Superficial Temporal Artery Dilatation in a Patient with Infectious Temporal Headache Clinically Mimicking Temporal Arteritis

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Abstract

A 57-year-old woman noticed a pulsatile shooting headache in her right temporal region 3 days after extraction of a tooth from the right mandibula. The following day, a localized headache over the right superficial temporal artery (STA), low grade fever, and jaw claudication appeared and progressed subacutely. Seven days after the onset, magnetic resonance imaging and angiography (MRI/MRA) disclosed inflammatory swelling of the right temporal muscle and dilatation of the right STA. All the symptoms disappeared following antibiotic treatment, and neuroimaging findings were improved. In conclusion, MRA is thought to be useful to non-invasively identify reversible inflammatory dilatation of extracranial vessels.

Key words: magnetic resonance angiography (MRA), vasodilatation, extracranial lesion

Introduction

Magnetic resonance angiography (MRA) has recently been a useful tool for non-invasively detecting vascular abnormal lesions in not only intracranial but also extracranial arterial occlusive disease (1, 2). However, the usefulness of MRA is still controversial for detecting vasculitic lesions (3, 4). Herein, we report the MRA findings in a patient with infectious temporal headache clinically resembling temporal arteritis/giant cell arteritis (TA/GCA), and briefly discuss the MRA findings in comparison with previous literature on patients with TA/GCA.

Case Report

A 57-year-old, right-handed woman had suffered from a localized headache for one week. The headache consisted of two different conditions; continuous localized dullness at the right temporal region and paroxysmal pulsatile shooting pain at the same region intermittently every 5 to 10 minutes. Nausea and vomiting did not accompany the headache. She had a history of tooth extraction at the right mandibula at a local clinic and she was taking oral antibiotics and a non-steroidal anti-inflammatory agent. Two days after the tooth extraction, she noticed right temporal dullness in spite of her medication, and 3 days after the tooth extraction, right temporal shooting pain, jaw claudication and intermittent high fever appeared, but she did not feel any pain in the mandibula. Since the symptoms aggravated rapidly, she visited our hospital and was admitted to treat her headache, seven days after the tooth extraction.

Physical examination showed a continued fever around 38°C and right temporal swelling with marked tenderness just over the right superficial temporal artery (STA). The arterial pulsation at that region was almost preserved as compared to the opposite side, and the skin surface appeared to be normal. Jaw claudication was also observed, i.e., she could open her mouth open only 5 mm. Bilateral optic fundi were normal, and no neurological deficit was shown.

Laboratory examinations showed mild leukocytosis (11,100/µl) and elevated C-reactive protein (16.4 mg/dl). Serologically, Wasserman reaction was negative, and no autoantibodies including antinuclear, anti-DNA, Sm and antineutrophil antibodies were identified. Rheumatoid factor, ASLO and ASO were all within the normal range. Brain CT scan showed a swelling of the right temporal muscle and no abnormality was detected in the intracranial space.

In order to visualize the inflammatory activity of the right temporal structures and to recognize the vascular abnormality of the right STA, brain MRI and MRA were studied on the day of her admission. On T1 weighted images, MRI revealed a swelling of the right temporal muscle in association with high T2 signals of the subcutaneous perimuscular connective tissue. The right temporal muscle was partly enhanced by using a gadolinium contrast material [Gd, gadolinium-diethylene-
triamine penta-acetic acid-bis-methoxyethyl-amide (Gd-DTPA-BMA]) (Fig. 1A and B). Three dimensional time-of-flight MRA (Magnetom Vision, 1.5T, Siemens Co.) showed a longitudinal dilatation of the right STA corresponding to the right temporal muscle swelling (Fig. 2A and B).

After admission, her symptoms were gradually relieved by administration of intravenous piperacillin 4 g/day and oral anti-inflammatory drugs. In 3 weeks, her inflammatory signs and laboratory data were completely improved. Steroid therapy was not performed during her clinical course. Two months after the onset, when her clinical symptoms disappeared, follow-up brain MRI and MRA were performed. The swelling of the right temporal muscle and dilatation of the right STA had disappeared completely (C and D of Figs. 1 and 2).

