◎原　著

A Case of Giant Cell Arteritis Associated with Rheumatoid Arthritis Monitored by Magnetic Resonance Angiography

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Abstract: We report a patient who developed giant cell (temporal) arteritis during treatment of rheumatoid arthritis. The patient was a 57-year-old Japanese woman and had been well controlled with anti-rheumatic drugs, when she presented to our hospital with severe bitemporal headaches and marked fatigue. An exclusive diagnosis was rapidly made and she was diagnosed as having giant cell arteritis based on the classification criteria by American College of Reumatology. Additionally, magnetic resonance (MR) angiography was performed, from which stenotic change in the bilateral superficial temporal arteries was strongly suspected and then corticosteroid therapy was quickly started . The patient followed an uneventful course without developing any complications such as visual dysfunctions. Therapeutic effect was confirmed by MR-angiographic findings obtained 4 weeks after the initiation of therapy.

Key words: giant cell arteritis, temporal arteritis, rheumatoid arthritis, MR-angiography

Introduction

Giant cell (temporal) arteritis (GCA) is a granulomatous vasculitis mainly accompanied by giant cells in the temporal artery, whose occurrence is considered relatively rare in Japan. The disease develops mainly in elderly women over 50 years of age, being known to be frequently associated with polymyalgia rheumatica (PMR). The diagnosis is usually based on clinical signs and findings of a temporal artery biopsy.

We described here a patient with rheumatoid arthritis (RA) progressed from juvenile rheumatoid arthritis (JRA) complicated by GCA in whom MR-angiographic scanning of the superficial temporal arteries was useful in assisting to make a diagnosis, deciding the time at which treatment was initiated, and judging the treatment effect.

Case Report

The patient was a 57-year-old Japanese woman

whose chief complaint was bitemporal headaches. In 1952, when she was 12 years old, she developed JRA, which progressed RA. In 1995, she was admitted to our hospital because of hematemesis (the source remained unknown). In September 1997, she was hospitalized because of melena (the source also remained unknown). There was no family history of RA or other rheumatic diseases. She had been treated in the department of internal medicine of our hospital since she was 24 years old for RA. She was treated mainly with salazosulfapyridine 1g/day and prednisolone 5 mg/day and the disease activity of RA was well controlled. From February 1998, sometimes she had been suffered from slight vertigo, but examinations performed on March 1998 showed no abnormal findings, including normal ranges of CRP or erythrocyte sedimentation rate (ESR). Around 5 May 1998, throbbing pain at the bilateral temple regions appeared and gradually aggravated. On 12 May 1998, severe bitemporal headaches and marked fatigue caused her to present to our hospital and she was hospitalized that day.

Physical examination on admission indicated that the patient was 154.5 cm tall and weight 43.5 kg without any history of weight loss. Her temperature was 36.7 ℃, blood pressure 146/96 mmHg on both sides, and pulse 80/min and regular. She was alert and without any neurologic findings. Visual acuity was not diminished. There were no abnormal findings in the heart, lung or abdomen. Lymphadenopathy and rheumatoid nodules were not observed. Muscle pain or weakness in the extremities and jaw claudication were not noted. Deformity or ankylosis resulting from RA were observed at joints such as proximal interphalangeal, metacarpophalangeal, wrist, knee and ankle joints. Thickening, tenderness and decreased palpable pulsation of the bilateral superficial temporal arteries, especially in the left superficial temporal artery (STA) were noted.

Laboratory examinations on admission showed slightly increased WBC (10,700/ μ l) and platelets (37.5×10^4/ μ l), elevated CRP (2.5mg/dl) and slightly elevated ESR (24mm/h). Immunological examinations showed normal immunoglobulin in either class of IgG, IgA and IgM. Rheumatoid factor (RF) slightly increased to 20.7 U/l. C3 (76mg/dl) and C4 (25mg/dl) were normal but serum level of complement (CH50) slightly increased to 55 U/ml. Circulating immune complex (Clq method), antinuclear antibody, antineutrophil cytoplasmic antibodies (ANCA) were all negative (Table 1).

