

Acute Paraplegia during Weightlifting:

An Unusual Vascular Catastrophe

Rushi V. Parikh, MD
Farzad Alemi, MD
Daniel R. Kievlan, MD
Thienkhai H. Vu, MD
Christopher F. Barnett, MD,
MPH

While weightlifting, a 58-year-old man developed sudden-onset, tearing chest pain, rapidly followed by the inability to move his legs. His medical history included the repair of a traumatic descending aortic laceration 30 years earlier. Physical examination was notable for unequal blood pressures (240/97 mmHg in the left arm and 164/87 mmHg in the right), absent femoral pulses, mottled lower extremities, and loss of motor and sensory function below the T6 spinal level.

Computed tomograms were obtained. One unexpectedly showed occlusion of the aorta distal to the left subclavian artery (Fig. 1). A chronic dissection was seen anterior to the thrombosed aorta at the level of the ligamentum arteriosum (Fig. 2). Distally, crescentic calcification was consistent with luminal displacement of intimal calcification secondary to an acute aortic dissection (Fig. 3). Otherwise, the proximal ascending aorta and distal aorta appeared to be free of disease. No collateral vessels bridged the occluded segment of the aorta, suggesting an absence of preexisting luminal obstruction. Despite emergent axillofemoral bypass, the patient's neurologic deficits persisted, and he remained paraplegic. Subsequent magnetic resonance imaging confirmed infarction of the spinal cord beginning at T6 (Fig. 4).

Comment

Section Editor:

Raymond F. Stainback, MD,
Department of Adult
Cardiology, Texas Heart
Institute, 6624 Fannin St.,
Suite 2480, Houston, TX
77030

From: Departments of
Medicine (Dr. Parikh),
Surgery (Dr. Alemi), and
Emergency Medicine (Dr.
Kievlan), University of Cali-
fornia, San Francisco, San
Francisco, California 94131;
and Departments of Radiol-
ogy (Dr. Vu) and Medicine
(Dr. Barnett), San Francisco
General Hospital, University
of California, San Francisco,
California 94110

Address for reprints:

Christopher F. Barnett, MD,
MPH, Rm. 5G1 Cardiology
SFGH, 1001 Potrero Ave.,
San Francisco, CA 94110

E-mail:

barnettc@medsfgh.ucsf.edu

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Severe chronic atherosclerosis can lead to aortic occlusion in aortoiliac occlusive disease (Leriche syndrome); however, well-formed collateral vessels prevent the development of ischemic complications. Acute aortic occlusion is a rare but devastating event associated with a mortality rate of 50% to 80%.¹ Nearly all reported cases have involved thrombotic or embolic occlusion of the abdominal aorta with predisposing factors such as severe atherosclerosis, left ventricular systolic dysfunction, or a prothrombotic



Fig. 1 Computed tomographic angiogram (sagittal view) shows occlusion of the thoracic aorta. Thrombus occludes the proximal descending aorta distal to the left subclavian artery (arrow). The proximal ascending and distal descending aortic segments appear to be normal. Contrast is present distal to the obstruction; quantitative measures reveal reduced attenuation in comparison with the ascending aorta, suggesting some communication even though no channel can be seen. No collateral vessels are visible.



Fig. 2 Computed tomographic angiogram (axial view) reveals diffuse calcification of the descending thoracic aorta above the main pulmonary artery. Anterior to the descending aorta at the level of the ligamentum arteriosum is a focal outpouching with a heavily calcified wall, consistent with a chronic dissection (asterisk). Thrombus fills the lumina of the aorta and chronic dissection.



Fig. 3 Computed tomographic angiogram (axial view of the descending thoracic aorta at the level of the pulmonary artery) reveals crescentic calcification at the distal end of the aortic occlusion, most consistent with intimal calcification that has been displaced into the aortic lumen as the result of an acute aortic dissection (arrow).

state.^{1,2} Only a few case reports have described acute thrombosis of the thoracic aorta presenting as paraplegia in the presence of a complex or chronic dissection.³

The imaging findings in our patient suggest that hemodynamic stresses—induced by weightlifting and focal aortic disease after prior aortic repair—led to an acute aortic dissection with subsequent thrombosis and occlusion of the descending aorta. Current guidelines

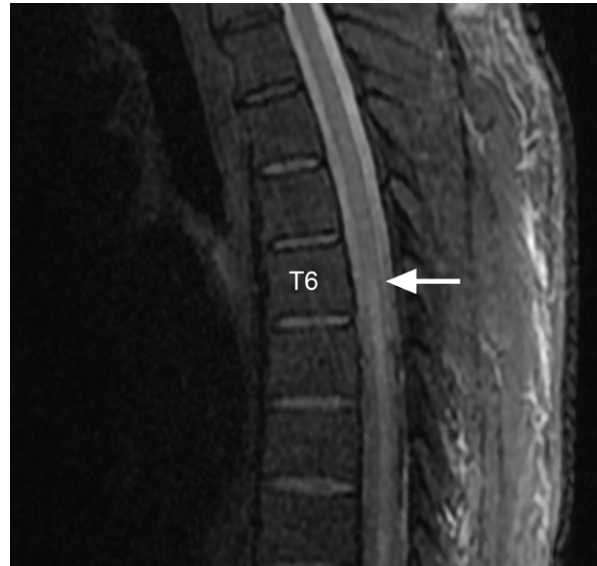


Fig. 4 Magnetic resonance image (sagittal view) of the thoracolumbar spine reveals an abnormal T2 signal within the spinal cord beginning at T6, indicating infarction (arrow).

recommend frequent imaging during the first year after thoracic aortic repair and then annually if the condition is stable; however, there are no specific imaging recommendations for remote dissections.⁴ Strong suspicion and rapid imaging by means of vascular-appropriate methods are necessary to detect vascular catastrophes in patients who present with acute neurologic abnormalities.

References

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