

Case Report

Clinical diagnostic utility of contrast-enhanced three-dimensional fluid-attenuated inversion recovery for selection of brain biopsy sites in neurosarcoidosis: A case report

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ABSTRACT

Neurosarcoidosis is difficult to diagnose, because definite diagnosis requires detailed histology of the central nervous system. Three-dimensional contrast-enhanced fluid-attenuated inversion recovery (CE-FLAIR) is more useful for detecting leptomeningeal lesions compared with 3D CE-T1 weighted imaging. However, the clinical diagnostic utility of 3D CE-FLAIR for neurosarcoidosis is unclear. We describe a case of a 46-year-old Japanese woman who was admitted to our department due to chronic headache with fever and diplopia. Using 3D CE-FLAIR, we performed brain biopsy from right cerebellar lesion. The histological examination revealed typical non-caseating granulomas, indicating neurosarcoidosis. Our findings suggest that 3D CE-FLAIR may detect leptomeningeal lesions that are candidates for biopsy in chronic meningitis undetermined etiology.

1. Introduction

Neurosarcoidosis is a rare neuroinflammatory granulomatous disease with various sequelae [1], and nervous system involvement occurs in about 5%–15% of patients with sarcoidosis [2]. The differential diagnoses for neurosarcoidosis include tuberculosis, mycosis, lymphoma, leptomeningeal metastases, and vasculitis [2]. Diagnosis is difficult, and requires detailed central nervous system histology.

Fluid-attenuated inversion recovery (FLAIR) imaging uses an inversion recovery pulse with an inversion time that nulls signal from the cerebrospinal fluid (CSF). During blood-brain barrier breakdown, intravenous injection of gadolinium chelates leads to increased signal inside the CSF on FLAIR images [3]. Leptomeningeal lesions caused by infective leptomeningitis and leptomeningeal carcinomatosis can be better detected on contrast-enhanced (CE) FLAIR than on CE T1-weighted imaging [4]. Therefore, CE-FLAIR is useful for assessing neoplasm- and infection-induced meningitis. However, the clinical diagnostic utility of CE-FLAIR for autoimmune leptomeningitis is unknown. We herein report a rare case of chronic leptomeningitis due to definite neurosarcoidosis, revealed via a brain biopsy conducted according to CE-FLAIR findings. The patient provided written informed consent for publication of this report.

2. Case report

A 46-year-old Japanese woman was admitted because of chronic headache with fever and diplopia. Four months previously, she had developed occipital headache and appetite loss. Three months previously, she had developed intermittent fever ($\geq 38^\circ\text{C}$), difficulty walking, nausea, and diplopia. Her primary care physician prescribed loxoprofen for the headache and fever. Her medical history and family history were unremarkable. On admission, her vital signs and physical examination were normal. Neurological examination revealed bilateral optic disc redness, decreased deep tendon reflex of the right biceps, and impairment of tandem standing/walking, but no neck stiffness. The Bielschowsky head-tilt test was positive only for tilting toward the left, indicating left trochlear nerve palsy. Visual acuity was 20/16 bilaterally. Bilateral plantar responses were flexor. All other findings were normal. The patient's laboratory results are shown in Table 1 (negative for general inflammatory response, electrolyte abnormalities, auto-antibodies, tumor marker elevation, and infection). Serum angiotensin-converting enzyme (ACE) and lysozyme concentrations were normal, whereas CSF analysis revealed pleocytosis, elevated ACE, elevated adenosine deaminase (ADA), and elevated $\beta 2$ -microglobulin. Oligoclonal bands were positive (two bands). Whole-body computed tomography showed mild mediastinal lymphadenopathy without pulmonary

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Table 1
Laboratory data on admission.

Hematology		Serology		CSF		Infection	
WBC (μL)	3800	ACE (IU/mL)	15.5	Pressure (mmH ₂ O)	180	Bacterial culture in CSF	Negative
RBC ($\times 10^4/\mu\text{L}$)	412	Lysozyme (IU/mL)	7.2	Cell (cells/ μL)	167	RPR	Negative
Hb (g/dL)	12.9	IgG (md/dl)	1000	Mononuclear (%)	100	TPHA	Negative
Platelet ($\times 10^4/\mu\text{L}$)	26.4	IgG 4 (md/dl)	59.5	Glucose (md/dl)	27	HIV Ab	Negative
ESR (mm/h)	10	sIL-2R (U/mL)	485	Protein (md/dl)	191	HSV DNA PCR in CSF	Negative
Biochemistry		CRP (md/dl)	0.1	ACE (IU/mL)	1.7	EBV DNA PCR in CSF	Negative
		ANA	Negative	ADA (U/L)	15.2	VZV DNA PCR in CSF	Negative
		anti-SS-A Ab	Negative	IgG index	0.73	β -D-glucan	Negative
		anti-SS-B Ab	Negative	β 2-MCG (md/dl)	11.5	Aspergillus IgG Ab	Negative
		anti-ds-DNA Ab	Negative	OB	Positive	Aspergillus antigen	Negative
		anti-Sm Ab	Negative	Cytology	Negative	Candida antigen	Negative
		anti-RNP Ab	Negative	Tumor Marker		Fungal culture in CSF	Negative
		MPO-ANCA	Negative			Cryptococcal antigen in CSF	Negative
		PR3-ANCA	Negative				
TP (g/dL)	7.3	Urinalysis		CEA (ng/mL)	2.3	Mycobacterium tuberculosis	
Alb (g/dL)	4.5			AFP (ng/mL)	1.6		
T-Bil (mg/dL)	1.11			CA19-9 (U/mL)	3.7		
AST (IU/L)	18			SCC (U/mL)	0.4		
ALT (IU/L)	24			NSE (ng/mL)	14.2		
ALP (IU/L)	140			CYFRA (ng/mL)	0.5	CSF culture	
γ -GTP (IU/L)	33			SLX (U/mL)	25.9		
LDH (IU/L)	183			Pro-GRP (pg/mL)	22.3		
BUN (md/dl)	11.8						
Cr (md/dl)	0.49						
Calcium (md/dl)	9.7	Calcium (mg/day)	277				
Glucose (md/dl)	124						
β 2-MCG (md/dl)	1.2						

