Resolution of Existing Intimal Plaque in a Patient with Carotidynia

SUMMARY: We present a case of carotidynia that was imaged with 3 techniques: sonography, CT angiography, and gadolinium-enhanced MR imaging. We describe a previously unreported finding, the resolution of an intimal plaque noted on imaging at the time of initial presentation, presumably due to changes induced by the healing phase of the carotid inflammatory process.

CASE REPORT

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Carotidynia is an unusual cause of vascular neck pain. Findings on sonography, CT, and MR imaging may support this diagnosis in the appropriate clinical context. We present a unique case of carotidynia with resolution of an existing intimal plaque that was imaged with 3 techniques: sonography, CT angiography (CTA), and gadolinium-enhanced MR imaging.

Case Report

A 46-year-old man presented with a 1-week history of right-neck pain. This was preceded by a flulike illness with low-grade fever, chills, fatigue, and myalgia. The pain was aching in character, 3/10 in severity, and aggravated by movement. He had no headache and his vision was normal. Medical history included gastroesophageal reflux disease and occasional migraines.

On examination, the patient was afebrile and normotensive. There was localized swelling and tenderness overlying the right carotid bifurcation. There was no associated lymphadenopathy, and no carotid bruits were audible. White blood cell count, erythrocyte sedimentation rate, C-reactive protein, and autoimmune markers (antineutrophil cytoplasmic antibody, rheumatoid factor) were all normal. Initial imaging was performed approximately 1 week after symptom onset. Sonography demonstrated a thickened vessel wall and an eccentric soft plaque in the distal right common carotid artery (CCA) extending into the proximal internal carotid artery (ICA) (Fig 1). Further evaluation with CTA showed a crescentic fatty plaque, mildly narrowed lumen, concentrically thickened arterial wall, and fat stranding in the carotid space (Fig 2A, -C). Gadolinium-enhanced MR imaging confirmed enhancement of the arterial wall and the presence of a fatty plaque (Fig 3). Sixty milligrams of prednisone per day was initiated and tapered during 8 months to 10 mg per day for ongoing minimal discomfort. All changes resolved at follow-up imaging 5 months after presentation, including the eccentric fatty plaque, which resolved at follow-up with residual irregular inflammatory tissue or scarring (Fig 2B, -D).

Discussion

The causes of vascular neck pain are extensive and include entities such as dissection, giant cell arteritis, thrombosis, fibromuscular dysplasia, aneurysm, Takayasu arteritis, and nonvascular etiologies such as lymphadenitis and submandibular gland disease. After the exclusion of these diseases, carotidynia needs to be considered.

Carotidynia is an idiopathic unilateral neck pain syndrome characterized by tenderness over the carotid bifurcation. Neck pain is usually self-limited or resolves with nonsteroidal anti-inflammatory drugs or steroids in <2 weeks. In 2004, the International Headache Society published modified criteria for carotidynia, classifying it as a syndrome rather than a distinct pathologic entity (Table). In particular, the criteria specify that patients with carotidynia should not have a structural abnormality of the carotid artery (criterion C).

In patients with history, physical, and laboratory results suggestive of carotidynia, imaging with sonography, CT, and MR has revealed fairly consistent findings suggestive of carotid inflammation. Our case and multiple others have demonstrated 1 or more of the following imaging findings: involvement of a contiguous segment of the distal CCA, bulb, or proximal ICA; mild luminal narrowing; concentric thickening or outward extension of the wall; and enhancing wall and fat stranding in the adjacent carotid space. One report confirmed histologic findings of nonspecific inflammation of the carotid adventitia. The etiology of this inflammation is not clear. These imaging findings suggest that a structural abnormality of the carotid artery is present in carotidynia and that criterion C should be revised.

Unique to our case is the appearance of a fatty plaque that resolved at follow-up imaging with minimal residual irregular inflammatory tissue or scarring.

In summary, in the appropriate clinical context, a diagnosis of carotidynia as a cause for vascular neck pain may be supported by characteristic radiologic findings. This case report supports the imaging features of carotidynia previously described in the literature. In addition, our case had a finding not
previously described, the resolution of a fatty plaque noted on imaging at the time of initial presentation, presumably due to changes induced by the healing phase of the carotid inflammatory process.

**References**

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