A 35-year-old woman was admitted to our institute with a 5-year history of fatigue and low-grade fever. Six months before admission, she had pain in the neck and in the infrascapular region. On admission, the patient’s heart rate was 80 bpm, her temperature was 37.3°C, and her blood pressure was 130/70 mm Hg in the right arm and 110/70 mm Hg in the left arm. Examination of the neck revealed no jugular venous distention but disclosed bilateral carotid murmurs. Cardiac examination revealed a regular rate and rhythm with a 3/6 holosystolic murmur audible at the left sternal border. Lung fields were normal. Abdominal examination findings were normal, except for the presence of a palpable spleen and an abdominal bruit at the umbilical left lumbar region. Laboratory examination revealed hemoglobin, 8.5 g/dL (reference ranges, 12.0 to 15.5 g/dL); leukocytes, 21.7×10⁹/L (3.5 to 10.5×10⁹/L); platelets, 460×10⁹/L (150 to 450×10⁹/L); mean corpuscular volume, 76.0 fL (81.6 to 98.3 fL); erythrocyte sedimentation rate, 120 mm/h (0 to 20 mm/h); and C-reactive protein, 76.4 mg/L (<6 mg/L). Polyclonal hypergammaglobulinemia (36% of 110.7 g/L total serum proteins) was present. Liver function tests and urinalysis were normal. A whole-body computed tomographic angiography revealed arteritic involvement of the thoracic and abdominal aorta, stenoses of the common carotid arteries, left subclavian artery, celiac trunk, and left renal artery, and occlusion of the superior mesenteric artery. A diagnosis of Takayasu arteritis was made according to the criteria proposed by the American College of Rheumatology.1

The patient underwent B-mode ultrasound of the carotid arteries that showed the presence of a long, smooth, homogeneous, concentric thickening of the arterial wall of both proximal common carotid arteries (Figure 1A and 1B).

These findings identified the typical vascular lesion for Takayasu arteritis in contrast with that seen in atherosclerotic plaques, which appear nonhomogeneous, often calcified, and associated with an irregular vessel wall.2

Subsequently, an ultrasound contrast agent (perfluoropropane-filled albumin microspheres; Optison, GE Healthcare, Nydalen, Norway) was injected into a peripheral vein. Contrast-enhanced imaging was performed using a 7L probe and a real-time contrast imaging modality, based on the pulse inversion principle, and a low mechanical index (0.08 to 0.10) was used.

The contrast agent improved image quality with a greater enhancement of vessel wall lumen and higher definition of the borders of the vascular lesion. Contrast-enhanced images (Figure 2A and 2B) clearly shows the presence of a large amount of a contrast signal within the lesions, as visualized by moving bright spots and linear flow of microbubbles within the vascular lesions (compare with a control subject in Figure 3). This phenomenon was principally seen on the adventitial side of the vessels and was considered to represent the contrast agent’s bubble signal coming from neovessels (online-only Data Supplement Movies 1 and 2).

As an internal control, a 5-frame high mechanical index flash was given during contrast agent administration to

Figure 1. B-mode ultrasound of the carotid arteries shows long, smooth, homogeneous concentric thickening of the arterial wall of the right (A) and the left (B) proximal common carotid arteries. In all panels, the asterisk marks the carotid artery lumen.
destroy the contrast and therefore to confirm that the signal was not an artifact. Thus, the gradual contrast replenishment, during the subsequent frames, demonstrates the perfusion of the vascular wall thickening (online-only Data Supplement Movie 3).

Contrast-enhanced ultrasound has been used for the detection of plaque neovessels in the carotid arteries and correlates well with histology (CD31 staining on endarterectomy specimens). Considering that an extensive plaque neovascularization is associated with features of plaque vulnerability and with clinically symptomatic disease, contrast-enhanced ultrasound imaging may provide valuable information for plaque risk stratification and for assessing the response to antiatherosclerotic therapies.

In the present case, the detection of contrast agent microbubbles within the vascular thickening as a marker of neovascularization is consistent with the initial inflammatory phase of Takayasu arteritis, which is typically characterized by inflammation and proliferation of the vessel vasa vasorum, which represent the main portal of entry for inflammatory cells into the vessel wall.

B-mode ultrasound can often help in identifying typical anatomic features of Takayasu arteritis, and contrast-enhanced ultrasound may allow the identification of inflammation-driven hyperemia and neovascularization, a potential marker of disease activity. Further studies are warranted to confirm these observations and the potential of this technique for monitoring disease activity and response to treatment in Takayasu arteritis and in other inflammatory arteritis.

Disclosures
Dr Coli received consultancy honorarium from GE Healthcare.

References

Key Words: inflammation, Takayasu arteritis, vasa vasorum, contrast-enhanced ultrasound, contrast agent
Assessment of Takayasu Arteritis Activity by Carotid Contrast-Enhanced Ultrasound
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Circ Cardiovasc Imaging. 2011;4:e1-e2
doi: 10.1161/CIRCIMAGING.110.960906

Circulation: Cardiovascular Imaging is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-9651. Online ISSN: 1942-0080

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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