the drains gradually were withdrawn and removed on day 10 after the operation. Antibiotic treatment was discontinued after 2 weeks. At that moment, the leukocyte count had dropped to 5.2×10⁹/L. Cultures obtained at the time of bronchoscopy and the operation were sterile. Many Gram-negative rods with diverse morphologic characteristics were demonstrated. The fistula in the bronchus healed spontaneously. A postoperative bronchoscopy on day 18 revealed a normal mucous membrane at the site of the fistula, and there was no sign of stenosis or granulation. The patient was discharged in good health on day 27 after the operation. There were no late complications.

DISCUSSION

This case report illustrates that a wisdom tooth extraction may be associated with serious complications. Between 1960 and 1991, 48 patients with descending necrotizing mediastinitis resulting from an oropharyngeal infection, were reported in the English-language literature. For 29 of these patients, the mediastinitis was odontogenic. A fistula to a bronchus as a complication of mediastinitis has not yet been reported, to our knowledge. Our patient presented with an alveolitis extending from the submandibular space to the pharyngomaxillary space and spatiun retropharyngeum and from there to the mediastinum. A special feature of this case is the fistula to the left main bronchus providing spontaneous drainage of the purulent mediastinitis. This fistula formation more than likely contributed to the successful outcome.

Deep infections of the neck should make one wary of the development of a descending necrotizing mediastinitis, with or without empyema of the pleura or the pericardium. The computed tomographic scan plays an important role in the early recognition of deep neck infections and mediastinitis. Therapy consists of, on the one hand, the administration of antibiotics which are aimed at the most commonly occurring microorganisms. Since these mostly consist of a mixed aerobic and anaerobic flora, a combination is given of penicillin, metronidazole, and an aminoglycoside. On the other hand, therapy consists of surgical drainage. A number of authors favor drainage of the mediastinum by way of a thoracotomy if the infection has spread below the level of the fourth thoracic vertebra. Puncture and incision, guided by a computed tomographic scan, is recommended by others. Thanks to the fistula to the main bronchus, our patient only needed an incision in the fossa jugularis and manual exploration of the mediastinum in order to place the drain. Recovery was uneventful.

Despite aggressive antibiotic and surgical therapy, the mortality rate for descending necrotizing mediastinitis remains high. Clear guidelines regarding risk factors and possible preventative measures are still required. Early recognition remains of vital importance.

ACKNOWLEDGMENT: The authors are grateful to Dr. J. J. Bredée, cardiopulmonary surgeon, and Dr. J. Bath, general surgeon, for their contribution to this publication.

REFERENCES

5 De Marie S, Tjon a Tham RTO, van der Meij AGL, Meerdink G, Van Furth R, Van Der Meer JWM. Clinical infections and nonsurgical treatment of parapharyngeal space infections complicating throat infection. Rev Infect Dis 1989; 11:975-82

Rupture of Thoracic Aorta Caused by Penetrating Aortic Ulcer*

Yuichi Ando, M.D.; Hironobu Minami, M.D.; Hideyuki Muramoto, M.D.; Michihiko Narita, M.D.; and Shuzo Sakat, M.D., F.C.C.P.

We present the findings in a 57-year-old man with a rupture of the thoracic aorta that originated in a penetrating atherosclerotic aortic ulcer. It formed a large hematoma that clinically mimicked a true saccular thoracic aneurysm. The possibility of penetrating aortic ulcer should be considered in the differential diagnosis of aortic aneurysm.

(Chest 1994; 106:624-26)

The occurrence of aortic rupture in the absence of trauma and without evidence of aneurysm or dissection is quite rare. We present the findings from a patient in whom a penetrating atherosclerotic aortic ulcer produced a complete aortic rupture to form a large hematoma that mimicked a true saccular thoracic aneurysm.

CASE REPORT

A 57-year-old man was transferred to our hospital with severe chest pain of sudden onset. There was no history of trauma or of treatment of hypertension.

