

CTA, MR, and MRA Imaging of Carotidynia: Case Report

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Carotidynia is a term first coined by Fay in 1927¹ to describe a particular type of idiopathic neck pain, tending to be unilateral and associated with tenderness to palpation over the carotid bifurcation. This reproduction of pain with palpation later came to be known as a positive Fay sign. Patients are afebrile and lack any other systemic symptoms associated with infection or malignancy. Despite this characterization, a large differential diagnosis exists.

There are a number of case studies on the subject of carotidynia. Included in these reports are various debates as to the existence of carotidynia as a distinct pathological entity, a “catch all” description of symptoms produced by different etiologies, or simply a poorly understood syndrome. In 1988, the International Headache Society accepted acute idiopathic carotidynia as a valid entity and defined a set of four criteria²: A. At least one of the following overlying the carotid artery: 1. tenderness, 2. swelling, or 3. increased pulsations; B. Appropriate investigations not revealing a structural abnormality; C. Pain over the affected side of the neck; may project to the ipsilateral side of the head; D. A self-limiting syndrome of less than two weeks duration. Explicit to these original criteria is the understanding that other organic diseases in the differential diagnosis have been ruled out. This includes a variety of vascular, inflammatory, neoplastic, neurologic and musculoskeletal conditions, including vascular injury and dissection. In 2004, carotidynia was removed from this society’s diagnostic criteria after cases were reported with imaging findings³.

The current understanding of carotidynia as a clinical syndrome has been criticized for its lack of attention to imaging characteristics. There have been case reports demonstrating imaging findings on ultrasound, computed tomography (CT), computed tomography angiography (CTA), magnetic resonance (MR), magnetic resonance angiography (MRA), and positron emission tomography (PET). In effect, carotidynia by definition is a symptom of pain, yet it is also the name for the specific diagnosis of an inflammatory carotid wall disease that is the subject of this report.

In the following case study, we describe clinical findings of a patient diagnosed with carotidynia who presented with neck pain, and demonstrate the CTA, MR, and MRA imaging including follow-up imaging.

CASE REPORT

A 51-year-old woman developed sudden onset of pain on the left side of her neck while lying in bed one evening. She

described a muscle like strain. The pain was dull in nature and did not radiate. She also complained of transient, mild, dull pain along the left side of her face, and described a fullness on the left side of her neck. There was no history of trauma, neck manipulation, or other activities associated with dissections. There were no other symptoms or focal neurological complaints. Over the next two days, the pain persisted but the patient was otherwise well. On the third day, she reported a transient headache that lasted 30 minutes and resolved after eating. The pain in her neck increased on the third day. She works as a registered nurse in a community hospital, so she arranged for a carotid doppler ultrasound. The ultrasound demonstrated increased echogenicity around the left distal common carotid artery and proximal internal carotid artery. There was no luminal stenosis and the velocities were normal. The initial interpretation was left carotid artery dissection. The patient was transferred to our tertiary care centre, where she was admitted to the intensive care unit and briefly started on heparin.

On examination, the patient was alert and oriented and vital signs were stable. Neurological exam was unremarkable and did not reveal any focal neurological deficits. The patient complained of mild neck pain that was worse with head turning; looking right was worse than looking left. There was no ptosis, miosis, or other signs of Horner’s syndrome. No bruits were heard over the carotid arteries. There was no palpable neck mass. She did not report pain on palpation of either carotid artery, yet did claim to feel a “fullness” upon palpation of the left carotid artery. The remainder of the neurological and physical examinations were normal. Hematological and biochemical testing was normal including markers of inflammation.

