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1998; 89:780–2

Severe Hypercapnia Induced by Acute Dissecting Aortic Aneurysm

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THE complications of acute dissecting aortic aneurysm include external rupture with fatal hemorrhage, aortic regurgitation, occlusion of the coronary arteries or other major branches of the aorta, and cardiac tamponade. In very few cases, the aneurysm may induce compression of the right pulmonary artery.[1–4] In these situations, diagnosis may be difficult because the clinical picture resembles a pulmonary embolism.[1] We report the case of a patient in whom severe hypercapnia developed because of a dissecting aortic aneurysm responsible for a compression of both the right pulmonary artery and the left mainstem bronchus.

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Received from the Hôpital Général, Centre Hospitalier Universitaire (CHU) de Dijon and the Centre Hospitalier Universitaire Pitié-Salpêtrière, Paris VI University, Paris, France. Submitted for publication September 25, 1997. Accepted for publication May 6, 1998.

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Key words: Complication; dissecting aortic aneurysm; shock; ventilatory/perfusion mismatch.

Case Report

A 42-yr-old man was admitted to hospital for acute ischemia of the right lower limb. Ten years previously, this patient underwent aortic valvular replacement for regurgitation. He was treated with acenocoumarol and flecainide. Preanaesthesia clinical assessment confirmed the diagnosis of lower limb ischemia. Cardiac and pulmonary status were normal, and results of electrocardiogram were normal. Prothrombin time was less than 10% of control. Emergency surgery was begun 30 min after admission. Induction of anesthesia was performed using etomidate sufentanil, and muscular relaxation was obtained using atra-

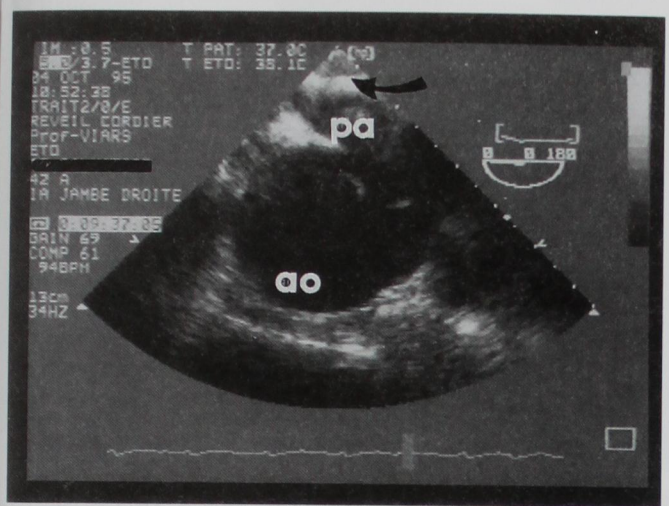

Fig. 1. Zero-degree image of the upper ascending aorta (ao) and the right pulmonary artery (pa), which is squeezed between the dilated aorta (ao) and a mediastinum haematoma (*arrow*).

curium. After tracheal intubation, hypoventilation of the left lung was noted by auscultation. This was not caused by a selective right bronchus intubation, as shown by chest radiography. Oxygen saturation by pulse oximetry (SpO_2) was maintained above 95% with a fractional inspired oxygen tension ($F_{I_{O_2}}$) of 0.50. No major hemodynamic event occurred during surgery, and end-tidal carbon dioxide initially was 24–26 mmHg. The surgeon diagnosed a dissection of the iliac artery, which was responsible for the acute lower limb ischemia, and iliofemoral bypass was performed. At the end of surgery, transesophageal echocardiography was performed, and dissecting aortic aneurysm, involving the thoracic aorta from 5 cm above the aortic valve, was diagnosed. The patient was admitted to the postanesthesia care unit for further examination and because performance of extracorporeal circulation was not available immediately.

Immediate, postoperative blood gas analysis showed severe hypercapnia (114 mmHg during mechanical ventilation with 20 l/min minute ventilation) with a low end-tidal pressure of carbon dioxide level (15 mmHg). The diagnosis of pulmonary embolism was considered. Chest radiography showed only an enlargement of the mediastinum and a small, left pleural effusion. Pulmonary angiography ruled out the diagnosis of pulmonary embolism but showed that right pulmonary artery blood flow was decreased severely and that the pulmonary veins were hardly visible even after a long delay. Another transesophageal echocardiography was performed and showed right ventricle dilatation and right pulmonary artery compression by a large aneurysm of the ascending aorta associated with an intimal dissection (fig. 1). Aortography showed a dissecting aortic aneurysm starting 5 cm above the aortic valve, associated with a 2-cm-diameter false aneurysm on the left side of the ascending aorta (fig. 2). Emergency aortic surgery was proposed again. Nevertheless, hypercapnia increased rapidly with severe acidosis, hypoxia occurred that could not be corrected by inhaled nitric oxide administration, and severe hemodynamic failure occurred and did not respond to increasing doses of catecholamines. The patient died 23 h after admission, before any surgical procedure was initiated.