Table 1. Laboratory Findings on Admission

Complete Blood Count		Serological Immunological Test	
White blood cell	10,700/μl	C reactive protein	2.5 mg/dl
Red blood cell	505 x 10^4/μl	Erythrocyte sedimentation rate	24 mm/h
Hemoglobin	14.7 g/dl	IgG	1167 mg/dl
Hematocrit	43.2%	IgA	327 mg/dl
Platelet	37.9 x 10^4/μl	IgM	60 mg/dl
Blood Chemistry		C3	76 mg/dl
Total protein	7.7 g/dl	C4	25 mg/dl
Albumin	64.1%	CH50	55 U/ml
α_1-gl	4.4%	Treponema pallidum hemagglutination	
α_2-gl	12.6%	test	negative
β-gl	10.4%	HBs antigen	negative
γ-gl	8.1%	HCV antibody	negative
Blood urea nitrogen	12.4 mg/dl	Rheumatoid factor	20.7 IU/ml
Creatinine	0.5 mg/dl	Anti-nuclear antibody	negative
Na	143 mmol/l	Anti-neutrophil cytoplasmic antibody	
K	4.3 mmol/l	Protainase 3 ANCA (p-ANCA)	< 10 EU
Ca	9.5 mg/ml	Myeloperoxidase ANCA (c-ANCA)	< 10 EU
Total bilirubin	0.23 mg/dl	Circulating immune complex (C1q)	< 1.5 μg/ml
Aspartate aminotransferase	22 IU/l		
Alanine aminotransferase	7 IU/l	Urinalysis	
Lactate dehydrogenase	126 IU/l	Occult blood	(-)
γ-glutamyltranspeptidase	27 IU/l	Protein	(-)
Alkaline phosphatase	143 IU/l	Sugar	(-)
Creatinephosphokinase	15 IU/l	Sediment	n.p.
Total cholesterol	228 mg/dl		
Fasting blood sugar	133 mg/dl	Stool	
		Occult blood	(-)

Head computed tomography (CT) findings were normal except an old small infarction in left putamen. Ophthalmoscopy did not reveal abnormal findings such as cotton-wool patches.

Based on head CT and neurologic findings obtained on the day admission, other headache showing diseases such as subarachnoid hemorrhage, meningitis, or encephalitis were excluded. The patient's age of 50 years or more, newly developed headache localized to bilateral temples and apparent abnormality of STAs (thickening, tenderness and decreased palpable pulsation), which fulfilled the classification criteria for GCA by the 1990 American College of Rheumatology (ACR), established a diagnosis of GCA. To assist the diagnosis, MR-angiographic scanning of the STAs was performed and revealed difficulty in visualizing both arteries (Figure 1A), from which stenotic change

was strongly suspected. Treatment with prednisolone (PSL) 30 mg/day was started. On hospital day 3, bitemporal headaches and tenderness of STAs disappeared and laboratory data on CRP (0.5 mg/dl) and ESR (19mm/h) also showed a slight improvement. On hospital day 8, CRP decreased to 0.0mg/dl. Approximately 4 weeks' continuous treatment with corticosteroid resulted in relief in thickening at bilateral STAs and improvement in pulsation palpability was observed. At that time, MR-angiography was performed once again to scan the STAs. Bilateral STAs could be visualized as far as the periphery, indicating improvement in stenosis (Figure 1B, Figure 2). During the treatment course, there were no abnormal findings such as diminished visual acuity.

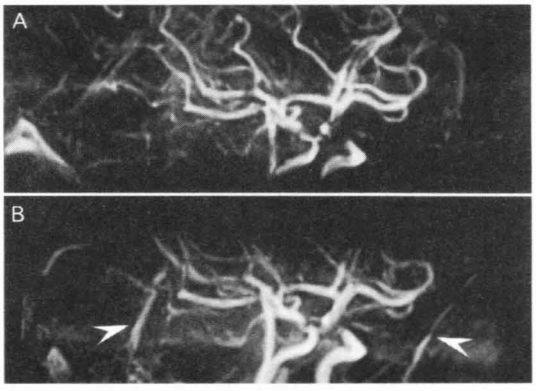

Figure 1. A) Magnetic resonance (MR) angiography on admission. Visualization of the periphery of bilateral superfical temporal arteries were extremely poor in any scanning conditions and their image can scarcely be read.
B) MR angiography of the same region after 4 week-treatment with corticosteroid. The bilateral superficial temporal arteries could be clearly visualized as far as the periphery (arrow).

