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Intraoperative Diagnosis of Hypertrophic Obstructive Cardiomyopathy

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A thickened interventricular septum, a decrease in the size of the left ventricular outflow tract, and systolic anterior motion of the anterior leaflet of the mitral valve characterize hypertrophic cardiomyopathy (HCM).[1–3] These changes may cause left ventricular outflow obstruction, giving origin to the term hypertrophic obstructive cardiomyopathy (HOCM) or, in older terminology, idopathic hypertrophic subaortic stenosis or asymmetric septal hypertrophy.[1,3] Once thought to be rare, HOCM may occur relatively commonly, especially in elderly women.[4–6]

HOCM represents a potential problem during anesthesia because positive inotropic drugs, decreased venous return, or decreased systemic vascular resistance may increase dynamic ventricular outflow obstruction.[1,3,7] In our case, intraoperative diagnosis of previously unrecognized HOCM facilitated the care of this patient.

REPORT OF A CASE

A 73-year-old 55-kg woman was scheduled for open reduction and internal fixation of the right hip. Preoperative evaluation disclosed good general health except for occasional palpitations and mild, untreated hypertension. The patient took no chronic medications and had undergone three previous genitourinary operations without incident. Physical examination disclosed no cardiovascular or pulmonary abnormalities. No cardiac thrills, murmurs, gallops, or clicks were detected before surgery. There was no clinical evidence of hypovolemia. Laboratory testing revealed no significant abnormalities. The chest radiograph was normal. The ECG showed a normal sinus rhythm at a rate of 75 beats/min with nonspecific ST-T wave changes.

Upon the patient's arrival in the operating room, a blood pressure cuff, a lead V5 ECG, and precordial stethoscope were applied. Auscultation via the stethoscope revealed a faint systolic ejection murmur for the first time.

Following the iv administration of thiopental 200 mg and succinylcholine 100 mg, the trachea was intubated. Anesthesia was maintained with enflurane, pancuronium 3 mg iv, and 50% nitrous oxide in oxygen. The arterial blood pressure was extremely labile, characterized by hypertension in response to stimulation and hypotension in response to low concentrations of enflurane (0.25–0.5%). Arterial blood pressure ranged from 70/40 mmHg during preparation of the operative site to 190/100 mmHg following incision. Ephedrine, 5 mg, was given iv on three occasions. Two of the three doses produced an increase in blood pressure. The third dose produced no effect.

Approximately 20 min after incision, a 200 ml surgical blood loss occurred rapidly. Auscultation through the esophageal stethoscope revealed a loud systolic click, followed immediately by an unequivocal increase in the intensity of the systolic murmur. An increasing murmur in the setting of an acute decrease in venous return prompted a presumptive diagnosis of HOCM. During the next hour, the patient received an 1,800-ml iv infusion of lactated Ringer's solution and one unit of packed erythrocytes reconstituted with normal saline to a total volume of 750 ml. The rapid iv administration of crystalloid increased the patient's tolerance for enflurane, which subsequently was reinstituted in an inspired concentration of 0.33% with fentanyl 150 mcg iv, pancuronium 3 mg iv, and nitrous oxide 50% in oxygen. During blood transfusion, the cardiac murmur, as perceived through the esophageal stethoscope, markedly decreased in intensity, and the systolic click disappeared.

Postoperatively, precordial auscultation was repeated in an effort to clarify the intraoperative events. At that time an early systolic click and a grade II/VI systolic murmur were confirmed. Performance of a Valsalva maneuver increased the murmur's intensity. Echocardiography revealed a thickened interventricular septum, a small left ventricular chamber, and a narrow left ventricular outflow tract consistent with HCM. The anterior leaflet of the mitral valve touched the interventricular septum during diastole but not during systole. Propranolol 20 mg po tid was instituted. Subsequently, the patient has experienced fewer palpitations.

DISCUSSION

This patient demonstrates many of the characteristics of HCM: an elderly woman with a history of hypertension and minimal distinctive preoperative symptoms.[4–6] The age distribution at diagnosis of HCM is bimodal, with a peak incidence early in the fifth decade and a second peak early in the seventh decade.[5] Petrin and Tavel[6] noted a 0.022% incidence of HCM in a 6-yr retrospective study in a large community hospital. Among elderly patients, most are female.[4–6]

Although approximately one-third of all cases are genetically transmitted in an autosomal dominant fashion,[4] this patient had no family history of cardiac disease. Sporadic cases may be related to long-standing hypertension or to an abnormal response by cardiac muscle to prolonged catecholamine stimulation.[1,6] Petrin and Tavel[6] noted that 78% of 32 females over the age of 50 years who developed HCM had hypertension by history.

