

TIPIC Syndrome: An Imaging Diagnosis in the Emergency Setting—A Case Report

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Abstract

A 34-year-old woman presented with sudden-onset pain in the left cervical region reaching the ipsilateral jaw and ear, with a tender, pulsatile swelling over the carotid bifurcation and normal inflammatory markers. Doppler ultrasound demonstrated focal hypoechoic wall thickening with preserved luminal flow, and contrast-enhanced CT confirmed isolated periadventitial inflammation without dissection. These findings established a diagnosis of carotidynia (TIPIC syndrome). Symptoms resolved rapidly with non-steroidal anti-inflammatory (NSAID), with imaging normalization at three weeks. This case underscores the role of targeted imaging in diagnosing carotidynia confidently and avoiding unnecessary invasive evaluation.

Keywords: CT Scan; TIPIC Syndrome; Vascular Inflammation; Dissection; Doppler US

Introduction

TIPIC syndrome or carotidynia is recognized by the presence of unilateral neck pain and tenderness overlying the carotid bifurcation and considered as an idiopathic, self-limiting, and non-life-threatening condition, reported first by Fay in 1927 [1]. Clinical manifestations typically consist of recurrent episodes of neck discomfort that resolve spontaneously within one to two weeks, although recurrences may occur over intervals ranging from several weeks to a few months [2]. Neurological manifestations are uncommon; however, isolated reports describing neurological complications have been documented in the literature [3]. Owing to its clinical presentation, carotidynia may mimic more serious vascular or neoplastic disorders, including carotid artery dissection and cervical malignancies, often prompting extensive diagnostic investigations and increasing patient concern [3].

Here, we present an illustrative case of a young woman whose acute neck pain and pulsatile cervical swelling raised initial concern for a vascular emergency, but whose imaging findings ultimately revealed the classic — yet often overlooked — features of carotidynia.

Case Report

A 34-year-old woman presented to the emergency department with severe unilateral pain in the left cervical area, accompanied by symptoms involving the ipsilateral ear and lower face. Physical examination revealed a tender, pulsatile nodularity over the left carotid bifurcation. Neurological examination was unremarkable. Laboratory investigations, including C-reactive protein concentration (CRP) and leukocyte count were within reference limits, with no biological evidence of systemic inflammation or infection.

Doppler ultrasound showed a short segment of hypoechoic wall thickening, centered on the carotid bifurcation, involving the left common and external carotid arteries with mild outward bulging and no significant luminal narrowing or flow acceleration (Figure 1).

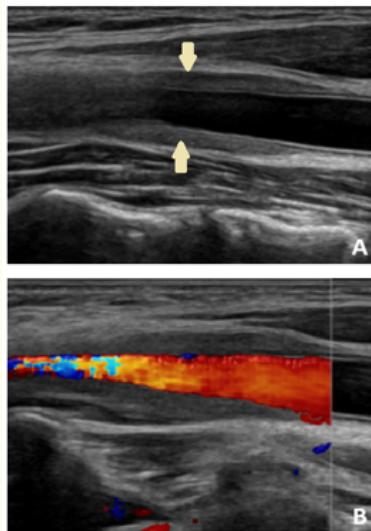


Figure 1: Doppler ultrasound: Hypoechoic eccentric thickening of the left carotid bifurcation (Yellow arrows) with preserved flow on color Doppler (A). No plaque or dissection flap observed. Features are consistent with inflammatory changes seen in carotidynia.

A subsequent contrast-enhanced CT scan confirmed localized perivascular inflammatory changes around the left common carotid artery without evidence of dissection or lymphadenopathy (Figure 2).

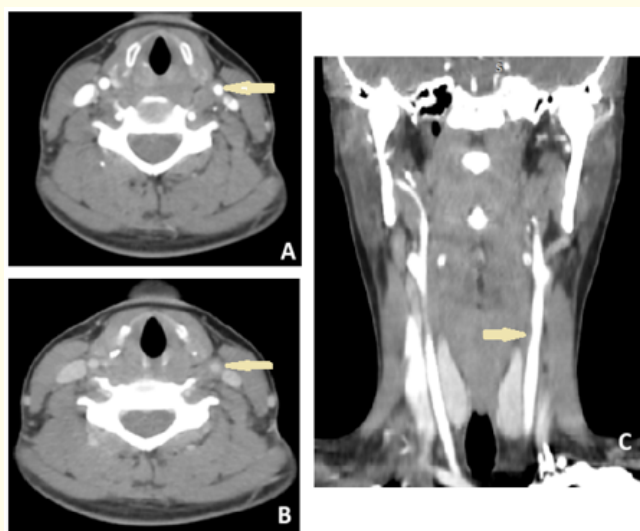


Figure 2: Contrast-enhanced CT: CT scan slices in the arterial phase — axial (A), sagittal (C) — and portal phase (B) show a focal perivascular soft-tissue thickening surrounding the left common carotid artery.

