dose amphotericin B therapy alone.

REFERENCES

11 Alexander WJ, Mowry BW, Cobbs CG, Dismukes WE. Prosthetic valve endocarditis caused by Histoplasma capsulatum. JAMA 1979; 242:1399-1400

An acute aortic dissection with rupture in the descending aorta led to suffusion of blood into the soft tissue of the neck with sufficient pressure to cause not only superior vena cava obstruction, but also tracheal obstruction.

(Chest 1991; 99:256-58)

RA = right atrium; LA = left atrium; PA = pulmonary artery; Th = thrombus.

Aortic dissection results from a tear in the aortic intima which allows blood to enter the aortic media and may then propagate distally and on occasion, proximally. Known risk factors for aortic dissection include hypertension, congenital abnormalities of the aortic valve, coarctation of the aorta, Marfan's syndrome, other connective tissue disorders and iatrogenic causes. The clinical presentation of aortic dissection can be quite variable and is often related to alteration in end-organ blood supply. The most common symptom is chest pain which is present in nearly all cases.1 Other common presenting features include syncope, dyspnea, neurologic impairment, aortic insufficiency, and left pleural effusion. Less frequent findings include vocal cord paralysis, Horner's syndrome, hemoptysis, hematemesis, heart block and, rarely, superior vena cava (SVC) syndrome.1

The following report describes a patient with acute aortic dissection presenting with symptoms of upper airway obstruction.

CASE REPORT

The patient was a 65-year-old caucasian man who had previously been well. On the day of admission, the patient had sudden onset

Aortic Dissection Presenting as Upper Airway Obstruction* 

Peter J. Giannoccaro, M.D.; Jean-Francois Marquis, M.D.; Kuan-Leung Chan, M.D.; Virginia Walley, M.D.; and Rosemary J. Chambers, M.B.

*From the University of Ottawa Heart Institute, Ottawa, Canada.
the neck. Peripheral pulses were all palpable and there was no radio-femoral delay. Soon after his arrival visible progression of the neck swelling lead to increasing respiratory distress with inspiratory stridor. He required intubation for airway control. His hypotension was improved with volume loading.

A portable chest x-ray examination before intubation showed widening of the aortic arch and superior mediastinum along with a large right apical density (Fig 2). The ECG showed sinus tachycardia, but was otherwise normal. A standard precordial 2D echocardiogram revealed mild concentric left ventricular hypertrophy, the aortic valve was normal, and no aortic insufficiency was detected. The aortic root was mildly dilated but the dramatic finding was compression of the right atrium and SVC by a large extracardiac mass. The aortic arch was not well visualized. A transesophageal echocardiogram was performed to assess the aortic arch and descending aorta. Compression of the right atrium and SVC was again demonstrated (Fig 3). In addition, aortic dissection involving the aortic arch and descending aorta was confirmed. There was massive hematoma surrounding the aortic arch consistent with rupture. A pseudoaneurysm was identified at the descending aorta just distal to the arch. Subsequently, aortography was performed which revealed a large mediastinal hematoma, but no tear was seen.

The patient underwent femoro-femoral bypass with hypothermic circulatory arrest. The chest was opened through a median sternotomy where the ascending aorta and arch were explored. There was no evidence of a tear in this area. The initial incision was modified and extended to allow exploration of the descending aorta. This revealed a dissection and external rupture just distal to the left subclavian take-off. The involved section of aorta was removed and a Dacron interposition graft was used to finish the repair. The patient had a prolonged “pump run” and there was considerable bleeding. After vigorous attempts were made to wean him off cardiopulmonary bypass, and despite inotropic support, the patient died.

At autopsy, evidence of mild essential hypertension and aortic medial degenerative changes were seen. The entry and rupture sites of the dissection had been excised at surgery, but the retrograde tracking of the dissection ending in the proximal aorta could still be seen, along with thrombus in the false lumen. A large hematoma in the mediastinal soft tissues could be seen and this extended into the right retropleural, right pleuro-apical and right neck soft tissues. The surgical sites were intact.

