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# Imaging of a case of benign carotidynia with ultrasound, MRI and PET–CT

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**Summary** We present a case of carotidynia that we believe supports its classification as a distinct inflammatory disease entity. Doppler sonography and cervical magnetic resonance imaging are appropriate imaging modalities for diagnosing presumed carotidynia.

 $\label{eq:carotidynia} \begin{array}{l} {\bf Keywords} \ {\rm Carotidynia} \cdot {\rm Carotitis} \cdot {\rm Magnetic} \ {\rm resonance} \\ {\rm imaging} \cdot {\rm Ultrasonography} \end{array}$ 

# Bildgebung eines Falls von benigner Carotidynie mittels Ultraschall, Magnetresonanz und PET-CT

**Zusammenfassung** Wir stellen einen Fall mit Carotidynie vor, der unseres Erachtens die Klassifikation dieser Erkrankung als eigenständige entzündliche Krankheit unterstützt. Doppler Ultraschall und Magnetresonanz der Halsregion sind geeignete bildgebende Verfahren zur Diagnose einer suspizierten Carotidynie.

# Introduction

The term carotidynia was first used in 1927 by Fay to describe tenderness over the carotid artery [1]. It was

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C. M. Domenig, MD Department of Vascular Surgery, Medical University of Vienna, Vienna, Austria classified as a distinct entity in 1988, based on clinical criteria and the absence of structural abnormalities [2]. As controversy developed in the literature because many head and neck symptoms were attributed to carotidynia that did not meet those criteria, the IHS removed carotidynia as a separate entity in 2004 and now refers to it as a syndrome of unilateral neck pain [3]. Yet, carotidynia remains a controversial and poorly understood diagnosis.

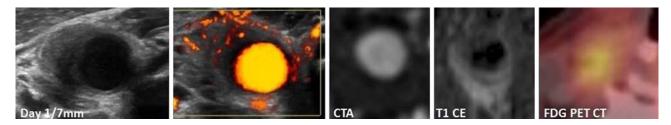
We present a case of carotidynia that we believe supports its classification as a distinct inflammatory disease entity.

#### Case report

A 52-year-old man, who presented with sudden onset of unilateral neck pain and moderate swelling, was referred by the ENT specialist for suspected thyroiditis. Laboratory tests including hemogram, erythrocyte sedimentation rate, C-reactive protein, rheumatologic markers, as well as tests for hepatitis b and c, HIV, and syphilis were negative or within normal ranges. No recent trauma could be confirmed. Ultrasound showed a circumscribed, 7 mm wall thickening on the posterolateral aspect of the right carotid bulb and distinct vascularization within the carotid wall. The lumen was not narrowed and normal spectral waves were obtained. The supplementary CTA was unremarkable, but MRA showed a diffuse contrast medium enhancement around the distal common carotid artery (CCA), and <sup>18</sup>FDG-PET-CT showed increased tracer uptake around the vessel (Fig. 1). No signs of dissection or morphologic disorders were present along all supraaortic vessels.

BMT consisted of aspirin 100 mg/day. Resolution of swelling was observed within 5 days. Continuous decrease of wall thickening and complete regression was seen within 55 days. (Fig. 2). The clinical course and

# case report



**Fig. 1** Baseline examinations. Sonography showed a posterolateral accented wall thickening with distinct vascularization. Computerized tomography angiogram appeared to be normal. Magnetic resonance imaging showed soft-tissue enhancement around distal common carotid artery on T1w postcontrast images and Positron emission tomographycomputed tomography (PET-CT) depicted increased tracer uptake around the vessel



Fig. 2 Follow-up examinations. Sonographic serial follow-up imaging showed that wall broadening halved after 15 days and appeared to be normal in the last sonographic control after 55 days

radiological findings indicated a final diagnosis of carotidynia caused by focal carotitis.

# Discussion

Stanbro et al. [4] reviewed 22 reports that showed consistent clinical and radiologic findings, such as wall thickening of the carotid artery or perivascular soft tissue enhancement that appeared to be specific for carotidynia. In 13 cases, color Doppler ultrasound depicted a focal soft tissue swelling around the affected carotid artery. In our case, we found a focal thickening of the carotid bulb that affected all layers of the carotid wall confirmed by MRI and Positron emission tomography-computed tomography (PET-CT). The detected distinct vascularization, suggested an underlying focal inflammatory process of the carotid wall. Upton et al. [5] reported active inflammation of the affected carotid artery in two patients who were undergoing carotid endarterectomy. Similar findings were reported by Farage et al. [6] who described an inflammatory pseudotumor within the carotid sheath after open surgical biopsy within the carotid wall.

The focal carotid thickening in patients with suspected carotidynia appears to be a self-limiting process, as seen in our case and in all cases reviewed by Stanbro et al. [4].

We agree with Stanbro et al. [4] that benign carotidynia or focal idiopathic carotitis seems to be an appropriate term for a painful focal swelling of the carotid wall. Ultrasound and MRI are appropriate imaging modalities for diagnosing presumed carotidynia.

## **Conflict of interest**

None of the authors state a conflict of interest

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