Chronic Pseudoaneurysm and Coarctation of the Aorta
A Rare Delayed Complication of Trauma

We report an unusual case of pseudoaneurysm and coarctation of the descending thoracic aorta after trauma. The coarctation of aorta resulted in hypertension, severe left ventricular dysfunction, and symptoms of congestive heart failure. Surgical bypass resulted in control of blood pressure and improvement of heart failure symptoms. The mechanism of aortic injury leading to the development of this rare combination is discussed. (Tex Heart Inst J 2006;33:368-70)

Aortic rupture secondary to trauma is lethal; only 1% to 2% of patients survive long enough without surgery to present with pseudoaneurysm. Very rarely, patients develop acquired coarctation following trauma. Only a few cases of this rare combination have been described in the literature. We report the case of a patient who presented with congestive heart failure and was subsequently found to have both pseudoaneurysm and coarctation of the aorta at the aortic isthmus secondary to trauma sustained 30 years earlier.

Case Report
In January 2005, a 48-year-old white woman with newly diagnosed congestive heart failure was referred from an outside hospital for cardiac catheterization after an attempt to perform retrograde catheterization through a femoral approach had failed due to inability to cross the distal end of the aortic arch with the guidewire. Her medical history included tobacco use and hypertension. On physical examination, her blood pressure was 160/80 mmHg in both arms and 90/40 mmHg in the legs. Her pulse was regular, with a radiofemoral delay. A left ventricular 3rd heart sound was heard at the apex. There was no murmur over the precordium or chest. No bruits were heard over the abdomen or back. Examination of the abdomen and of the respiratory and neurological systems was unremarkable. Electrocardiography revealed normal sinus rhythm, with T wave inversion in leads I, AVL, and V4 through V6. Laboratory investigations, including complete blood count, erythrocyte sedimentation rate, comprehensive metabolic profile, lipid profile, and urinalysis, yielded unexceptional results. The patient’s chest radiograph (Fig. 1) revealed a prominent aortic knuckle with calcification. Two-dimensional echocardiography showed dilatation of all 4 chambers with severe left ventricular dysfunction (left ventricular ejection fraction, 0.15) and pulmonary artery hypertension (estimated right ventricular systolic pressure, 60 mmHg). The descending aorta and the arch could not be seen due to a poor echocardiographic window.

We at first surmised that the heart failure was secondary to unrecognized congenital coarctation of the aorta. Contrast-enhanced 3-dimensional magnetic resonance angiography confirmed the clinical diagnosis, showing stenosis, a small pseudoaneurysm, and a chronic dissection of the aorta at the isthmus (Fig. 2). Subsequent catheterization performed from the right arm confirmed the magnetic resonance findings (Fig. 3). Collateral arteries, which are characteristic of congenital coarctation, were absent. Coronary angiography did not show evidence of luminal narrowing. Additional history revealed that the patient, in a major motor vehicle accident 30 years earlier, had sustained left-sided hemothorax, which had required chest-tube placement and multiple blood transfusions.
The patient was referred to surgery, because the stenotic segment could not be crossed with the guidewire. During the operation, a small calcified aneurysm was seen at the aortic isthmus, together with a 50-mmHg gradient between the ascending and descending aorta. The stenosed segment was successfully bypassed by means of a 14-mm Dacron (Hemashield®; Boston Scientific, Inc.; Oakland, NJ) graft between the left subclavian artery and the descending thoracic aorta (Fig. 4). After bypass, the gradient between the ascending and descending aorta decreased from 50 to 5 mmHg. Repeat transthoracic echocardiography done 1 month after surgery showed a substantial improvement in the patient's left ventricular ejection fraction, from 0.15 to 0.30.

**Discussion**

Traumatic aortic injury is a major cause of fatality in high-speed deceleration injuries (for example, high-speed motor vehicle and motorcycle collisions, pedestrian–motor vehicle collisions, and falls). Traumatic aortic injury commonly involves the isthmus, because it is a transition point between relatively fixed and mobile portions of the aorta. Only 10% to 20% of the patients who suffer traumatic aortic injury survive the initial injury and reach the emergency department. If left untreated, 30% of the initial survivors die within 6 hours, 40% to 50% die within 24 hours, and 90% die within 4 months. A chronic pseudoaneurysm develops in 1% to 2% of the patients whose injury is not diagnosed, and from this small pool of survivors, very few develop a coarctation.3,4,6,7

![Fig. 1 Posteroanterior chest radiograph shows an enlarged heart, mild pulmonary venous congestion, and blunting of the right costophrenic angle by a pleural effusion. In addition, calcification of the aortic knuckle (arrow) is noted.](image1)

![Fig. 2 Contrast-enhanced (gadolinium) 3-dimensional magnetic resonance angiograms of the thoracic aorta. A] There is a focal dilatation of the arch distal to the subclavian artery (arrows) and stenosis of the aorta (arrowhead). B] A coronal section demonstrates dilatation of the thoracic aorta with a filling defect suggestive of thrombus (arrow).](image2)

Chronic aortic pseudoaneurysms may remain asymptomatic or can gradually enlarge and cause dysphagia, hoarseness, or cough, and can present with rupture at any time.6,7 The gold standard for the diagnosis of pseudoaneurysm is angiography; however, newer noninvasive imaging techniques, such as magnetic resonance angiography, computed tomography with 3-dimensional reconstruction, and transesophageal echocardiography,10 are currently used more frequently. They provide information not only about the lumen but also about the vascular wall.

Surgical treatments of chronic pseudoaneurysms include direct aortic replacement (usually with the aid of left-heart bypass) or extra-anatomic bypass (ascending aorta-to-descending aorta; left subclavian artery-to-
descending aorta).8,9,11 Recently, favorable results (in comparison with those of surgery) have been described for endovascular graft placement. However, in our patient, heavy calcification and lack of guidewire passage across the lesion prevented such an approach. During surgery, the surgeon saw that the presence of extensive scarring tissue and adhesions to surrounding structures would render excision of the pseudoaneurysm technically difficult. Because the lesion was chronic, small, and heavily calcified, the surgeon concluded that the lesion was stable and decided not to attempt excision. Instead, he bypassed the lesion by placing a 14-mm Dacron graft between the left subclavian artery and the descending thoracic aorta (Fig. 4).

Our patient’s history of a major motor vehicle accident, with multiple rib fractures and left-sided hemothorax, clearly suggests that she sustained traumatic aortic rupture and subsequent pseudoaneurysm and coarctation as a result of this trauma. The acquired coarctation led to an increase in cardiac afterload, resulting in hypertension and ultimately in congestive heart failure.

The examining physician should be aware of the remote possibility of pseudoaneurysm and coarctation as a delayed complication of trauma; but more especially, when a patient presents with a major trauma accompanied by rib fractures and hemothorax, the physician should recall the much greater likelihood of aortic rupture.

References