MRI Findings in Carotidynia

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Citation

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Abstract

The diagnosis of carotidynia, an idiopathic pain syndrome of the neck, remains controversial in part due to a paucity of associated radiographic findings. However, MRI/MRA provides diagnostic information in the evaluation of presumed carotidynia. Moreover, MRI/MRA findings localize the inflammatory process associated with carotidynia to the carotid sheath. We present a single case report in conjunction with a literature review and review the utility of magnetic resonance imaging [MRI] and magnetic resonance angiography [MRA] in confirming the diagnosis of carotidynia. We conclude that MRI/MRA should therefore be used as the primary radiologic modality by which to assess patients with presumed carotidynia.

BACKGROUND

Historically considered an idiopathic, self-limiting pain syndrome of the neck, “carotidynia” was first described by Dr. Temple Fay in 1927 as a diagnostic sign similar to atypical neuralgia.[1] More recently, the International Headache Society Classification Committee [IHSCC] recommended criteria for definitive diagnosis of idiopathic carotidynia, a self limiting syndrome of less two weeks duration, including at least one of the following overlying the carotid artery: tenderness, swelling and/or increased pulsations; Additionally, the IHSCC proposed that appropriate investigations be performed to exclude any structural abnormality of the affected carotid artery.[2] However, the committee did not provide specific investigational guidelines.

No imaging technique has defined pathognomonic findings of carotidynia. Therefore, it has been suggested that carotidynia should be considered a symptom rather than an independent syndrome. Unfortunately, few authors investigating the incidence and characteristics of carotidynia have regularly employed imaging techniques as part of the initial patient assessment.

CASE REPORT

A 58 year old otherwise healthy woman presented with a second episode of severe right sided neck pain. She reported that her pain was exacerbated by any neck movement. The first episode had spontaneously resolved after 1 week without medical intervention. There were no other reported associated symptoms. Review of systems was otherwise negative. Physical exam was remarkable for a positive Fay's test (pain elicited during palpation at the level of the bifurcation of the carotid artery). A contrast enhanced MRI was performed (Figures 1-3) which revealed moderate to intense enhancement of the carotid sheath on contrast enhanced T1 weighted images (Fig 1) and increased signal intensity attributed to the carotid sheath on T2 weighted images (Fig 2). MRA of the neck vessels was within normal limits (Fig 3).

Figure 1

Figure 1: Axial contrast enhanced T1 weighted image with fat saturation reveals moderate to intense enhancement of the carotid sheath at the level of the bifurcation of the right carotid artery.

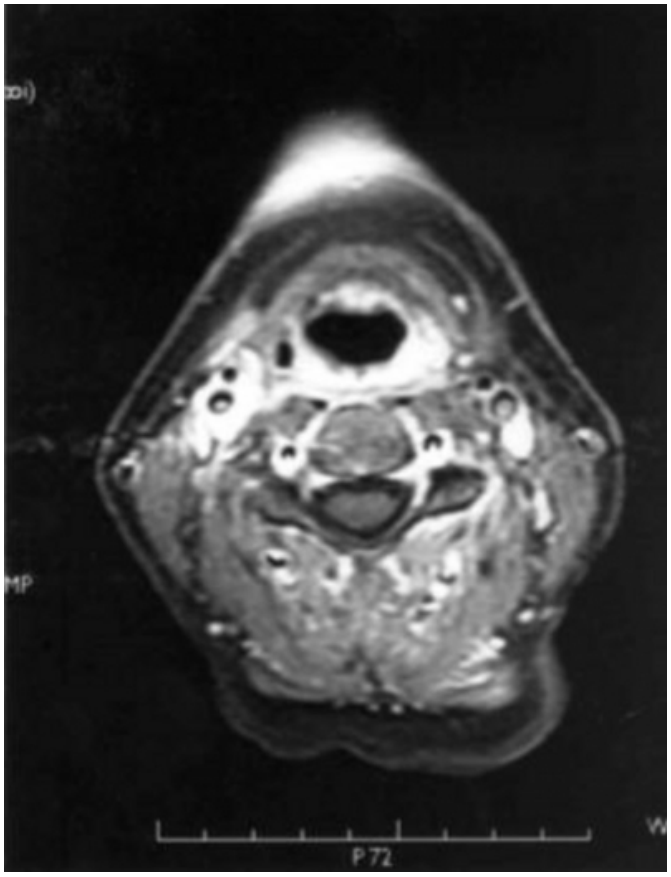


Figure 2

Figure 2: Axial fast STIR image revealing T2 weighted information shows increased signal of the carotid sheath at the level of the bifurcation of the right carotid artery.