On admission, the severity of the chest pain had lessened. The blood pressure was 130/80 mm Hg, without pulse deficits. A chest roentgenogram showed an enlarged aortic arch and left-sided pleural effusion. Diagnostic thoracentesis revealed a yellowish exudative effusion. Echocardiography showed mild pericardial effusion, with no signs of tamponade or valvular insufficiency. Computed tomography (CT) of the chest revealed an eccentric mass measuring 6 cm that arose from the left of the arch. A dynamic CT image enhanced by contrast medium (Fig 1) showed a saccular-type aneurysm with a thick wall of thrombus. Because there was a delay in filling the aneurysm in the contrast medium, we considered the orifice of the aneurysm to be unusually narrow. No false lumen or intimal flap was observed. Arte- riography was not performed. The patient suffered from dia- betes mellitus (glucose level, 183 mg/dl) and mild hepatic dysfunc-

*From the Departments of Internal Medicine (Dr. Ando, Minami, and Sakai) and Radiology (Dr. Muramoto), the Japa- nese Red Cross Nagoya First Hospital, and the First Department of Pathology, Nagoya University School of Medicine (Dr. Nar- ita), Nagoya, Japan. Reprint requests: Dr. Ando, Japanese Red Cross Nagoya First Hospital, 3-35 Michishita-cho, Nakamura-ku, Nagoya 455, Ja- pan.
tion (serum aspartate aminotransferase, 97 IU/L; serum alanine aminotransferase, 65 IU/L). He was positive for anti-hepatitis C virus antibody.

An impending rupture of a true thoracic aneurysm was diagnosed. Because of the patient's complex medical status, he was not considered a candidate for surgery. Conservative treatment with antihypertensive agents was begun. Despite good control of blood pressure (the systolic pressure was about 100 mm Hg), the patient experienced mild back pain and chest discomfort on the 13th day of hospitalization. A chest roentgenogram and magnetic resonance (MR) imaging (Fig 2) then showed that the size of the mass on the left aortic arch had increased. The patient died on the 24th day of hospitalization.

At autopsy, a defect 8 mm in diameter was found at the left of the aortic arch, surrounded by atheromatous plaques. There was no aneurysmal dilatation of the aorta and no evidence of a false lumen. An 8-cm saccular hematoma surrounded by fragile fibrous tissue impinged on the left upper pulmonary parenchyma and was contiguous to the defect. Microscopic sections (Fig 3) revealed a complete transmural rupture, with disruption of the adventitia. We found no medial disease, inflammation, or other pathologic changes that would explain the rupture.

**DISCUSSION**

Aortic rupture occurred in our patient via a small aortic defect and produced a hematoma that mimicked a true saccular aneurysm. The only pathologic finding that would explain the origin of the rupture was severe atherosclerosis, because there was no evidence of a true aortic aneurysm or dissection.

Atherosclerosis had not been regarded as a cause of aortic rupture until Stanson et al.\(^5\) recently suggested penetrating atherosclerotic aortic ulcer as a new clinical entity. This lesion is characterized by an ulcerating atherosclerotic lesion that penetrates the intimal elastic lamina and extends into the media, with the formation of an intramural hematoma. The lesion can also deeply penetrate the aortic wall but rarely leads to complete transmural rupture.\(^5,6\) The diagnosis of this disease was previously confused with that of classic aortic dissection or rupturing aneurysm. Although the characteristic CT images of penetrating aortic ulcer are not fully described,\(^5,6\) they usually include focal ulceration, intramural hematoma, calcified displaced intima, and a thickening or enhancement of the aortic wall, rarely with a saccular false aneurysm.\(^5\)

Before the publication by Stanson et al.\(^5\) a relationship between atherosclerosis and aortic rupture without evidence of aneurysm or dissection was described in two pa-
tients.\textsuperscript{1,2} Rodriguez and Rivera reported the findings in a 58-year-old woman with a spontaneous rupture of the thoracic aorta. A defect 15 mm in diameter in the aorta distal to the left subclavian artery was found at autopsy. The defect was surrounded by atheromatous plaques, from which a large clot protruded and displaced the pulmonary parenchyma. Castleman and McNeely\textsuperscript{2} described a 62-year-old man with a rupture of the ascending aorta that clinically mimicked dissecting aneurysm or myocardial infarction. An external tear (1 by 0.5 cm) below the arch produced the hemopericardium and hemothorax. A section taken from the perforation revealed severe atherosclerosis. Since there was no evidence of aortic aneurysm, dissection, or other changes that would clearly explain the rupture in either of the previous cases, a penetrating atherosclerotic ulcer appeared to be the cause.\textsuperscript{1,2}