Discussion

Taking the clinical history and efficacy of antibiotics of this
Temporal Headache and MRA

Figure 2. Vascular images serially obtained by magnetic resonance angiography. The day of admission (A and B). Viewing from both axial and frontal images, the right superficial temporal artery (STA) is significantly dilated as compared to the opposite side. The location of the dilatation is in good agreement with the region of temporal headache and muscle swelling shown in Fig. 1. Two months after the disease onset (C and D). The abnormal dilatation of the right STA returned to normal. Ant.: anterior, Post.: posterior, R: right, L: left.

First, the injured muscles are known to be enhanced by Gd either slightly or markedly, and the destruction of muscles was histopathologically proven by biopsy of the muscle with abnormal MR intensity (5). Therefore, in spite of the absence of pathological findings in the present patient, swelling of the right temporal muscle with Gd enhancement suggested localized reactive myositis or muscle injury secondary to the inflammation of the masticator space, and this could cause localized continuous dullness.

Secondly, the pulsatile temporal shooting pain and marked tenderness of STA were possibly related to the inflammatory dilatation of the right STA as shown by MRA. The pathophysiological mechanism of inflammatory vasodilatation has not been studied extensively. It appears likely that the local vascular dilatation, in turn, might lead to the continuous dull pain, which is a feature of temporal headache.

case into account, the etiology in the present patient was considered to be possible bacterial infection in association with tooth extraction followed by inflammatory spread. The infection might have occurred primarily at the right oromandibular masticator space, which is composed of the upper ends of several muscles including the masseter, medial pterygoid and the lower part of the temporal muscle bundles. Since the superior border of this space is a deep layer of temporal muscle, inflammation of this area could have extended easily to the temporal muscles. Jaw claudication in the present patient was likely to be derived from inflammatory involvement of this space. In the present patient, serial MRI/MRA studies were very useful to recognize the pathophysiology of the patient’s headache.
been clarified yet. In general, inflammatory processes essentially require excess blood as a response to internal/external inflammatory trigger. Furthermore, in the trigeminal territory especially within the oromandibular region, it is known that painful neural afferent stimuli can cause sustained local vasodilatation (6). Sensory neuropeptides including calcitonin-gene related peptide, substance P, and others are considered to play some roles in such situation (6). Interestingly, the inflammation in the present patient derived from the oromandibular area and it can theoretically be speculated that the release of neuropeptides in response to shooting pain may have caused the reactive STA dilatation. Since this is not conclusive from a single case, further observations in human and animal studies are warranted.

Understanding the pathophysiological vascular changes shown on MRA seems to be important, especially in cases such as the present patient. Considering the clinical manifestation of the present patient, temporal arteritis/giant cell arteritis (TA/GCA) should be considered as a candidate of differential diagnosis (7). It is evident that earlier diagnosis and treatment of TA/GCA lead to a better outcome, and histopathological evaluation is usually required to make a definite diagnosis of the disease. In the present patient, MRI/MRA was rapidly performed to confirm the inflammation because the patient was initially hesitant to undergo biopsy of the right temporal region. Sellar postulated that MRA did not supercede conventional angiography in identifying vasculitic lesions (3), and another recent study also disclosed that 17% of intracerebral vasculitis lesions identified by conventional angiography showed negative results by MRA (4). In contrast, the usefulness of MRA has also been reported in cases with peripheral arteriopathy including various kinds of vasculitis (8). Mitomo et al reported a unilaterally stenotic STA lesion in a patient with TA/GCA clearly shown by MRA (9). Harada et al also documented a treatable bilateral STA narrowing in a patient with clinically diagnosed TA/GCA (10). These reports are consistent with the pathophysiology of TA/GCA. In addition, vascular dilatation of the distal part of stenotic vessels, like in the present patient, is known as a paradoxical dilatation in conventional angiography. Since MRI machines and scanning procedures have rapidly improved, future accumulation of such findings of MRA in TA/GCA or abnormal temporal artery will provide more useful information to non-invasively make or exclude a diagnosis in this kind of disorder. It is concluded that MRA is thought to be a useful tool to identify inflammatory dilatation of extracranial vessels.

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References