resulting in the swollen, painful limb that has the characteristic bluish discoloration. I emphasize that these symptoms are the result of severe venous hypertension. A minority of patients will have thrombosis of their venules, and it is these patients who progress to venous gangrene. Whether patients with thrombosed venules progressing to venous gangrene can be treated successfully is doubtful.

Most of the patients with clinical presentation of phlegmasia cerulea dolens will have iliofemoral deep venous thrombosis, which in most is reversible, with a treatment strategy designed to remove the thrombus. The patients with venous gangrene have venular thrombosis, and in our experience this has been resistant to thrombolysis and anticoagulants, resulting in tissue loss. Interestingly, a number of these patients have had extensive infrainguinal thrombosis, but their iliac veins were patent. This represents quite a different form of disease. Also, all of the patients with venous gangrene in our experience had an associated malignancy.

In summary, the definition of phlegmasia cerulea dolens is the clinical presentation of the patient and is not defined by venular thrombosis. I remain skeptical that intraarterial catheter-directed thrombolysis (or intrathrombus thrombolysis) will be effective in these patients with small-vessel thrombosis. However, that does not diminish the benefit observed in treating phlegmasia cerulea dolens caused by deep venous thrombosis with a strategy designed to eliminate the clot and restore unobstructed ipsilateral venous drainage.

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A case report of inflammatory aneurysm of the thoracic aorta

To the Editors:

Although the incidence of "inflammatory aneurysm" of the abdominal aorta has been reported to be 10% to 20% of all abdominal aortic aneurysms, inflammatory process with a markedly thickened wall and prominent periaortic fibrosis has seldom been reported in the thoracic aorta. In this report we describe a rare case of inflammatory aneurysm of the thoracic aorta.

CASE REPORT

A 50-year-old man presented with thoracoabdominal aneurysm of chronic DeBakey type I dissection. He had received the diagnosis of a chronic type I dissection, aortic and mitral regurgitation, and infrarenal aortic aneurysm at the age of 48 years. He underwent mitral valve repair and replacement of the aortic valve, ascending aorta, aortic arch, and arch vessels with a composite graft of a bileaflet mechanical prosthesis (Carbomedicus 23A) and a woven Dacron graft (Intervascular 26 mm). One year later computed tomography demonstrated an abdominal aneurysm that was 55 mm in diameter and had marked thickening of the periaortic tissue and bilateral hydronephrosis. After he received predonisolone therapy, he underwent a straight Dacron (Gelseal 20 mm) grafting of the abdominal aorta. The pathologic specimen of the aneurysmal wall demonstrated typical inflammatory cell infiltration. On March 16, 1994, replacement of the thoracoabdominal aneurysm was performed. Preoperative computed tomography (Fig. 1) showed that a large false lumen existed in the entire descending aorta and in the suprarenal portion of the abdominal aorta. The maximal size of the aneurysm was 65 mm at the distal arch portion, and severe thickening of periaortic tissue was present contiguous from the abdominal aorta to the lower and middle portion of the descending thoracic aorta. After spiral incision and difficult dissection of severe periaortic fibrosis at the peridiaphragmatic portion of the aorta were performed, cardiopulmonary bypass was instituted via the femoral artery, the femoral vein, and the pulmonary artery. At a nasopharyngeal temperature of 18° C the aneurysmal wall was incised, and a woven Dacron graft (Hemashield 24 mm) was sutured to the previous arch graft during 30 minutes of cerebral circulatory arrest and retrograde cerebral perfusion by our method.[1] The aneurysmal wall of the distal portion of the descending aorta was glistening, whitish, solid, and markedly thickened. Histologic evaluation of the lower portion of the descending aorta (Fig. 2) demonstrated a typical inflammatory reaction: the wall was 8 mm in maximal thickness and had deposits of focally calcified atheroma containing cholesterol clefts in thickened intima, inflamed attenuated fibrotic media in which the muscle and elastic tissue were severely damaged, and the adventitial and periadventitial fibrosis. Severe atherosclerotic change of the media and intima was seen. The postoperative course was uneventful, and the patient was discharged on April 28, 1994.

