General Paresis with Reversible Mesial Temporal T2-weighted Hyperintensity on Magnetic Resonance Image: A Case Report

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Abstract- We report a patient with general paresis, whose magnetic resonance image (MRI) showed a T2 high-intensity lesion in bilateral mesial temporal regions. Serum rapid plasma reagin test showed reactive at 64 dilutions and serum Treponema pallidum haemagglutination test was 1:20480. Cerebrospinal fluid analysis showed: RBC 111/mm^3, WBC 8/mm^3, Venereal Disease Research Laboratory reactive at 1 dilution and protein 60 mg/dl. His neuropsychiatric symptoms recovered gradually after penicillin treatment two months later. Repeated MRI revealed resolution of the bilateral mesial temporal lesions. We demonstrated the first Taiwanese patient with general paresis whose clinical improvement was associated with the disappearance of the temporal lobe MRI abnormality. The diagnosis of neurosyphilis must be considered when MRI shows mesial temporal lesions. MRI may be used to predict prognosis in patients with general paresis.

Key Words: Neurosyphilis, General paresis, Magnetic resonance image

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INTRODUCTION

General paresis, one form of neurosyphilis, is characterized by psychiatric symptoms, dementia, and mental changes. The association of MRI findings and clinical course in general paresis has been reported[1,2]. Most common MRI findings were cortical atrophy and gliosis. Few case reports presented lesions in medial temporal areas. We report a patient with general paresis whose MRI showed bilateral temporal abnormalities that were resolved after treatment of antibiotics.

CASE REPORT

A 52-year-old man presented with emotional disturbance for nine months including an irritable mood, a suspicious attitude, hyper-talkativeness and hyperactivity. Two months before admission, he experienced progressive memory impairment, urinary incontinence and incoherent speech. Neurologic examination showed disorientation to time, place, and person. Impaired recent memory, poor attention, acalculia and dysarthria were also noted. He had normal pupils, light reflexes, gait,

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muscle power and deep tendon reflexes. There were no cerebellar or sensory dysfunctions.

The results of routine blood chemistry and complete blood counts were normal. Serum rapid plasma reagin (RPR) was reactive in 64 dilutions. The CSF showed positive Venereal Disease Research Laboratory (VDRL) (reactive in 1 dilution, 1 DIL) and negative polymerase chain reaction (PCR) for herpes virus and mycobacterium tuberculosis. The level of serum Treponema pallidum haemagglutination (TPHA) was 1:20480. The serum levels of vitamin B12, folate and free T4 were normal and the human immunodeficiency virus (HIV) test by enzyme-linked immunosorbent assay (ELISA) was negative. The laboratory tests are summarized in Table 1.

Fluid-attenuated inversion recovery (FLAIR) imaging showed high-intensity lesions in bilateral mesial temporal regions (Fig. 1A). T1-weighted MRI revealed cortical atrophy without post-contrast enhancement (Fig. 1B). Diffusion-weighted image showed high-intensity in bilateral mesial temporal regions (Fig. 1C).

The patient received penicillin G (24 MU/day) therapy for two weeks. His neuropsychiatric symptoms

Table. *Laboratory data*

	On admission	8 months later
CSF		
RBC	111/cumm	3/cumm
WBC	8/cumm (N/L: 4/4)	1/cumm (lymphocyte)
Protein	60 mg/dl	60 mg/dl
Sugar	67 mg/dl	123 mg/dl
Indian Ink stain	Negative	
Acid-Fast stain	Negative	
Fungus culture	No growth	
TB culture	No growth	
VDRL	Reactive at 1 dilution	Non-reactive
Blood		
RPR	Reactive at 64 dilutions	Reactive at 16 dilutions
TPHA	1:20480	1:5120
HIV	Negative	
Vitamine B12	189 pg/ml (130-785 pg/ml)	
Folate	3.8 ng/ml (3-17 ng/ml)	
FT4	9.98 pg/ml (7-19 pg/ml)	
TSH	1.35 uIU/ml (0.4-4 uIU/ml)	

CSF: cerebrospinal fluid; WBC: white blood cell; N/L: neutrophil/lymphocyte; VDRL: Venereal Disease Research Laboratory; RPR: Rapid plasma reagin; TPHA: Treponema pallidum haemagglutination; HIV: human immunodeficiency virus; FT4: free T4; TSH: thyroid stimulating hormone.

