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CASE REPORT

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A case of giant cell arteritis simultaneously diagnosed with chronic subdural hematoma

Yuichiro Otani | **Keishi Kanno** | **Yuka Kikuchi** | **Takahiro Kametani** | **Tomoki Kobayashi** | **Susumu Tazuma**

Department of General Internal Medicine, Hiroshima University Hospital, Hiroshima, Japan

Correspondence
Yuichiro Otani, Department of General Internal Medicine, Hiroshima University Hospital, 1-2-3, Kasumi, Minami-ku, Hiroshima 734-8551, Japan.
Email: yuichiro-otani@hiroshima-u.ac.jp

Abstract

Here, we describe a case of giant cell arteritis (GCA) simultaneously diagnosed with chronic subdural hematoma. In this case, head to chest computed tomography angiography was useful for the diagnosis and treatment of GCA.

KEYWORDS

chronic subdural hematoma, computed tomography angiography, giant cell arteritis

1 | INTRODUCTION

Giant cell arteritis (GCA) is an important cause of secondary headache in elderly people and is characterized by chronic inflammation of large- and medium-sized vessels of unknown cause. Generally, GCA is diagnosed in accordance with the American College of Rheumatology (ACR) vasculitis classification criteria as follows: (a) age at disease onset >50years; (b) new headache; (c) temporal artery abnormality; (d) elevated erythrocyte sedimentation rate; and 5. abnormal artery biopsy findings. Three of the five criteria must be met for the clinical diagnosis of GCA, distinguishing it from other forms of vasculitis with a sensitivity of 93.5% and a specificity of 91.2%.[1]

Conversely, chronic subdural hematoma (CSDH) is regarded as a traumatic lesion and is most commonly caused by head injury. Consistent with this, a large study of 1000 patients revealed that 61.7% patients had head trauma experience prior to CSDH.[2] Typically, symptoms occur days or weeks after head injury (unnoticed or noticed) with drowsiness, headache, confusion, or mild hemiparesis; such symptoms occur most frequently in elderly people. The most common presentation in CSDH is a degree of altered mental status (50%–70% of patients). The reported incidence of headache varies among studies—ranging from 14% to 80%—but is generally lower among older people than among younger people, partially due to the large available intracranial space.[3] Headaches in patients with CSDH typically fluctuate in severity, sometimes with changes in head position.[4]

Here, we report the case of a 75-year-old Japanese man in whom GCA and CSDH were simultaneously diagnosed using computed tomography angiography (CTA). A pathological connection between GCA and CSDH may not exist; however, this combination of diseases was extremely rare and considered evocative to remind us of the variety of headache diagnosis and treatment of the elderly.

2 | CASE REPORT

A 75-year-old Japanese man presented to our hospital with a 2-month history of headache. He reported that the headache lasted the entire day, did not change according to the time of day or head position, and affected the entire head. He also complained of low-grade fever, general fatigue, and weight loss of 7 kg in the prior 2 months. His medical history included hypertension, diabetes, dyslipidemia, hyperuricemia, and insomnia. His medications were azilsartan, amlodipine besylate, bezafibrate, topiroxostat, eicosapentaenoic acid, brotizolam, trazodone, and diazepam. He smoked 1 pack of cigarettes per day and