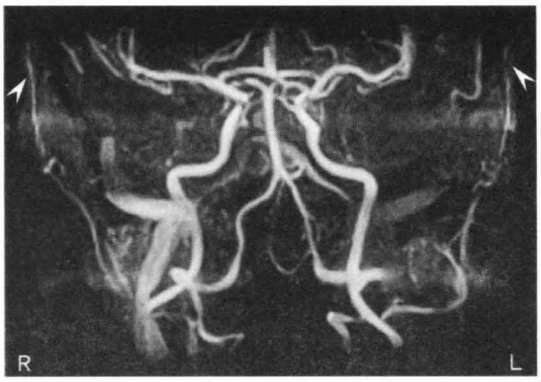

Figure 2. MR-angiography after 4 week- treatment with corticosteroid. Arrows show bilateral superficial temporal arteries visualized as far as the periphery. The diameter of the left superficial temporal artery seem to be slightly reduced at the periphery.

Discussion

GCA involves vasculitis of medium-sized and larger vessels, found mainly patients over 50 years of age. Though it was considered that morbidity for GCA is higher in Caucasians than in Asians, GCA may not necessarily be a rare disease considering the increasing number of reports on GCA in Japan. GCA is accompanied by a new type of headache, fever, jaw claudication, and PMR symptoms, and frequently complicated by irreversible visual loss in the early stage of the disease, which is considered as a serious complication[1,2]. The incidence of this complication can be decreased by administration of corticosteroid in the early stage[3-5].

This patient showed a low level of RF, negative immune complex, and no decrease in the complement level, therefore vasculitis associated with RA was denied, although she had a 45-year history of RA. Additionally, there are few reports on GCA associated with RA, though PMR is known to be frequently accompanied by GCA[6]. As 3 of 5 items in the diagnostic criteria for GCA by 1990 ACR were

apparently fulfilled in this case, a diagnosis of GCA was made based on clinical findings; however a biopsy of the diseased part, *i.e.* STA is usually performed to obtain a definite diagnosis. It may be a requisite examination making a decision to initiate corticosteroid therapy, but in this case, it could not be carried out because the patient was in an extreme anxiety state and strongly refused any surgical treatments, and so only a scan of STAs by MR-angiography was performed. Arterial biopsy is actively pursued in GCA, because a biophy of a diseased blood vessel is relatively easily performed, differing, from that in Takayasu's arteritis affecting mainly the aorta and its major branches.

Although reports of Takayasu's arteritis on an early diagnosis or evaluation of corticosteroid therapy by MR imaging or angiography have been sporadically observed[7-9], recent remarkable advances in diagnostic imaging technology have enabled us to perform MR-angiography of a STA which has a very small diameter[10]. In this case, we used MR-angiography to evaluate the diseased blood vessels in the early phase after onset of GCA, then used subsequent MR-angiography to confirm the therapeutic effect after continuous treatment with corticosteroid. We presented here a case of demonstrating the usefulness of MR-angiography for diagnosis and treatment of GCA.

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MR-angiographyで経過を観察しえた慢性関節リウマチに合併した巨細胞動脈炎の1例

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要旨：慢性関節リウマチ治療経過中に巨細胞動脈炎（側頭動脈炎）を合併した1例を報告した。症例は抗リウマチ剤投与で良好にコントロールされていた慢性関節リウマチの57歳の女性で，強い両側頭部痛と全身倦怠感を主訴に来院した。アメリカリウマチ学会(ACR)の分類基準に基づき巨細胞動脈炎と診断し，MR-アンギオグラフィーで両側浅側頭動脈の狭窄性変化を強く疑い，発症早期よりステロイド投与を開始した。眼合併症等を来すことなく良好な経過をとり，治療開始4週間後のMR-アンギオグラフィー再検でその治療効果を確認し得た。MR-アンギオグラフィーで巨細胞動脈炎病変部を観察しえた報告は極めて少なく，文献的考察を加え報告する。

索引用語：巨細胞動脈炎，側頭動脈炎，慢性関節リウマチ，MRアンギオグラフィー