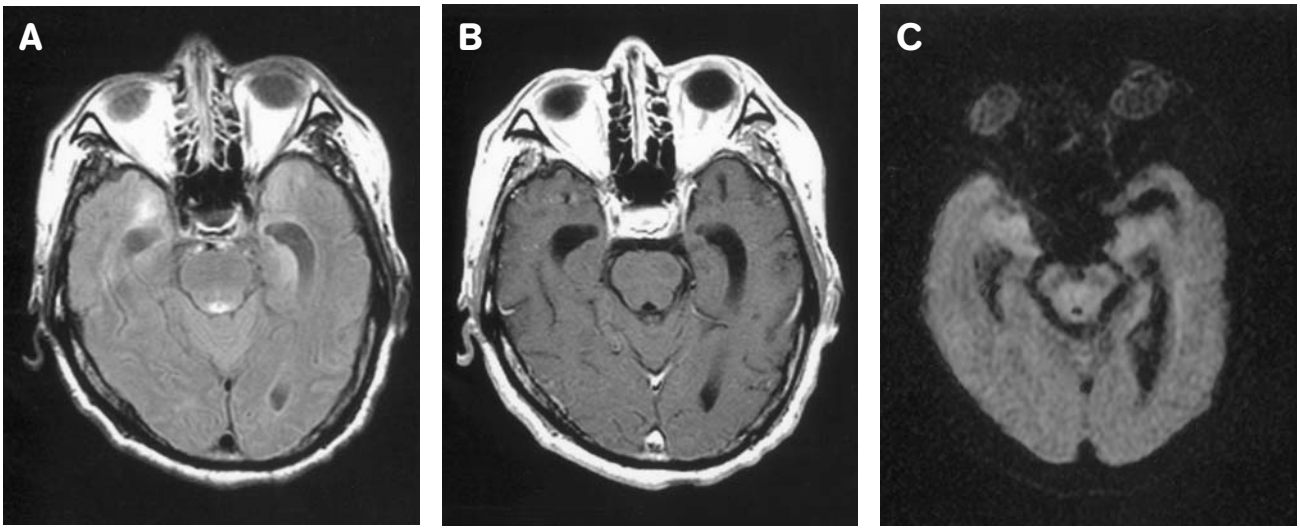

Figure 1. On admission. (A) Axial FLAIR imaging revealed high-intensity lesions over bilateral mesial temporal regions. (B) Axial T1-weighted imaging showed no contrast enhancement. (C) Diffusion-weighted imaging showed high-intensity lesions over bilateral mesial temporal regions

improved greatly. His speech became coherent and his mood was not irritable. He became orientated, but still had short-term memory impairment. Mini-Mental Status Examination (MMSE) score was 28 points. He was discharged and resumed his job as a truck driver. Two months later, a follow-up MRI showed that the previous lesions almost disappeared (Fig. 2). Eight months after admission, repeated serum RPR was reactive in 16 dilutions. MRI showed that bilateral mesial temporal lesions diminished greatly (Fig. 3).

DISCUSSION

MRI findings in patients with general paresis have been reported[2,3]. Cortical atrophy was the most common presentation, including the mesial temporal lobes. Drug therapy was reported effective if the severity of the cortical atrophy was only moderate. Whereas, severe cortical atrophy, especially in the mesial temporal lobes, signal loss in the basal ganglia or high-intensity T2 lesions in subcortical regions were reported to indicate poor prog-

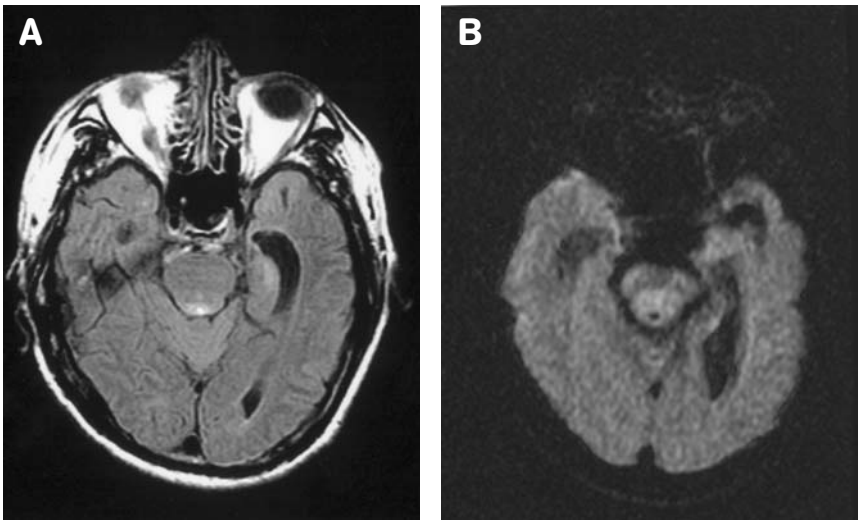

Figure 2. Two months after admission. (A) Axial FLAIR imaging, (B) Diffusion-weighted imaging, both revealed that previous high-intensity lesions over bilateral mesial temporal regions almost completely resolved.