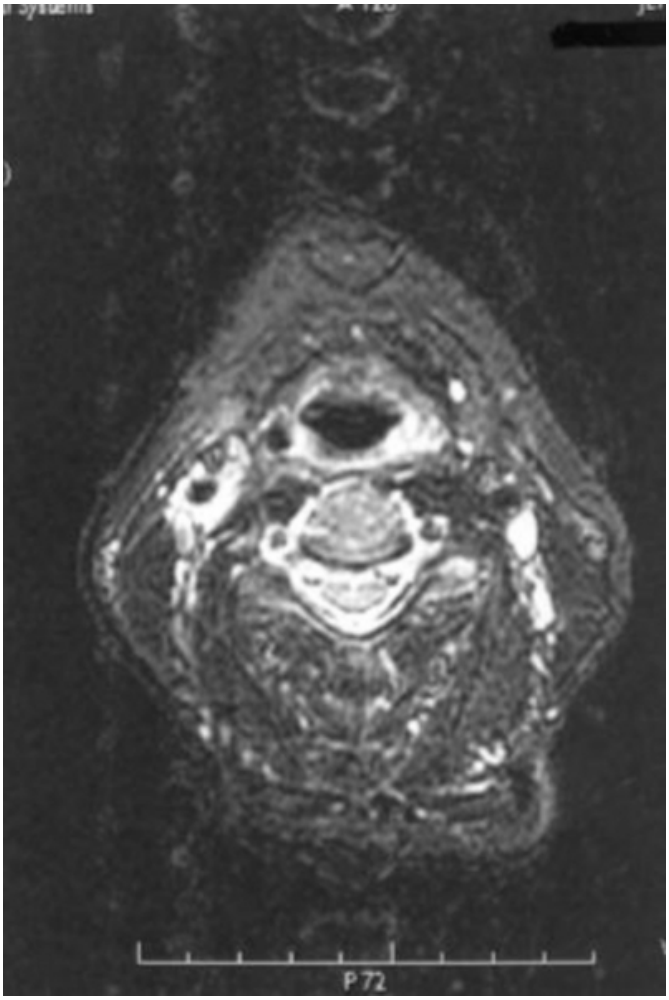
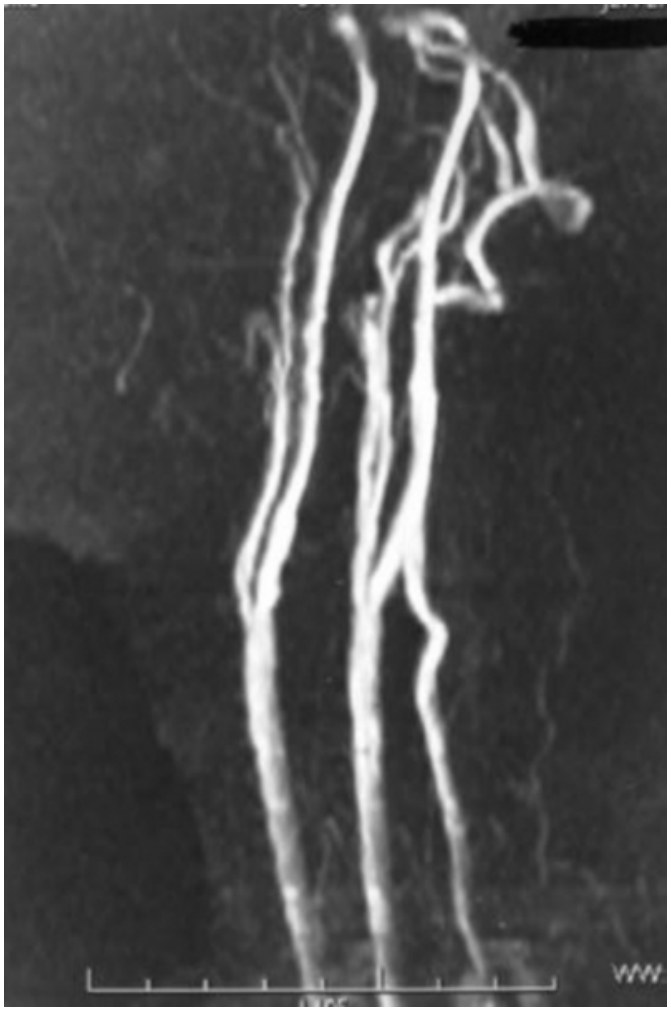


Figure 3

Figure 3: MRA of the neck vessels using time of flight technique reveals normal vessels, including no carotid artery stenosis or other physical abnormality inherent to the carotid artery on either side.



DISCUSSION

Although previous authors [3,4] had published case reports describing a carotidynia-type syndrome following Dr. Fay's initital description, D. Roseman[5] first proposed carotidynia as a distinct syndrome. Roseman reported 33 patients with a positive "Fay Test" who demonstrated specific clinical features including exacerbation of neck pain with head movement, chewing and swallowing. He proposed that this syndrome had a larger prevalence in the young adult population, specifically females; a finding which has been supported by other authors.[6]

Medline literature review reveals no large scale report of the incidence incidence or prevalence of carotidynia. However, Cannon[7] reports 25 of 1500 clinic patients identified with carotidynia. Emanmanuelli et al.[6] report 112 cases of diagnosed carotidynia at their institution over 10 years, an incidence of approximately one case per month.