consumed ≥500 mL of beer daily, but did not use illicit drugs. He had no memory of any recent head trauma or fall. He did not report jaw claudication, amaurosis fugax, or a history of polymyalgia rheumatica. On presentation, he was alert and oriented. His body weight was 55 kg, and height was 162 cm. His vital signs were as follows: blood pressure, 146/70 mm Hg; pulse rate, 95 beats per min; body temperature, 36.2°C; respiratory rate, 12/min; and oxygen saturation, 99% on room air. Physical examination revealed marked swelling of the temporal arteries with bilateral tenderness; he appeared otherwise normal. Meningeal signs were not observed. Neurological examination revealed muscle weakness of 4 to 5 in the right arm and a mildly positive Barre's sign; he was otherwise normal neurologically. His cognitive function was normal, and his score on the Mini-Mental Status Examination was 30/30. Because the patient's headache was new onset and progressively worsening accompanied with neurological deficit, following the American College of Radiology Appropriateness Criteria (ACR Appropriateness Criteria), contrast-enhanced head CT was planned.[5] However, marked dilatations and tenderness of both temporal arteries revealed on physical examination strongly implicated GCA and prompted us to widen the imaging range to the thoracic aorta. It revealed bilateral inflammatory changes in the surrounding soft tissue of the temporal arteries (Figure 1A,B) and circumferential wall thickening of the descending aorta (Figure 1C). Notably, CTA also revealed CSDH over the left cerebral hemisphere (Figure 2). Magnetic resonance imaging of the head, performed subsequently, did not reveal any newly developed brain infarction. Laboratory data were as follows: white blood cell count, 7660/μL; red blood cell count, 402×10^4/μL; platelet count, 41.7×10^4; C-reactive protein, 13.91 mg/dL; and erythrocyte sedimentation rate, 114 mm/h. Table 1 shows other test