**DISCUSSION**

There have been no reported cases in the literature of upper airway obstruction due to dissection of the descending aorta. SVC obstruction associated with aortic dissection has been described in several case reports. The mechanism of venous obstruction is usually compression by a dilated or aneurysmal ascending aorta. However, hematomas can compress the SVC or a fistulous communication between the aorta and the SVC can occur. All these case reports describe dissections involving the ascending or proximal aorta. This case is unique in that the patient had a dissection of the descending aorta. Rupture or leak in this anatomic site characteristically leads to left pleural effusion. In this case, the blood travelled through the soft tissue planes into the neck with sufficient pressure to cause compression of both the SVC and trachea. It may be that old pleural disease led to scarring which prevented rupture into the left pleural space. Blood exiting the aorta will follow the path of least resistance, and in this case may have led to a large mediastinal hematoma which subsequently entered the right apical region and encroached on the trachea leading to

---

**Figure 2.** Portable AP chest x-ray film shows widened superior mediastinum and large right apical pleural density.

**Figure 3.** Transesophageal echocardiogram at the level of the aortic root shows the aortic valve in short axis. A large mass compressed the right atrium near the entrance of the superior vena cava.
Noncardiogenic Pulmonary Edema Complicating Massive Diltiazem Overdose*

Vernon H. Humbert, Jr., M.D., F.C.C.P.;† Nancy J. Munn, M.D.;‡ and Randall F. Hawkins, M.D.

Non-cardiogenic pulmonary edema has not been previously described in calcium channel blocker overdose. We describe a case of non-cardiogenic pulmonary edema occurring during the course of therapy for massive diltiazem overdose in a young patient with anorexia nervosa. Review of the current literature suggests that major and minor pulmonary complications occur with some frequency in the setting of calcium channel blocker overdose although their exact incidence remains unclear. (Chest 1991; 99:258-60)

Although mild arterial desaturation is common after calcium channel blocker overdose, no well-documented case of non-cardiogenic pulmonary edema in this setting has previously been reported. Prior reports of pulmonary edema occurring after calcium channel blocker use or misuse have generally ascribed the condition to negative inotropy related to the drug or to fluid resuscitation during overdose-induced hypotension.

CASE REPORT

A 30-year-old nurse was hospitalized after having been found apathetic and unable to ambulate. She was known to be taking diltiazem for migraine headaches and had a long history of stress disorder and depression and had previously been evaluated for anorexia nervosa.

On examination, she was lethargic but responsive to noxious stimuli. The blood pressure varied from 58/22 mm Hg to 90/60 mm Hg and the pulse from 35 to 45 per minute. The respiratory rate was 18. She had warm skin and full pulses distally.

The initial chest radiograph showed a very small heart, no infiltration, and a paucity of vascular markings. The electrocardiogram showed atrial inactivity with a slow ventricular escape rhythm. Arterial blood gases on a 100 percent rebreathing mask revealed a blood pH of 7.23, Pco2 of 27 mm Hg, and a PaO2 of 305 mm Hg. The initial serum albumin level was normal.

Initial therapy with atropine, isoproterenol and calcium gluconate raised the heart rate to 45 to 50 per minute; sinus bradycardia alternated with ventricular escape rhythm. Infusions of saline solution (totaling 4L during the first 24 hours) and dopamine gradually raised the blood pressure to a consistent 90/60 mm Hg over several hours. Urine flow remained copious.

After 24 hours of steady improvement, the patient developed shortness of breath and suffered respiratory arrest. Endotracheal intubation and mechanical ventilation were promptly instituted. Repeat chest radiograph showed uniformly diffuse pulmonary infiltrates without change in heart size (Fig 1). Mechanical ventilation with 100 percent oxygen and positive end-expiratory pressure of 15 cm H2O was needed to elevate the Fio2 to 67 mm Hg.

Pulmonary artery pressure of 32/12 mm Hg and a pulmonary capillary wedged pressure of 9 mm Hg. Cardiac output by thermodilution was 6.50 L/min (5.07 L/min/m²). Although extensive bacterial cultures later proved negative, she developed a fever of 39.3°-39.6°C 36 hours after intubation. Therapy with broad

*From the Department of Medicine, Divisions of Cardiology and Pulmonary Medicine, Marshall University School of Medicine, Huntington, WVA.
†Supported in part by the Marshall University Foundation.
‡Presented in part at the 1989 Scientific Meeting, Pennsylvania and West Virginia Regions, American College of Physicians, Pittsburgh, October 6, 1989.

Reprint requests: Dr. Munn, 1801 Sixth Avenue, Huntington, WVA 25755-9410

Figure 1. Chest radiograph shortly after endotracheal intubation showing diffuse, uniform pulmonary infiltration and a small cardiac silhouette compatible with non-cardiogenic pulmonary edema.