The minimal findings on preoperative evaluation are also typical of HCM. Since left ventricular outflow obstruction represents a dynamic process, many elderly patients may not develop symptomatic obstruction during

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daily activities. Common nonspecific cardiac symptoms may include dyspnea, angina, presyncope, syncope, and palpitations.[2,4,8] In a study of 27 elderly patients with HCM, Albin *et al.*,[4] could not distinguish those patients from others with atherosclerotic, hypertensive, or valvular cardiac disease on the basis of symptoms. They noted that elderly patients with HCM might be completely asymptomatic and that the disorder could exist without any clinical findings.

Physical examination may be inconclusive. The murmur may be noted only after a prolonged history of hypertension.[6] Petrin and Tavel[6] suggest that HCM should be considered in any elderly patient in whom a systolic murmur develops during long-standing hypertension. Classically, maneuvers that decrease venous return, such as hypovolemia, the Valsalva maneuver, and vasodilator drugs, may increase the intensity of the systolic murmur.[2,3] Conversely, intravascular volume expansion, squatting, and pharmacologic vasoconstriction may decrease the intensity of the murmur.[2,3] A fourth heart sound[2,3,4,8] and an increase in murmur intensity of the beat immediately following a premature ventricular contraction[3] also may occur.

Routinely obtained preoperative tests may not suggest the diagnosis of HCM. Although left ventricular enlargement may be noted on chest radiography,[3,4] it may be entirely normal.[4] Electrocardiographic changes are nonspecific, including atrial and ventricular arrhythmias,[2,4] ST-T wave changes,[2] and evidence of left ventricular hypertrophy.[2,8] Ten to 15% of patients will have no evidence of left ventricular hypertrophy, despite a greatly increased cardiac muscle mass.[2] Echocardiography or cardiac catheterization usually establish the diagnosis. Echocardiography should identify an interventricular septum that is more than 1.3 times as thick as the posterior left ventricular free wall, a decreased luminal diameter of the left ventricular outflow tract, or systolic anterior motion of the anterior leaflet of the mitral valve.[2,3] Cardiac catheterization may disclose a pressure gradient within the left ventricular outflow tract with a normal aortic valve pressure gradient.[3] Provocative measures may be required to elicit outflow obstruction during echocardiography or cardiac catheterization.[2,7] Provocative maneuvers were not performed in this patient, possibly accounting for the failure to demonstrate outflow obstruction during echocardiography.

The intraoperative diagnosis in our patient depended upon the observation that a low-grade systolic murmur, unnoticed before surgery, increased dramatically following rapid hemorrhage. The intensity of the murmur did not increase with either of two hypotensive episodes occurring before the incision. Perhaps the absence of an increase in murmur intensity during hypotension precipitated by enflurane was due to the negative inotropic properties of enflurane[9] or to the absence at that time of surgically induced sympathetic discharge. Similarly, the small iv doses of ephedrine employed may have increased blood pressure without accentuating murmur intensity because the vasoconstrictor effects exceeded the inotropic effects of a mixed alpha and beta adrenergic drug in this patient.[10] Surgical stress may have increased myocardial contractility, which, in conjunction with a sudden reduction in venous return, produced a marked increase in outflow obstruction.

Following the presumptive diagnosis of HOCM, our patient responded well to simple iv volume administration, a procedure intended to increase venous return and thereby increase the dimensions of the left ventricular outflow tract. Volume expansion is an appropriate early therapeutic response to hypotension in most patients undergoing anesthesia. Alternative diagnoses have to be considered if volume expansion alone does not resolve the hemodynamic difficulties. In particular, it is necessary to distinguish left ventricular failure from dynamic outflow tract obstruction, since left ventricular failure may require administration of potent beta-adrenergic drugs, an intervention that can aggravate outflow tract obstruction in HOCM. Consequently, treatment of more severe hemodynamic disturbances may require measurement of cardiac output and pulmonary artery occlusion pressure. For instance, an increasing pulmonary artery occlusion pressure, a decreasing cardiac output, and an increasing murmur in response to beta-adrenergic therapy may suggest worsening outflow tract obstruction. Hemodynamic monitoring also will permit assessment of the effects of beta-adrenergic blocker or vasoconstrictor drugs if these have been used in an effort to decrease outflow obstruction.[2,3,7,8]

We present this case as an illustration of HOCM in elderly, asymptomatic individuals in whom the diagnosis may not be suspected preoperatively. The occurrence of hypotension and an increase in the intensity of a systolic murmur during anesthesia should alert personnel to the possibility of HOCM. Although the diagnosis cannot be established conclusively intraoperatively, its possibility should prompt modification of intraoperative resuscitative measures.