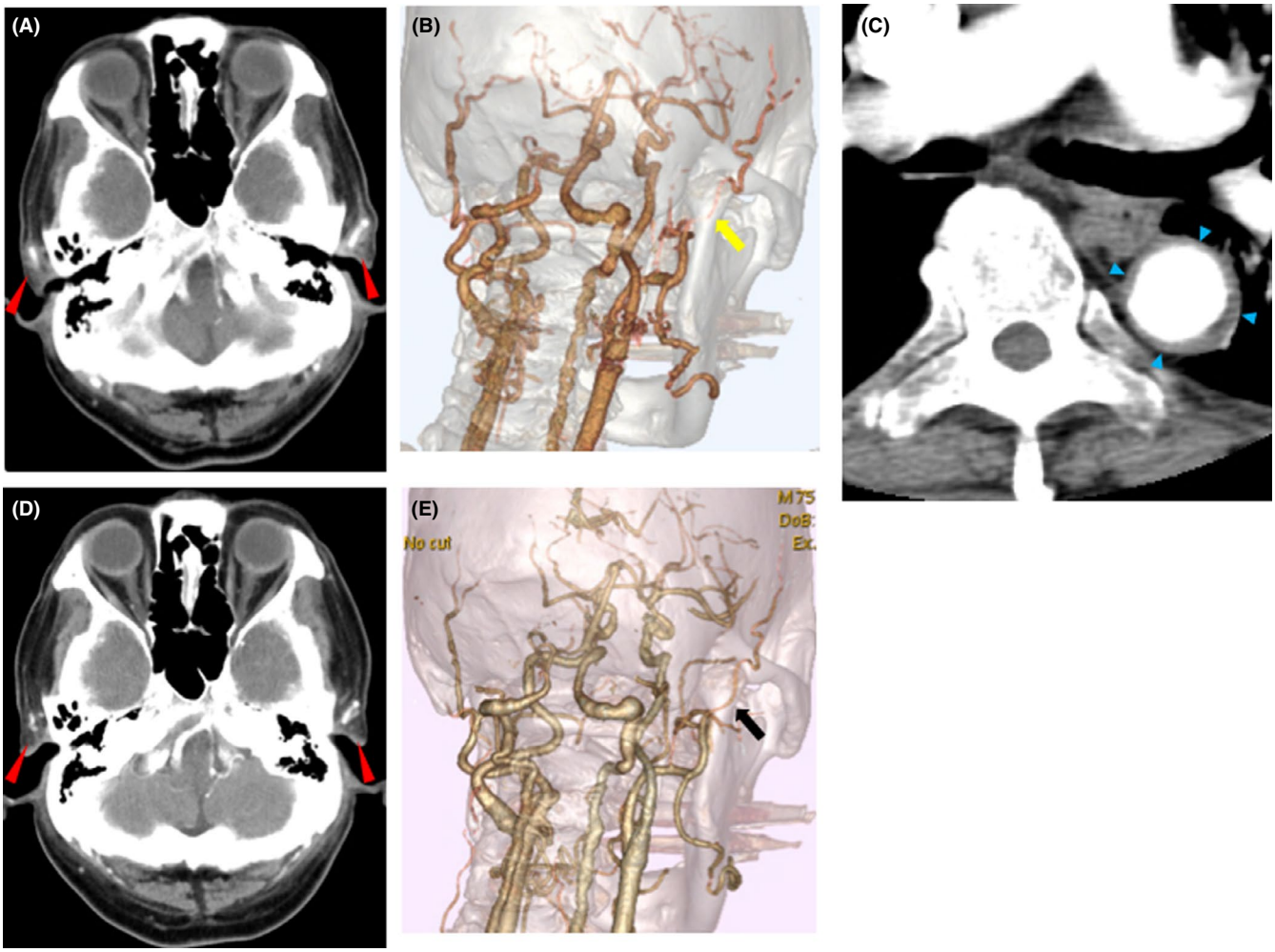

FIGURE 1 A, Transverse contrast-enhanced computed tomography (CT) image of the head obtained at the initial visit demonstrates wall thickening of bilateral temporal arteries with inflammatory changes in the surrounding soft tissue (arrowheads). B, Reconstructed three-dimensional (3D) computed tomography angiography image obtained at the initial visit demonstrates unilateral left temporal artery stenosis (arrow). C, Transverse contrast-enhanced CT image obtained at the initial visit demonstrates circumferential wall thickening of the descending aorta (arrowheads). D, Transverse contrast-enhanced CT image of the head, obtained 4 mo after the initiation of steroid treatment, demonstrates resolution of perivascular inflammation. E, Reconstructed 3D-CTA image, obtained 4 mo after the initiation of treatment, demonstrates decreased left temporal artery stenosis (arrow)

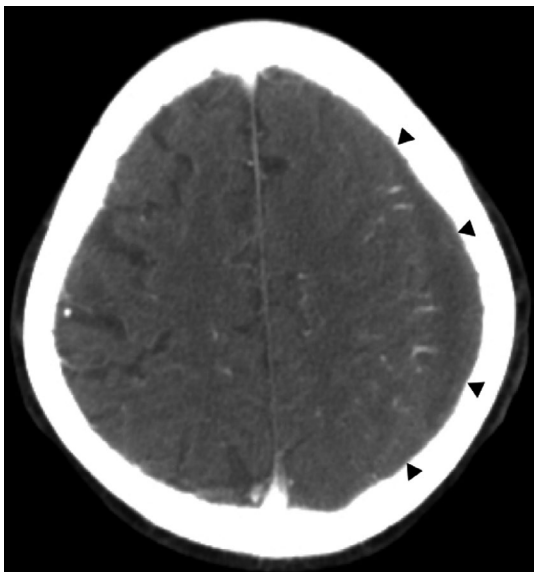

FIGURE 2 Transverse contrast-enhanced computed tomography image of the head obtained at the initial visit demonstrates liquefying homogenous hematoma, which extends from the left parietal lobe to the frontal lobe (arrowheads)

