General Anesthesia in a Child with a Dynamic, Vascular Anterior Mediastinal Mass

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THE patient with an anterior mediastinal mass presents the anesthesiologist with a formidable challenge. Severe respiratory and cardiovascular complications, including the inability to resuscitate, can ensue on induction of general anesthesia. We describe the anesthetic management of a child with a large dynamic vascular malformation located in the neck and mediastinum who underwent percutaneous sclerotherapy.

Case Report

A 9-yr-old girl was admitted to our institution for sclerotherapy of a large mediastinal vascular malformation. At age 2 yr, the patient was noted to have a small mass in the left neck, initially believed to be a hemangiomma, and was treated with steroids with no effect. She remained asymptomatic until age 7 yr, when she was evaluated for dyspnea. Workup at that time suggested the mass was an arteriovenous malformation that was intermittently compromising respiration. Magnetic resonance imaging and magnetic resonance angiography revealed an extensive, lobulated mass in the left neck and upper chest, extending from the angle of the mandible through the neck and thoracic inlet into the mediastinum below the aortic arch (figs. 1 and 2). The trachea was mildly compressed and displaced anteriorly and to the right. The left carotid and left vertebral arteries appeared encased by the mass. Extension of the vascular malformation into the retropharyngeal region of the hypopharynx was noted. Initial angiography showed the predominant blood supply to the malformation to be intimately associated with a feeding vessel to the anterior spinal artery, with venous drainage to the paraspinal veins andazygous system. Because of the risks of intervention, the patient was treated conservatively for 2 yr. During the 6 months before admission, however, she began to experience increasingly frequent episodes of dyspnea and stridor associated with neck swelling. There was progressive orthopnea, and she required three pillows for sleep. Pulmonary function tests demonstrated a mild obstructive impairment (table 1). As an extension of the mass, the patient was noted to have a soft tissue mass in the left thorax and mediastinum on chest radiography and computed tomography. There was some concern that this might be a malignant component of the lesion.

Received from the Departments of Anesthesiology and Radiology, Children's Hospital and Harvard Medical School, Boston, Massachusetts. Submitted for publication February 23, 1995. Accepted for publication December 15, 1995.

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Anesthesiology. V 84, No 4, Apr 1996

Fig. 1. A photograph of the child was taken at 7 yr of age, before treatment, demonstrating the anterior neck and suprachavicular soft-tissue mass.

Fig. 2. Magnetic resonance imaging of the chest before and after treatment. A: Axial T2-weighted image showing the extensive extent of the left carotid artery (LCA) and left vertebral artery (LVA) extending into the mediastinum.

2 blade: revealed the laryngeal inlet to be at the level of the cricoid cartilage, with no associated tracheal compression. Spontaneous respiration was maintained with a controlled respiration associated with the use of muscle relaxants. Direct laryngoscopy with a Macintosh
case reports

Fig. 2. Magnetic resonance imaging of the neck and mediastinum before treatment. (A) Coronal T1-weighted, gadolinium-enhanced image through the level of the trachea demonstrates the extensive enhancing lesion surrounding the left carotid artery (LCA) and displacing the trachea (T) to the right. (B) Axial T1-weighted image demonstrating the anteroposterior extent of the malformation at the level of the lower neck, again showing the mass to surround the LCA and left vertebral artery (LVA) and displace the trachea to the right.

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Discussion

Our patient's symptoms of stridor and orthopnea were suggestive of significant airway narrowing. Many patients with an anterior mediastinal mass will present with cough, dyspnea, stridor, or orthopnea. Absence of symptoms, however, does not assure a trouble-free anesthetic.1-3 Several authors have suggested that preoperative pulmonary function tests performed in the supine position may be helpful; flow-volume curves showing limitation of expiratory flow may presage airway collapse with general anesthesia.4,5 Measurement of tracheal cross-sectional area by computed tomography may be helpful: Patients with tracheal narrowing greater than 50% appear to be at significantly greater risk for airway obstruction. The combination of pulmonary function test abnormalities (increased residual volume and decreased peak expiratory flow ratios in supine compared to sitting position) and computed tomography may serve to enhance awareness of the patient with a mediastinal mass and significant airway...
compromise for whom general anesthesia is a serious risk.