We consider that the complete aortic rupture in our patient was consistent with a diagnosis of penetrating aortic ulcer (specifically, a rare transmural rupture of the aorta). Ulceration was confirmed both radiologically and pathologically as a defect of the aorta. The radiologic finding of a relatively narrow orifice compared with a large lesion may be helpful in differentiating a transmural rupture with the hematoma from a true saccular thoracic aneurysm. While this condition is less common, it produces significant clinical symptoms.\textsuperscript{5,6} In the differential diagnosis of aortic aneurysm, especially of the saccular type, as in the case presented, one should therefore consider the possibility of transmural rupture caused by the penetrating aortic ulcer.

REFERENCES

Reversal of Refractory Hypotension in Septic Shock by Inhibitor of Nitric Oxide Synthase*

Pyng Jing Lin, M.D.; Chau-Hsiung Chang, M.D., F.C.C.P.; and Jen-Ping Chang, M.D., F.C.C.P.

Septic shock is a life-threatening condition that results from exposure to bacterial endotoxin. It is mediated by the release of cytokines. Some of these cytokines cause the release of vasoactive substances. We report the case of a 62-year-old male patient who received redor operation for replacement of the degenerative porcine aortic and mitral prostheses. High cardiac output shock developed on the seventh postoperative day with severe metabolic acidosis and oliguria. Systemic vascular resistance and mean arterial pressure elevated within 5 min and stabilized 60 min after the start of a single dose of intravenous administration of N\textsuperscript{6}-monomethyl-L-arginine (50 mg), a potent and selective inhibitor of nitric oxide synthesis. These findings indicate that nitric oxide overproduction is an important contributor to refractory hypotension in high cardiac output septic shock. Our findings suggested the utilization of nitric oxide synthase inhibitor in the treatment of septic shock in humans.

(Chest 1994; 106:626-29)

L-NMMA=N\textsuperscript{6}-monomethyl-L-arginine; NO=nitric oxide

Septic shock, a life-threatening complication of bacterial infection, is characterized by cardiovascular collapse and multiple metabolic derangements that are due largely to a bacterial endotoxin.\textsuperscript{1,6} Nitrite accumulates when cultured mouse endothelial cells are exposed to immunomodulators and endotoxin.\textsuperscript{8} This nitrite arises from the L-arginine-dependent nitric oxide (NO) synthetic pathway. Since NO is a potent endothelium-derived relaxing factor, these studies suggested that overproduction of NO might account for the cardiovascular changes associated with endotoxin or cytokine administration. Indeed, animal studies clearly demonstrated that N\textsuperscript{6}-monomethyl-L-arginine (L-NMMA), a selective inhibitor of NO synthase,\textsuperscript{7} could completely reverse the hypotensive response elicited by tumor necrosis factor\textsuperscript{8} or endotoxin.\textsuperscript{9} However, there were only a few reports concerning the usage of L-NMMA to reverse the NO effect in septic shock in humans.\textsuperscript{10,11} In the present report, we treated a patient having parapneumonic empyema after double-valve replacement for degenerative porcine bioprostheses with L-NMMA during septic shock.

CASE REPORT
This 62-year-old male patient was admitted to the hospital because of congestive heart failure. Echocardiography showed

*From the Division of Thoracic and Cardiovascular Surgery, Chang Gung Memorial Hospital, Chang Gung Medical College, Taipei, Taiwan, Republic of China.

Reprint requests: Dr. Lin, Chang Gung Memorial Hospital, 199 Tun-Hwa N Road, Taipei, Taiwan 10591 ROC.