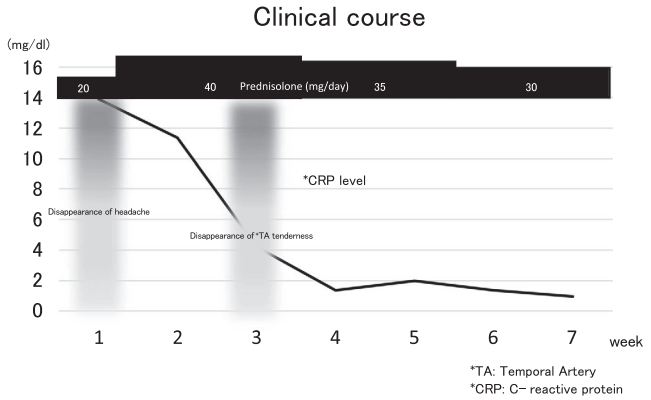

FIGURE 3 Clinical course of the patient presented in this report

results. Because the patient had met four of the five ACR classification criteria, we diagnosed him with GCA complicated by CSDH. Following consultation with a brain surgeon and an ophthalmologist, we chose to prioritize the treatment of GCA and postponed the surgical evacuation of hematoma because of the patient's mild neurological manifestations. Given that we had observed radiological findings of temporal artery and aortic inflammation, we began oral administration of prednisone without temporal artery biopsy (TAB). With reference to the Guideline for Management of Vasculitis Syndrome (Japanese Circulation Society 2008), we set the initial dose of prednisone as 40 mg.[6] However, this patient had diabetes and wished for outpatient treatment. We initially administered a lower dose of 20 mg to observe the pharmacological effect; this was increased to 40 mg 7 days later. Also, as described previously, this patient had diabetes mellitus, and his hemoglobin A1c level was 6.5% (National Glycohemoglobin Standardization Program); thus, dietary guidance, periodic blood glucose, and HbA1c level checks were scheduled. One week after the initiation of steroid administration, headache disappeared, so did the tenderness in 2 weeks. Fatigue and low-grade fever gradually disappeared, as inflammatory marker levels were subsequently decreased

TABLE 1 Laboratory data during the initial visit

Complete blood count		**Serological test**		**Urine**	
WBC	7660 x10^3/μL	CRP	13.91 mg/dL	occult blood	neg
N-Seg	77.7%	IgG	1075 mg/dL	ketone	neg
Eosino	0.3%	IgA	191 mg/dL	glucose	4+
Baso	0.6%	IgM	42 mg/dL	protein	1+
Mono	10.5%	ANA	<40	**Urinary sediment**	
Lymph	10.8%	MMP3	99 ng/mL	RBC	0-1 /HPF
RBC	402 x10^4/μL	PR3-ANCA	<1.0 U/mL	WBC	0-1 /HPF
Hemoglobin	12.5 g/dL	MPO-ANCA	<1.0 U/mL	Squamous cell	0-1 /HPF
Platelet	41.7 x10^4/μL	Anti-CCP antibody	<0.6 U/mL	cast	neg
Biochemistry		PCT	0.04 ng/mL		
AST	14 U/L	T-SPOT	negative		
ALT	7 U/L	RPR	<1.0		
LD	131 U/L	TPHA	x80		
CK	33 U/L	HCV Ab	0.03		
UN	10.4 mg/dL	HBs Ag	0 IU/mL		
Creatinine	0.72 mg/dL	**ESR**	114 mm/h		
Blood Sugar	157 mg/dL				
HbA1c	6.7%				

TABLE 2 Clinical characteristics of the reported cases of GCA complicated by CSDH

	case1 (9)	case2 (10)	this case
Age	75	68	75
Sex	male	female	male
Headache	yes	yes	yes
Known recent trauma	no	no	no
ESR (mm/h)	73	112	114
History of PMR	no	no	no
Hypertention	yes	yes	yes
Diabetes	unknown	yes	yes
Surgical evacuation of hematoma	yes	yes	no

(Figure 3). Three weeks after the initiation of steroid administration, complete response had been achieved, and we began steroid tapering thereafter. A computed tomography (CT) image and a three-dimensional contrast-enhanced CT reconstruction of the head obtained 4 months after the initiation of the steroid treatment demonstrated resolution of perivascular inflammation (Figure 1D) and decreased left temporal artery stenosis (Figure 1E). Thus far, we have not detected any subsequent exacerbation of the hematoma and GCA; moreover, the strength of the patient's right arm has recovered gradually, such that we have not yet performed surgical intervention for his CSDH. The patient is currently under a steroid tapering regimen.

3 | DISCUSSION

Although this patient could not recall previous head trauma, his medical history revealed a combination of several known risk factors for the development of CSDH, including old age, high alcohol consumption, diabetes, hypertension, and use of an oral antiplatelet agent.[2] Concerning two previously reported cases in which GCA was complicated by CSDH, the patients had a history of hypertension and/or diabetes (Table 2).[7,8] Considering the case reports describing intracranial hemorrhagic complications in patients with GCA were extremely rare, we believed that the etiology of CSDH in these patients, including our case, might be attributable to the substantial risk factors described above. Because the headache did not change according to the time of day or head position and head CT showed a relatively high degree of brain atrophy, we believe that the more likely cause of headache in this patient was GCA and not CSDH. There are no expert guidelines for the management of CSDH; however, in a series of 24 cases of CSDH, the authors concluded that age greater than 70 years, decreased cognitive level, brain atrophy, and the absence of increase of intracranial pressure are signs that enable the selection of conservative treatment.[9]