We chose to induce anesthesia with inhalational agents and continue spontaneous ventilation, as recommended by Ferrari et al. In addition, we maintained 5 cmH2O positive end-expiratory pressure to prevent pharyngeal collapse and provide a modest defense against the loss of functional residual capacity that accompanies anesthetic induction. General anesthesia in the supine patient can exacerbate extrinsic airway compression by reducing chest wall diameter and promoting cephalad displacement of the diaphragm, thus reducing overall thoracic volume and decreasing the space for the mass. By increasing intrathoracic pressure, positive pressure ventilation may have resulted in a blood volume shift from the intrathoracic to the extrathoracic portion of the vascular malformation, creating a compressive effect on extrathoracic airway structures. This hypothesis is similar to the intrathoracic fluid shifting found by Warner et al. in their evaluation of chest wall motion and is supported by lack of recurrence in this patient after the initial embolization of the neck portion of the mass. It is not uncommon to have to place patients with a mediastinal tumor in semi-Fowler's or sitting position to decrease their orthopnea and maintain airway patency; often these patients choose such positions for comfort, as did ours. By performing tracheal intubation under deep inhalational anesthesia without the use of muscle relaxants, positive pressure ventilation was avoided, and a more normal transpulmonary pressure gradient was maintained, which may have decreased the shifting of blood to the extrathoracic portion of the malformation. In addition, airflow through conducting airways may be better maintained with spontaneous ventilation in such patients. Although not an issue in this case, respiratory problems can occur on emergence from anesthesia as a result of edema or surgical manipulation.

Table 1. Improvement in Pulmonary Function Tests after Sclerotherapy

<table>
<thead>
<tr>
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<th>% of Predicted</th>
<th>Difference after Sclerotherapy (%)</th>
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<tr>
<td>October 7, 1993</td>
<td></td>
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<tr>
<td>FVC (L)</td>
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<td>92</td>
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<tr>
<td>FEV1 (L)</td>
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<td>83</td>
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<td>FEV1/FVC</td>
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<td>PEFR (L/s)</td>
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<td>FEF 25-75% (L/s)</td>
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<td>76</td>
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<tr>
<td>December 22, 1994</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FVC (L)</td>
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<tr>
<td>PEFR (L/s)</td>
<td>56</td>
<td>26</td>
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<tr>
<td>FEF 25-75% (L/s)</td>
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<td>26</td>
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FVC = forced vital capacity; FEV1 = forced expiratory volume in 1 s; PEFR = peak expiratory flow rate; FEF = forced expiratory flow.

Hypoxemia at the end of the procedure (SpO2 of 84%) with an inspired oxygen concentration of 0.4 may have been related to progressive atelectasis with shunting, and acutely exacerbated by movement of fresh thrombi from the malformation into the central circulation. This was accompanied by mild hypotension, although the end-tidal carbon dioxide did not change. Treatment with 100% O2 and lightening of the anesthetic promoted resolution within several minutes. When administered in the venous circulation, the vasospasm effects of sclerotherapy are immediate, locally restricted, and involve the vascular endothelium and platelets in the area of injection, producing a locally hypercoagulable state. Although pulmonary vascular effects generally are believed to be benign after sclerotherapy with absolute alcohol, cardiac vascular collapse, preceded by hypoxemia and hypercarbia, has been reported in four patients undergoing ethanol embolization or sclerotherapy. In these cases, the mechanism was believed to involve pulmonary vasoconstriction after sclerosant injection.

Decreased cardiac output and/or blood pressure, even when ventilation is apparently adequate, is also a risk in patients with a mediastinal mass. The etiology of these hemodynamic changes is considered to be compression of the great vessels and right atrium. Studies in animals have confirmed that cardiac output decreases in the presence of an anterior mediastinal mass. This appears to be the result of an increase in right ventricular afterload with corresponding right ventricular dysfunction and appears independent of muscle paralysis or spontaneous versus mechanical ventilation. Others have suggested that, with positive pressure ventilation, pulmonary arterial and venous flow might decrease, so that blood volume in a mass with intrathoracic and extrathoracic components might be expected to shift from the intrathoracic to the extrathoracic space. With our patient breathing spon-

References

taneously for the entire anesthetic, perhaps the preservation of normal vascular transit time was the most important issue in minimizing the expansion of the extrathoracic portion of the malformation.

This is the first reported case to our knowledge of a dynamic anterior mediastinal mass causing significant adverse cardiorespiratory changes with induction of anesthesia. Although vascular malformations of the mediastinum are relatively uncommon, comprising only 5% of pediatric mediastinal tumors in one large series, their dynamic nature makes them particularly hazardous for general anesthesia. Advance planning should include consideration for special aspects of anesthetic management of patients with vascular mediastinal masses undergoing scoliosis therapy, such as maintenance of spontaneous ventilation, anticipation of the vascular and histologic consequences of sclerosing agents used in the central circulation, and close cooperation with other members of the operating team.

References