Discussion

Since Walker et al. first described "inflammatory aneurysm,"[2] many investigators[3] have reported the incidence of this particular type of aortic aneurysm to be 10% to 20% of all abdominal aortic aneurysms. The histologic features of inflammatory aortic aneurysms differ from those of retroperitoneal fibrosis only in that the aorta is dilated, and the unifying term "chronic periaortitis" has been proposed for these conditions. However, the thoracic aorta has been considered relatively free from the inflammatory infiltration, and to our knowledge only one report in the English literature has described an inflammatory thoracic aneurysm.[4] Crawford et al.[4] reported three cases of inflammatory aneurysm that extended from the descending aorta to the infrarenal aorta; these cases were successfully treated by thoracoabdominal graft replacement.

The outer wall of chronic aortic dissection was known to cause chronic periaortic inflammation resulting from reaction against the dissection. However, in this case the

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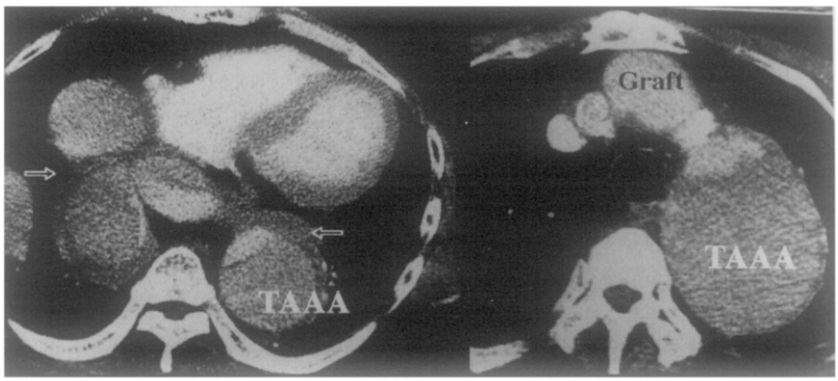

Fig. 1. Computed tomography before last operation. *Right,* Large thoracoabdominal aortic aneurysm *(TAAA)* occupied left hemithorax. *Graft,* Arch graft of previous procedure. *Left,* At peridiaphragmatic portion, tortuous TAAA traversed from left pleural cavity to right. Marked periaortic fibrosis was noted *(white arrows).*

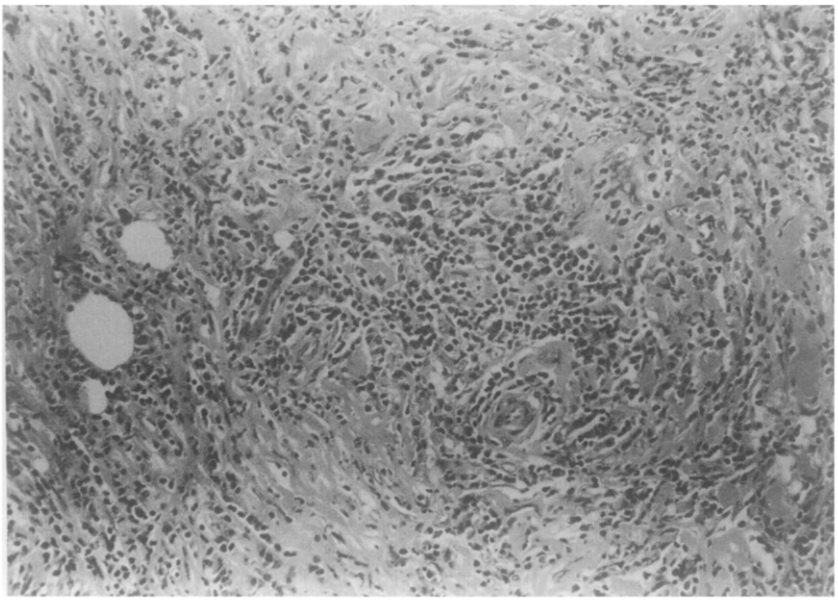

Fig. 2. Histologic evaluation of lower portion of descending aorta demonstrated typical inflammatory reaction: thickened intima with deposits of focally calcified atheroma containing cholesterol clefts, attenuated fibrotic media in which muscle and elastic tissue were severely damaged, and adventitial and periadventitial fibrosis.

involved portion of the aorta, which was the peridiaphragmatic part, showed the characteristic inflammatory reaction of a markedly thickened wall with prominent periaortic fibrosis,[5] which was quite different from the other portion of the dissection. Although a definitive etiology of inflammatory aneurysm remains unknown, the periaortic inflammatory reaction usually subsides spontaneously after graft replacement. One of the unique features in this case was that the inflammatory infiltrate remained or recurred and extended to the thoracoabdominal aorta after the abdominal aortic aneurysm was grafted.

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24/41/63749

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