The patient was treated with NSAIDs, resulting in rapid pain relief and resolution of the palpable nodularity within days. Follow-up ultrasound at three weeks demonstrated significant decrease in wall thickening and normalization of the perivascular tissues, confirming the transient and self-limiting nature of the condition.

Discussion

Carotidynia, or TIPIC syndrome (Transient Perivascular Inflammation of the Carotid artery), is relatively uncommon and self-limiting inflammatory condition characterized by acute unilateral neck pain centered over the carotid bifurcation, sometimes radiating to the ear or face [4]. Its etiology remains uncertain, with proposed links to viral infections, autoimmune mechanisms, or migraine variants [5]. The clinical presentation typically involves point tenderness without overt signs of infection or neurological deficits, and the pain is often triggered or worsened by head movements or palpation [2]. Imaging is crucial to the diagnosis, both to identify characteristic findings and to exclude potentially serious differential diagnoses such as carotid artery dissection, neoplasms, or vasculitis [6]. The clinical presentation of our patient closely mirrors that reported in the literature; in the multicenter study by Leclerc, *et al.* [2], patients typically presented with acute unilateral neck pain localized over the carotid bifurcation, often radiating to the face, jaw, or ear [2]. Similarly, our patient experienced severe left-sided cervical pain radiating to the ipsilateral jaw and ear, associated with focal tenderness over the carotid bifurcation. As in most reported cases, no neurological deficits or systemic inflammatory manifestations were identified, supporting the benign inflammatory nature of the condition.

High-resolution ultrasound is the first-line modality, revealing a focal, eccentric, hypoechoic, focal mural thickening centered on the carotid bifurcation usually extending outward without significant luminal narrowing or hemodynamic disturbance features that help differentiate it from atherosclerosis or dissection. Color Doppler confirms preserved flow [2]. CT and especially MRI may show perivascular soft tissue infiltration, limited to the adventitia, with mild contrast enhancement and no associated thrombus or vessel wall dissection [6]. The imaging findings observed in our case are also consistent with those described in previous reports. Ultrasound demonstrated focal eccentric hypoechoic wall thickening with preserved luminal flow and without hemodynamically significant stenosis, features considered highly suggestive of TIPIC syndrome. Similar findings have been reported by Rafailidis, *et al.* and Leclerc, *et al.* [2,4], who identified eccentric perivascular thickening centered on the carotid bifurcation as the hallmark imaging feature of the disease [2,4]. Contrast-enhanced CT in our patient further confirmed localized perivascular inflammatory changes without evidence of dissection, aneurysm, thrombosis, or cervical lymphadenopathy, findings that closely resemble those reported in the largest imaging series.

Unlike vasculitis, the inflammation does not involve all layers of the vessel wall [6]. In some cases, PET-CT demonstrates increased FDG uptake localized to the affected carotid segment, which can be advantageous in ambiguous cases or when other imaging is inconclusive [7].

A hallmark feature of carotidynia is the spontaneous resolution of both symptoms and imaging abnormalities within 1 to 2 weeks, which can be confirmed by follow-up imaging. Treatment is conservative, typically involving NSAIDs, and recurrence is rare [6]. Imaging not only aids in diagnosis but also plays a key role in avoiding unnecessary interventions and reassuring both clinicians and patients of the self-limited nature of the disease.

Conclusion

This case illustrates the value of a stepwise imaging approach, beginning with high-resolution Doppler ultrasound, in establishing a confident diagnosis of carotidynia in the emergency setting. Recognition of the characteristic peri-adventitial wall thickening with preserved luminal flow allows clinicians to exclude carotid artery dissection and other serious differentials without resorting to more invasive or resource-intensive investigations. Given its self-limited course and excellent response to conservative management, increased awareness of carotidynia among emergency physicians and radiologists can reduce diagnostic delay, limit unnecessary testing, and improve patient reassurance and outcomes.

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