The pathophysiology of carotidynia remains to be elucidated, yet several causes of carotidynia have been proposed. The syndrome is most commonly attributed to an unspecified virus. A viral etiology is supported given the self-limited nature of the disease. Other proposed etiologies include giant cell arteritis[8], vascular headache[4], migraine-like headache[9], dilation of the carotid artery[10]. Although carotidynia is self-limited by definition, suggested treatment includes non-steroidal anti-inflammatories [NSAIDs] or a short course of corticosteroids. No therapeutic regimen has been proven effective in a controlled study.

Despite the aforementioned reports of carotidynia, in 1993 Biousse and Bousser claimed carotidynia was not an independent entity, indicating that despite the hundreds of reported cases in the literature, the objective clinical findings forming the basis of its diagnosis had never been specified.[11] Importantly, Biousse and Bousser criticize the paucity of diagnositic imaging, specifically vascular investigations.

Syms and Burton support the usefulness of MRI and MRA in the diagnosis of carotidynia with characteristic findings particularly in T1 weighted images.[12] However, they reported no fast-spin echo T2 weighted findings such as those presented in this report. One year later, Burton et al.[13] published MRI results generally comparable to those found by Syms and Burton in 5 patients with carotidynia. Originally, Syms and Burton proposed that their lack of T2 signal abnormalities indicated an abnormality of the carotid vessel wall itself.[12] However, Burton et al attributed their findings, which included T2 weighted increased signal intensity, to inflammation within the carotid sheath. Burton et al. do not emphasize their T2 weighted findings, commenting on only one case with subtle T2 findings and one case with T2 weighted findings confounded by fat saturation failure too low in intensity to distinguish from carotid flow void. Most recently, Buetow and Delano describe the first case report of computed tomography [CT] findings revealing soft-tissue infiltration of the carotid sheath.[14] An MRI of this same patient revealed enchancing tissue in the carotid sheath on T1 weighted images as well as increased intensity on T2-weighted images, similar to the findings presented in this paper. Our findings in conjunction with those previously reported by Buetow and Delano serve as further evidence to support that carotidynia is indeed an independent syndrome with characteristic radiographic

findings representing a unique pathophysiology connected to inflammation of the carotid sheath. Significantly, we note characteristic T1 and T2 weighted findings not associated with any structural abnormality of the carotid artery.

CONCLUSION

All the cases of carotidynia with associated MRI findings found in the otolaryngology literature meet criteria of IHSCC for the diagnosis of idiopathic carotidynia. These objective findings support classification of carotidynia as a unique syndrome likely associated with carotid sheath inflammation. The MRI/MRA findings delineated in this paper help to further elucidate the pathophysiology of carotidynia by localizing the syndrome's characteristic inflammation to the carotid sheath. MRI/MRA should therefore be used as one of the primary diagnostic modalities by which to assess patients with presumed carotidynia.

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References

1. Fay T. Atypical Neuralgia. Archives of Neurology and Psychiatry 1927;18:309-15.
2. Headache Classification Committee of the International Headache Society. Classification and diagnosis criteria for headache disorders, cranial neuralgias and facial pain. Cephalgia 1988;8 (suppl 7):48-9.
3. Davies JVSA. Carotid Pain: Report of six cases. British Medical Journal 1961;2:1528-29.
4. Lovshin LL. Vascular Neck Pain: A common syndrome seldom recognized. Cleveland Clinic Quarterly 1960;27:5-13.
5. Roseman DM. Carotidynia. Archives Otolaryngology 1967;85:103-6.
6. Emmanuelli JL, Gutierrez JR et al. Carotidynia: a frequently overlooked or misdiagnosed syndrome. Ear, Nose & Throat Journal 1988;77(6)462-4,466,469.
7. Cannon CR. Carotidynia: an unusual pain in the neck. Otolaryngology-Head and Neck Surgery 1994;110(4):387-90.
8. Pearse HE, Hinshaw JR. Bilateral arteritis simulating carotid body tumors. Surgery, Gynecology & Obstetrics 1956; 103:263-66.
9. Ford FR. The carotid pain syndrome; report of two cases which suggest that, in some instances, migraine is responsible. Bulletin of Johns Hopkins Hospital 1964;114:266-68.
10. Hilger JA. Carotid pain. Laryngoscope 1949;59:829-38.
11. Biousse V, Bousser MG. The Myth of Carotidynia. Neurology 1994;44(6):1993-95.
12. Syms MJ, Burton BS, Burgess LPA. Magnetic Resonance Imaging in carotidynia. Otolaryngology-Head and Neck Surgery 1997;117(suppl):156-9.
13. Burton BS, Syms MJ et al. MR imaging of patients with carotidynia. American Journal of Neuroradiology 2000;21(4):766-9.
14. Buetow M, Delano M. Carotidynia. American Journal Roentgenology 2001;177(4):947.

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