A CT head without and with contrast was normal. A CTA of the head and neck showed a soft tissue halo around the left common carotid artery and proximal internal carotid artery extending from approximately C7-T1 to just above the carotid bifurcation at C3-4 (Figure 1). The halo was thicker along the

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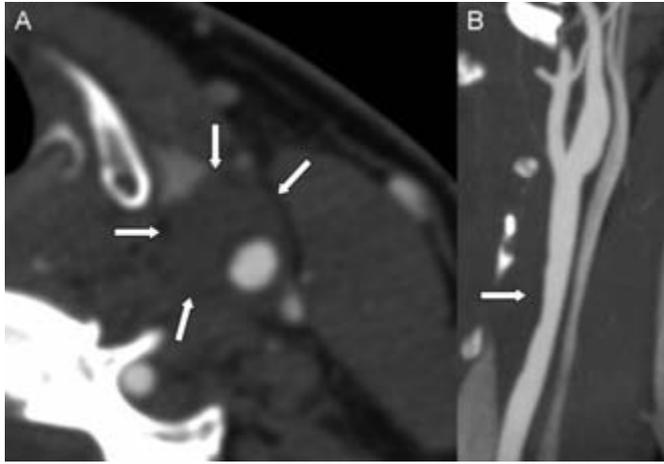


Figure 1: Axial CTA image of the left common carotid artery (A) demonstrates a halo of soft tissue around the artery (arrows) without significant luminal narrowing. The tissue is circumferential but thicker along the anteromedial aspect of the vessel. Oblique CTA multiplanar reformation (B) shows slight luminal narrowing and minor luminal irregularity.

anteromedial aspect of the carotid artery but appeared to surround the vessel circumferentially. There was slight narrowing and irregularity of the common carotid artery at the C5-6 level with a small focus of hypoattenuation along the anteromedial luminal wall. No intimal flap was present. There was no abnormality present in the right carotid artery.

On the next day, a MR showed a soft tissue cuff around the upper third of left common carotid and proximal internal carotid artery, thickest at the anteromedial aspect, minimally distorting the lumen (Figure 2). The tissue was T1 hypointense, T2 hyperintense, and enhanced following gadolinium. There was no hyperintensity on T1 imaging with fat saturation to suggest methemoglobin within the soft tissue. MRA showed no intimal flap or plaque present, but did show minor luminal narrowing and irregularity similar to the CTA. The rind of soft tissue was smooth in appearance, unlike the nodular appearance seen with paragangliomas. The conclusion made from the clinical information and imaging studies was the diagnosis of idiopathic inflammation of the carotid artery wall, also known as carotidynia. The heparin was stopped and the patient was discharged without any further treatment.

Three weeks later, a repeat MR and MRA of the head and neck was performed. The soft tissue cuff around the carotid artery near completely resolved with marked decreased thickness and decreased enhancement (Figure 3). Six months later the soft tissue cuff and enhancement had resolved (Figure 3). The minor luminal narrowing and irregularity had also resolved.

The enhancing soft tissue around the carotid artery suggests inflammation⁴. The lack of narrowing or irregularity of the vessel lumen implies that the significant pathology lies within the carotid adventitia itself as opposed to within the vascular endothelium as in vasculitides⁴⁻⁷. The symptoms associated with carotidynia are possibly due to the expansion of the vessel wall irritating the nerves surrounding the carotid artery wall.



Figure 2: Axial T2 image of the left common carotid artery (A) shows hyperintense soft tissue around the artery. Axial T1 with fat saturation and without contrast (B) shows hypointensity of the soft tissue indicating an absence of methemoglobin. Following gadolinium, axial T1 with fat saturation (C) shows homogeneous soft tissue enhancement. Maximal intensity projection MRA image of the left carotid artery (D) shows slight luminal narrowing and minor luminal irregularity, similar to the CTA.

