Carotidynia: Similar Clinical Picture, Different Radiological Findings

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Carotidynia is an unusual condition that is characterized by pain at the carotid artery bifurcation without any abnormality except for tenderness on palpation. The independent existence of this entity is generally doubted. Recently, some radiological and histological clues to this disease have been reported. Although the exact cause of carotidynia is unknown it is thought that this entity is a self-limiting inflammatory process and may be a subset of vasculitis. Treatment is conservative and anti-inflammatory drugs are effective. In this paper, we report three carotidynia patients with similar clinical features but different radiological findings.

Key Words: Carotid artery diseases, anterior neck pain, inflammation, vasculitis

Introduction
In routine otolaryngology practice, pain is related to infectious causes in many cases. When a patient suffers from atypical anterior neck pain without significant clinical findings, diagnosis may be difficult for many otolaryngologists. In cases of acute anterolateral neck pain, the diagnosis of ‘carotidynia’ should be considered. However carotidynia patients usually remain undiagnosed and are prescribed antibiotics for tonsillitis or lymphadenitis.

In carotidynia, the pain is typically dull, throbbing, continuous and may be progressive. The pain may be aggravated with swallowing, coughing, sneezing and head movements. The only physical sign is tenderness on palpation of the carotid bulb. Carotidynia is a self-limiting condition, and fortunately improves within a couple of weeks (1). Anti-inflammatory drugs and steroids are the drugs of choice and are highly effective (1). Although considered a disease of the elderly, we report three relatively young patients suffering from carotidynia with similar clinical features and different radiological findings.

Case reports
Case 1
A 33-year-old woman presented with right neck pain for 10 days without any other symptoms. She denied any radiation of the pain but the pain was becoming worse with swallowing. The patient revealed that she had been diagnosed as having tonsillitis and had used an antibiotic for 1 week but that the pain had become worse. Her past medical history was not significant except for gastritis. Routine otolaryngological examination of the patient was normal except for tenderness on palpation of the right carotid bulb. Neck ultrasonography and carotid Doppler sonography did not reveal any structural abnormality. With a presumptive diagnosis of carotidynia, a 2-week course of etodolac therapy was prescribed and the patient’s pain subsequently resolved.

Case 2
The second patient was a 35-year-old man who presented to the ENT clinic with a history of left neck pain for 15 days. The patient stated that the pain had improved spontaneously, but had recurred a few...
days earlier. He described the pain as ‘throbbing’, continuous and as radiating to the jaw. His past medical history was not significant. Physical examination of the head and neck revealed no pathological findings other than tenderness at the left carotid bifurcation. Neck ultrasonography revealed a reactive enlarged lymph node of 15 mm diameter, at the level of the left carotid bifurcation. Carotid Doppler sonography was normal. Etodolac treatment provided rapid relief of the symptoms.

Case 3
The third patient was a 38-year-old man who presented with a 10-day history of right neck pain, which was aggravated by swallowing and radiated to the ear, eye, jaw and teeth. He reported that he had been given a 5-day course of antibiotic therapy that had not helped. His past medical history was not significant. The left carotid bifurcation was painful on palpation but other otorlaryngological examination was normal. Neck ultrasonography did not reveal any pathological findings. In carotid Doppler sonography, increased echogenicity of the pericarotid tissues and an enlargement in the whole carotid artery posterior wall layers 15 mm segment at the carotid bifurcation were seen (Figure 1). The carotid posterior wall was 3.5 mm at the thickest point and the carotid lumen and flow pattern were all normal. The patient was given etodolac and the pain resolved dramatically.

Discussion
There is some controversy about the definition, existence, aetiology and characteristics of this entity. Carotidynia is generally defined as unilateral neck pain sometimes radiating to the ear, teeth, mandible and eye. The only physical sign is tenderness on palpation of the carotid artery. The term ‘carotidynia’ was initially described by Fay in 1927, and since the first description, carotidynia cases have rarely been reported in the English medical literature (2). In 1988, acute idiopathic carotidynia was classified by the International Headache Society (IHS) Classification Committee based on four diagnostic criteria: A) At least one of the following overlying the carotid artery: 1. tenderness, 2. swelling, 3. increased pulsations. B) Appropriate investigations do not reveal 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 2-weeks duration (3). However, carotidynia was subsequently removed from the classification in 2004 (4).

Although some authors believe that “carotidynia” is underdiagnosed, others think that it does not even exist. In 1994, Bousse and Bousser reviewed the literature and concluded that ‘carotidynia’ was a non-validated entity and should be preferably expunged from the medical nosology (5). According to the authors, carotidynia was simply a syndrome of unilateral neck pain with local tenderness that could be due to numerous vascular and nonvascular causes and did not imply a carotid origin. However, radiological and histological evidence has recently been reported and the existence of carotidynia is now less doubtful (6-9). In 2003, Upton et al. (6) reported the histological confirmation of carotidynia. The authors reported pathological changes consisting of vascular proliferation, proliferation of fibroblasts and low-grade chronic active inflammation beside gross carotid artery abnormalities in a 70-year-old man.

The aetiology of carotidynia is not known but it has been proposed to be a subset of vasculitis (10). Reports of elevated levels of high-sensitivity C-reactive protein, erythrocyte sedimentation rate and serum amyloid A protein levels support the inflammation thesis in or around the carotid artery wall (10, 11). Recent reports in the literature demonstrated the presence of amorphous enhancing soft tissue surrounding the carotid bifurcation and wall thickening on magnetic resonance, ultrasonography and Doppler in patients with carotidynia, suggesting the presence of an inflammatory process (7-9). Positron emission tomography evaluation of a carotidynia patient demonstrated a short segment of increased [18F]fluorodeoxyglucose activity corresponding to the region of soft tissue thickening within the carotid sheath (11). These findings strongly support the presence of inflammation.

The treatment of carotidynia remains largely medical (1). The patient should be reassured that no serious underlying problem exists. A short course of an anti-inflammatory drug or steroids is highly effective, probably by healing the local inflammation (1). In our cases, etodolac, a strong anti-inflammatory drug, effectively improved the symptoms.
In our opinion carotidynia is a symptom of pain arising in or around the carotid artery. This symptom may also be a result of vascular and nonvascular causes which should be excluded initially in carotidynia patients (Table 1) (5, 8, 11-16). However idiopathic “carotiditis” or “pericarotiditis” may cause carotidynia and there may not be a detectable lesion in imaging studies. We believe that local inflammation initiates the disease process and that the degree of inflammation determines radiological findings. Neck pain and tenderness on palpation of the carotid bulb are invariable symptoms but radiological findings may differ according to the severity and duration of inflammation.

**Conclusion**
As in our cases, inflammation may be undetectable, or may cause reactive enlargement of an adjacent lymph node or the carotid wall.

**Conflict of interest**
No conflict of interest was declared by the authors.

**References**
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