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Treatment of a Duro-cutaneous Fistula Secondary to Attempted Epidural Anesthesia with an Epidural Autologous Blood Patch

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Several cases of duro-cutaneous fistula following attempted entry into the subarachnoid or peridural spaces in the lumbar region for diagnostic, therapeutic, or anesthetic purposes have been reported.[1–4] We describe a case in which the duro-cutaneous fistula was treated successfully using an autologous blood patch epidural.

REPORT OF A CASE

A 43-year-old woman (G_1P_0) was admitted in active labor. After initial history and physical examination revealed no apparent contraindications to regional anesthesia, the patient was placed in the left lateral position and prepared for continuous epidural anesthesia. An initial attempt to identify the epidural space was made in the midline of the L3-4 interspace using a 17-g Tuohy needle and the hanging drop technique; bone was encountered on this attempt. The needle then was withdrawn, and a second attempt was made at this same interspace (again using hanging drop technique). This attempt resulted in a free flow of clear fluid from the needle, and the needle was removed. A third attempt was made one interspace lower, using the loss-of-resistance technique. No CSF or blood was encountered; a test dose of 2 ml 0.25% bupivacaine was given through the needle without effect, and the catheter then was introduced without difficulty. After a second test dose was given through the catheter, continuous epidural anesthesia was begun. Delivery was uneventful, but analgesia during that time was described as "spotty perineal." The epidural catheter was removed in the delivery room postpartum and the patient transferred to the recovery room.

Approximately 18 h postpartum she complained of a headache and the ward nurse noticed "a wet spot about 6 inches wide" in the bed beneath her lumbar region. Her severe frontooccipital headache was relieved only partially by recumbency. She denied diplopia. Examination of her back revealed three apparent puncture sites: two in the L3–4 interspace and one in the L4–5 interspace. Clear fluid was dripping from one of the sites at L3–4 at a rate of 10–12 drops/min (with the patient in the left lateral position). The fluid was tested for glucose using a Dextrostix® reagent strip and found to contain 80–120 mg/dl glucose. The patient then was transferred back to the recovery room, where a surgical prep with povidone iodine was done on the lumbar and right antecubital regions. With the patient in the right lateral position, sterile drapes were applied and two attempts to identify the epidural space using the loss-of-resistance technique in the midline of the L2–3 interspace were made. On both occasions free flow of clear fluid was encountered at the same moment that loss of resistance was obtained. Moreover, after encountering fluid during the second attempt, the Tuohy needle was slowly and carefully withdrawn, while alternately aspirating and testing for loss of resistance; resistance was encountered at the same point where fluid no longer could be aspirated. The needle was removed and the patient positioned in the sitting position and reprepped and draped. A third attempt to enter the epidural space via the midline in the L2–3 interspace was made using the loss-of-resistance technique; no CSF return was noted in the sitting position. In retrospect, the fluid obtained following loss of resistance probably represented an epidural "pool," which was drained by gravity in the sitting position.

Autologous blood, 10 ml, then was injected via the needle into the epidural space. The needle then was flushed with 2 ml sterile saline and removed and the area sterilely dressed. Over the following 30 min, the patient received 2 l of Plasmalyte A® iv with dramatic improvement in her headache. No significant changes in blood pressure or pulse were noted following the administration of this solution.

Following the blood patch, the patient remained afebrile and asymptomatic during 3 days of in-hospital observation, and she was discharged with instructions to contact us immediately if any new symptoms developed. On follow-up appointments she has continued to be asymptomatic and has had no difficulty in keeping the rigorous pace of a new mother.

DISCUSSION

Duro-cutaneous fistula following attempted entry of either the subarachnoid or peridural spaces is a very rare event. Factors were present in the only three other cases reported[1–3] that could alter wound healing: 1) use of steroids in the peridural space, with possible deposition

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