Necropsy was performed and confirmed the diagnosis of dissecting aortic aneurysm. Hematoma in the Theile sinus was responsible for the right pulmonary artery compression, and the left mainstem bronchus was obstructed by the aneurysm on the left side of the aorta. No pulmonary embolism was found.

Discussion

Compression of the right pulmonary artery by dissecting aortic aneurysm has been reported but remains rare.[1–5] In the reported case, the patient was admitted for acute lower limb ischemia induced by the dissection. Severe hypercapnia associated with low end-tidal pressure of carbon dioxide occurred in the postoperative period, and the diagnosis of massive pulmonary embolism was suspected[5] but not confirmed. Pulmonary angiography showed a major decrease in right pulmonary artery blood flow. The precise cause of left lung hypoventilation remained unknown until necropsy. Tomodensitometry may have been the best evaluation but was not performed because of the unstable hemodynamic status. Fiberoptic bronchoscopy also could have diagnosed the bronchial compression, as previously reported.[6]

Severe hypercapnia associated with a very low end-tidal pressure of carbon dioxide clearly was related to

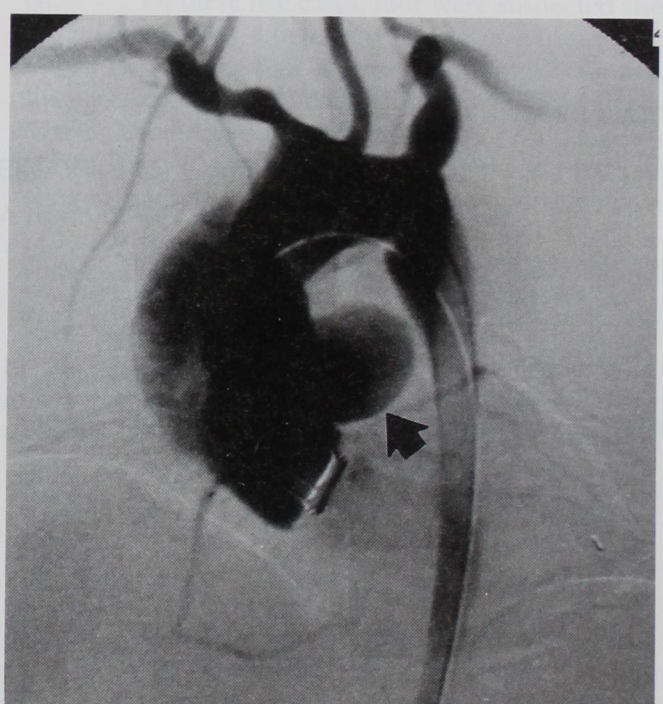

Fig. 2. Aortic angiography that shows the aortic dissecting aneurysm involving the whole aorta and a false aneurysm on the left side of the ascending aorta, which was responsible for left-mainstem bronchus compression (arrow).

major ventilation/perfusion mismatch. In the right lung, the ventilation/perfusion ratio was very high, whereas in the left lung, this ratio markedly was decreased. Moreover, ventilation/perfusion mismatch may have been worsened by hypoxic vasoconstriction. The lack of response to nitric oxide may be explained by the finding that an increase in left lung perfusion was useless because of poor ventilation and an increase in right lung perfusion did not occur because of right pulmonary artery compression. It is doubtful whether any therapeutic intervention, apart from surgery, would have had any beneficial effect on the patient's gas exchange, hemodynamic status, or both. It is clear that the preoperative assessment of this patient with acute limb ischemia was incomplete and that the diagnosis of aortic dissection should have been considered before surgery; thus, transesophageal echocardiography or tomodensitometry may have been indicated preoperatively.[7] Surgery should have been performed very early on, despite the desperate status of the patient. An earlier and more complete understanding of the mechanisms of hypercapnia, acidosis, and hemodynamic failure might have helped to take the right decision earlier. Physicians should be aware of this rare complication to precisely diagnose the cause of severe hypercapnia and to proceed accordingly.

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Anesthesiology
1998; 89:782–4

Early Fat Embolism after Liposuction

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FAT embolism syndrome (FES) is an uncommon and severe complication that occurs mainly in patients with long-bone fractures. This clinical syndrome includes acute respiratory failure, global neurologic dysfunction, and petechial rash following the precipitating event by 12 to 72 h. Nevertheless, one or more of these findings may be absent, making the diagnosis difficult to establish clinically, particularly after a surgical procedure such as liposuction. The authors report a case of isolated acute dyspnea early after liposuction.

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Key words: Bronchoalveolar lavage; computed tomography of the chest; fat embolism syndrome; liposuction.

Case Report

A 29-yr-old woman who was a healthy ballet-dancer without previous significant medical history underwent suction lipectomy of the abdomen, hips, and trocanteric area. The operation was uneventful, removing a total of only 600 ml of fatty tissue. Anesthesia (which lasted 2 h) consisted of fentanyl, midazolam, propofol (loading dose of 2