We initiated prednisone administration without performing TAB, in line with the patient's wish, because we had confirmed the presence of inflammatory changes in the temporal arteries and aorta from the findings of CTA images obtained at the initial visit. In this regard, a small case–control study of 14 patients has recently been performed to evaluate the diagnostic utility of head CTA for the diagnosis of GCA; it has reported a diagnostic accuracy of 78.6%, sensitivity of 71.4%, and specificity of 85.7%.[10] Importantly, the primary outcome in the above study was blurring of the superficial temporal artery vessel wall and enhancement of perivascular soft tissues, which are similar to the findings in our patient. Notably, in a retrospective evaluation of images obtained consecutively for the follow-up of patients with CSDH, even noncontrast-enhanced head CT could help identify a high-intensity area around the temporal arteries, which was alleviated as treatment progressed (Figure 4). Taken together, head to chest CTA is a useful imaging modality for the diagnosis and treatment of patients with GCA. When evaluating the head CT of elderly patients with headache, we must pay attention not only to the intracranial spaces but also to the temporal arteries and the surrounding tissue.

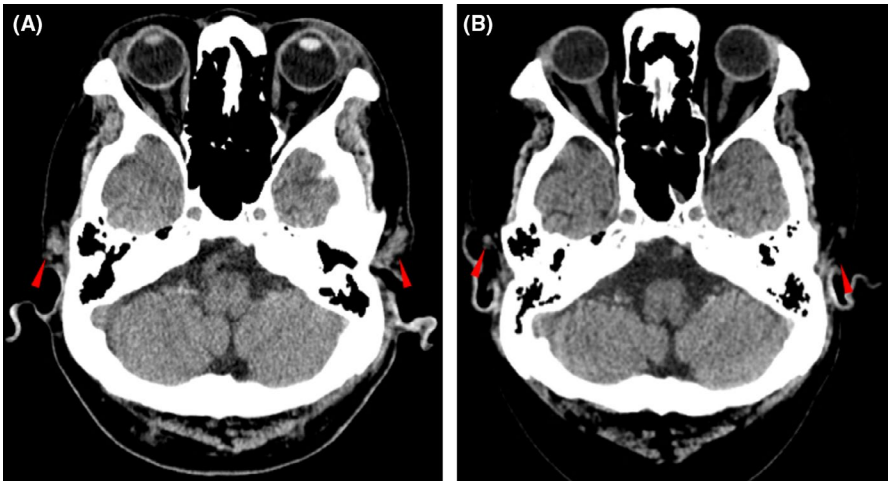

FIGURE 4 Transverse noncontrast-enhanced computed tomography images of the head showing pre- (A) and post-treatment (B) findings in the present case. Note that the high-intensity area around temporal arteries noted in the pretreatment image had nearly disappeared in the post-treatment image. White arrowheads indicate the temporal arteries and the surrounding tissue

4 | CONCLUSION

We presented the case of a 75-year-old Japanese man in whom GCA and CSDH were simultaneously diagnosed. Although we did not perform TAB, CTA performed at the initial visit showed inflammatory changes in bilateral temporal arteries, leading to the diagnosis of GCA; furthermore, a subsequently performed follow-up head CT was useful in reflecting the treatment effects. The formation of CSDH in this patient was attributable to the substantial risk factors exhibited by this patient and not to GCA. Because CSDH appeared to have caused only mild neurological symptoms, and had not disturbed cognitive function, we prioritized the treatment of GCA.

CONFLICT OF INTEREST

None declared.

AUTHORS CONTRIBUTION

YO: contributed to treat the patient and drafted the manuscript; KK, YK, TK, and TK: contributed to diagnose and treat the patient; and ST: critically reviewed the literature and involved in writing. All authors approved the final manuscript.

ORCID

Yuichiro Otani https://orcid.org/0000-0003-1884-591X

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