Laboratory data on admission. WBC: white blood cell, RBC: red blood cell, Hb: hemoglobin, TP: total protein, Alb: albumin, T-Bil: total bilirubin, AST: aspartate aminotransferase, ALT: alanine aminotransferase, ALP: alkaline phosphatase, γ -GTP: γ -glutamyl transpeptidase, LDH: lactate dehydrogenase, BUN: blood urea nitrogen, Cr: creatinine, ACE: angiotensin-converting enzyme, sIL-2R: soluble interleukin-2 receptor, CRP: C-reactive protein, Ig: immunoglobulin, ANA: antinuclear antibody, Ab: antibody, MPO-ANCA: myeloperoxidase-antineutrophil cytoplasmic antibody, PR3-ANCA: proteinase 3-antineutrophil cytoplasmic antibody, ADA: adenosine deaminase, β 2-MCG: beta2-microglobulin, OB: oligoclonal band, CEA: carcinoembryonic antigen, AFP: alpha-fetoprotein, CA19-9: carbohydrate antigen 19-9, SCC: squamous cell carcinoma antigen, NSE: neuron-specific enolase, CYFRA: cytokeratin 19 fragment, SLX: sialyl Lewis Xi antigen, Pro-GRP: pro-gastrin-releasing peptide, RPR: rapid plasma regain, TPHA: *Treponema pallidum* hemagglutination assay, HIV: human immunodeficiency virus, HSV: herpes simplex virus, EBV: Epstein-Barr virus, VZV: varicella zoster virus, IGRA: interferon-gamma release assay.

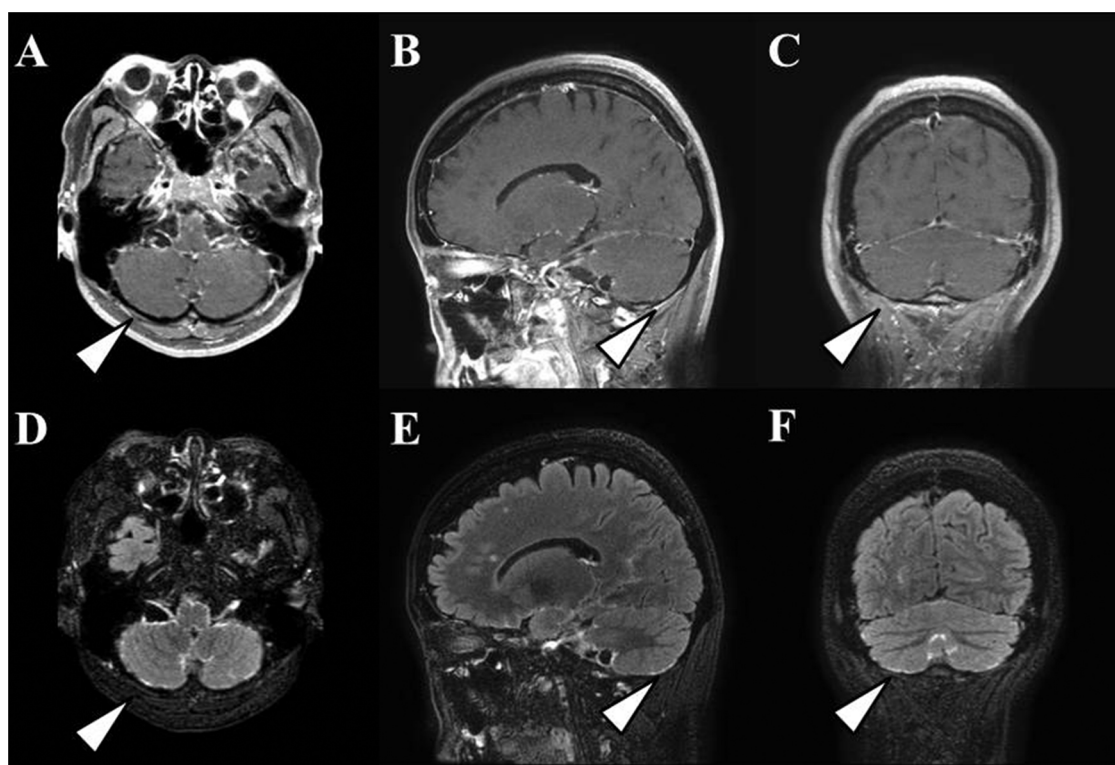


Fig. 1. Brain magnetic resonance imaging. Unlike three-dimensional (3D) contrast-enhanced (CE)-Cube T1-weighted imaging (A–C), 3D CE-Cube FLAIR imaging (D–F) revealed possible bilateral cerebellar leptomeningeal lesions. Arrowheads indicate the biopsied leptomeningeal lesion.

abnormalities. Gallium-67 scintigraphy revealed no abnormalities. Brain magnetic resonance imaging (MRI) 1 day after admission revealed gadolinium-enhancing meningeal lesions, primarily on the posterior cranial fossa, thereby indicating basal meningitis (Fig. 1A–F). Three-

dimensional CE-FLAIR (Fig. 1D–F) more clearly visualized the cerebellar meninges lesions than did 3D CE-T1-weighted imaging (Fig. 1A–C). Furthermore, diffusion-weighted imaging revealed a hyperintense white matter lesion in the right hemisphere. The MRI

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