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

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An 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 a 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 i.v. 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 i.v. crystalloid infusion and the i.v. 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 i.v. 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 i.v. doses of fentanyl. The anesthetic and operative course were uneventful 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
increase this dynamic obstruction by intensifying the
position of the anterior mitral leaflet against the septum:
1) Increased myocardial contractility; 2) decreased pre-
load; 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 nor-
mal, 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 con-
tractility and oxygen consumption, increases the left ven-
tricular compliance, and prevents marked increases in
the heart rate. The clusc 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 gly-
cosides, isoproterenol, nitroglycerin, amyl nitrate, high
airway pressure (Vasala maneuver), and hypovolemia
have been demonstrated to increase the obstructive gra-
dient.1,14-17 Regarding inhalation anesthetics, halothane
decreases myocardial contractility and as such reduces
the ventriculoadiastolic gradient. The cardiovascular he-
odynamics in these patients have been improved with halothane and fluoroxxene.18,19 In contrast, enflurane and
cisflurane might not be suitable because they both de-
crease 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 pe-
ripheral 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 pheneylephrine or methoxamine should
be given because they increase systemic vascular resistance
while being devoid of any positive chronotropic or in-
otrophic properties. Methoxamine has been demonstrated
to consistently abolish or reduce the obstructive grad-
ient.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 car-
diomyopathy. Cardiac catheterization provides a good
example of this hemodynamic provocation, when amy-
l nitrate and phenolamine are used to provoke and eval-
uate the obstructive gradient.24 Similarly, during anes-
thesia, a combination of hypovolemia-and catecholamine-
induced increases in heart rate and myocardial contrac-
tility could provoke left ventricular outflow obstruction.
IHSS, although uncommon, should not be forgotten in
those patients who suddenly deteriorate after the intra-
operative 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 adminis-
tration produced severe hypotension, tachycardia, and con-
sequent 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 cor-
onary blood supply. The marked tachycardia also served
to decrease the coronary perfusion time during ventric-
ular 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 com-
ponent was not present in the basal resting state and that
the effects of nitroglycerine directly on the coronary ves-
sels vix. coronary vasodilation and improved collateral
flow26 outweighed its ability to provoke the obstructive
component. Braunwald et al. did note that, in spite of
nitroglycerin-induced increases in the ventriculoadiastolic
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. Hemo-
dynamic 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 ef-
s of sympathetic blockade contributed to increasing the
left ventriculoadiastolic gradient with subsequent severe
myocardial ischemia.

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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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{4} These qualities make it suitable