Currently, imaging is critical to the understanding of carotidynia because it is difficult to confirm clinically and is poorly understood histologically. Since the carotid arteries are not typically biopsied, unlike the temporal arteries in temporal arteritis, few studies exist in the literature describing the histology. In 2003, Upton et al. described the unusual appearance of the adventitia around the internal carotid artery of a man who was experiencing neck pain prior to undergoing right carotid endarterectomy⁸. The adventitia was subsequently biopsied and proved to be negative for both gram stain and cultures. However, pathologic changes were present with vascular proliferation, proliferation of fibroblasts and low-grade chronic inflammation. This consisted of mainly lymphoblasts with a few poly-

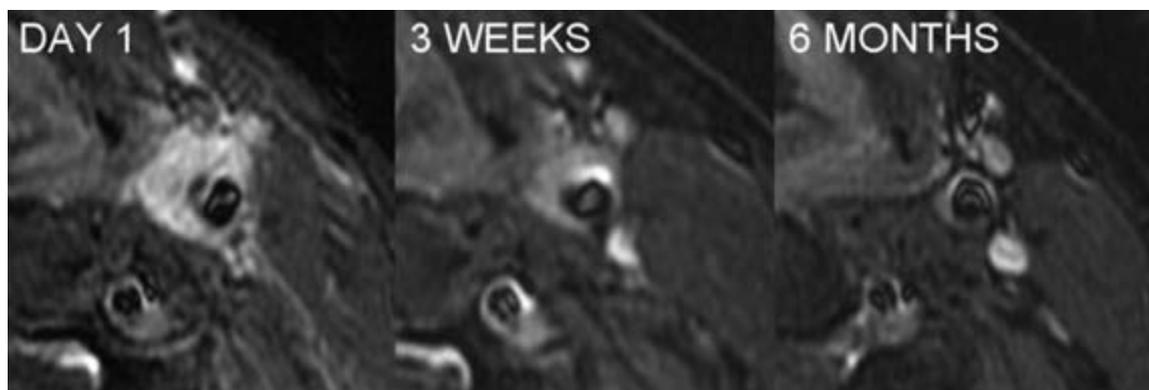


Figure 3: Axial T1 with fat saturation and with gadolinium of the left common carotid artery performed at presentation Day 1, 3 weeks after presentation, and 6 months after presentation. The enhancing soft tissue around the left carotid artery progressively decreases to resolution by six months.

morphonuclear cells and mast cells. Early fibrosis was noted to be present. These findings are significant in that they confirm that the changes were not consistent with an abscess wall or granulation tissue. Neither were they consistent with inflammation around a large vessel caused by vasculitis as no giant cells were present. More recently, Farage et al. performed a biopsy on their case after eight days of steroid and antibiotic treatment. The biopsy revealed only chronic inflammation without evidence of granuloma⁹.

In our case, the soft tissue cuff around the carotid was hyperechoic on ultrasound, hypodense on CT, hypointense on T1, hyperintense on T2, and enhanced on T1 post gadolinium. Most reported cases of carotidynia have shown hypoechoic^{10,11} wall thickening, some with mild luminal narrowing and an outward extension of the vessel wall. Most have reported hypodensity of the soft tissue on CT^{6,12}. On MR, there have been reports of both T2 hyperintensity^{4,6} and T2 hypointensity⁹. On T1, most report the soft tissue to be hypointense or isointense^{4,9}. Enhancement with gadolinium appears to be a constant finding^{4,6,9}. There is one report of carotidynia demonstrating activity on fluorodeoxyglucose-18 PET imaging¹³.

Our patient and those of other authors¹³ received no treatment and the disease resolved on its own. Others have treated with calcium channel blockers¹⁴, steroids^{9,15}, triptans¹⁶, and non-steroidal anti-inflammatory drugs¹⁷, also with resolution. Whether these cases would have resolved without treatment is unknown.

While the underlying cause remains elusive, it is apparent that the diagnosis of carotidynia refers to a distinct pathological and clinical entity which may be characterized with ultrasound, CT, MR and PET imaging. The case reported here characterizes the imaging findings and natural history. Such characterization may prove to be a valuable addition to the criteria for the diagnosis of carotidynia. A set of imaging characteristics specific to carotidynia may help rule out other more ominous causes of neck pain. Perhaps with improved diagnostic criteria for carotidynia, there will be an increase in the interest in the condition and subsequently further research leading to a better understanding of the etiology.

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