garnet (Nd–YAG) laser, resection of these airway lesions was done primarily by carbon dioxide laser. Use of the carbon dioxide laser is restricted by a wavelength (10,600 nm) that is too long to permit passage of significant energy through a flexible bronchoscope.[6] Thus, lesions not visualized through a rigid bronchoscope are inaccessible. The argon laser has a shorter wavelength (514 nm) that will pass through a fiberoptic filament. However, argon energy is absorbed by hemoglobin, thus limiting tissue penetration. Nd–YAG laser (wavelength 1,064 nm) is conducted readily through fiberoptics, is poorly absorbed by hemoglobin, and has good tissue penetration.[4] These qualities make it suitable

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