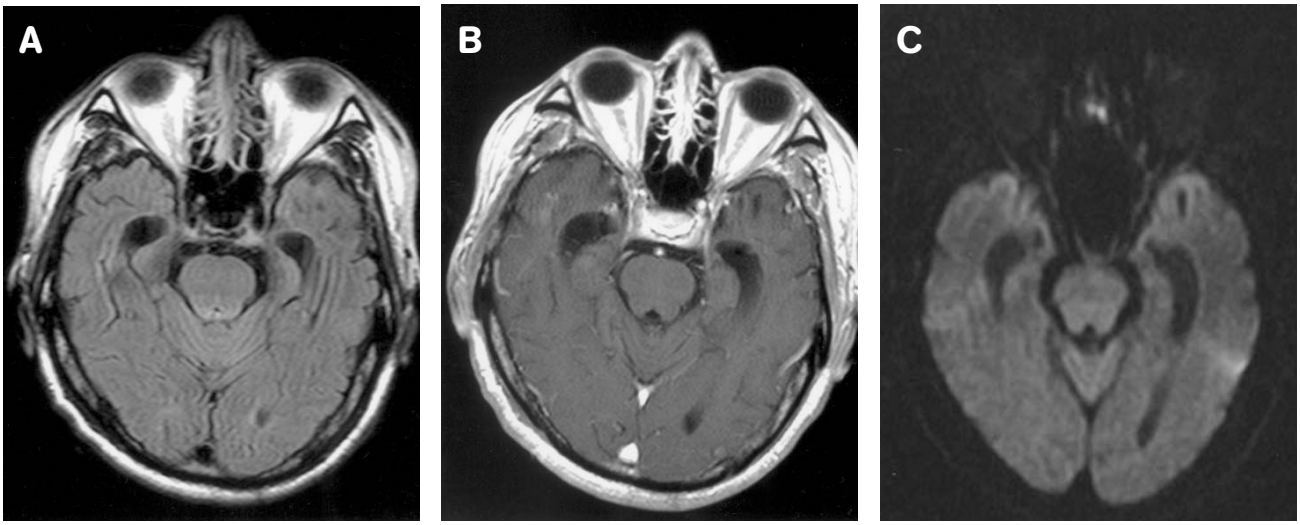

Figure 3. Eight months after admission. (A) Axial FLAIR imaging and (B) T1-weighted imaging post-contrast and (C) Diffusion-weighted imaging all showed normal appearance over bilateral mesial temporal regions.

nosis. Our patient's MRI showed high-intensity T2 lesions and diffusion-weighted images without contrast enhancement over bilateral mesial temporal regions. In contrast, he did not have any poor prognostic MRI findings. In line with previous studies, our patient showed a good prognosis[2,3]. Herpes simplex encephalitis and paraneoplastic limbic encephalitis were ruled out based on the clinical course and laboratory data.

Meningovascular lesions are more common in patients with neurosyphilis, including cortical or subcortical infarctions, leptomeningeal enhancement, meningitis and arteritis, but none of these involved limbic lesions[4,5]. Cases with reversible high signal T2 lesions in the mesial temporal region had also been reported[6-9]. Of these cases, one showed a transient global amnesia-like attack[7] without returning to normal mental state, and later progressed to dementia. One month after antibiotics treatment, the MRI signal abnormalities improved. Another patient presented with complex partial status epilepticus with bilateral mesial temporal lesions on MRI[9]. A second brain MRI showed a residual right mesial temporal high-intensity lesion one month after carbamazepine treatment. The first bilateral mesial temporal high signals might be due to transient edema caused by seizures[10]. The residual T2 hyperintense lesion might have a vasculitic origin, which may be responsible for seizures. Two other cases presented with a similar clinical course and imaging changes as in our case[6,8]. In one of the four cases reported by Zifko et al., MRI showed atrophy and gliosis over bilateral hippocampi[2].

In our patient, the medial temporal lesion may be due to reversible microglial hypertrophy, cytotoxic and vasogenic edema before permanent damage, which resulted in atrophy and gliosis[6,8]. Contrast enhanced T1-weighted images did not show abnormal findings. This implies that that there was no encephalitis, which might occur at mesial temporal regions in patients with herpes simplex infection or paraneoplastic limbic encephalitis. In the previous reported hyperintense mesial temporal lesions in patients with syphilis infection, they didn't present contrast enhanced images. With meningitis, permeability of the blood-brain-barrier may be increased, which causes vasogenic edema. Small vessels may be involved, which causes ischemic cytotoxic edema and in more severe cases, gliosis, such as the case reported by Marano et al.[9]. There is no strong evidence of meningovasculitic involvement or parenchymal inflammation as seen in general paresis attributing to the abnormal findings on MRI. However microglial hypertrophy is pathologically seen in the cerebral cortex in general paresis[6]. In a review of the current literature, limbic lesions have not been described in syphilitic meningovasculitis[4,5]. To sum up, the high-intensity T2-weighted findings in neurosyphilis may be due to cytotoxic, vasogenic edema, inflammation, meningovasculitis, or microglial hypertrophy. When disease progresses without treatment, irreversible infarction or gliosis may occur. Since a repeated MRI showed reversible high-intensity T2-weighted findings without obvious atrophy, it was reasonable that our patient had a good recovery after adequate antibiotics treatment.

There is no reliable marker to predict the outcome in patients with general paresis after treatment. This is the first case report of a Taiwanese patient with general paresis, showing reversible MRI abnormality in the temporal lobe. Further observations will answer whether clinical improvement is always associated with disappearance of such MRI lesions. Reversible MRI lesions in patients with general paresis after treatment correlate to clinical improvement, which should alert the physicians that early diagnosis and aggressive treatment are worthwhile. We suggest that MRI may be repeated in patient presenting high-intensity T2-weighted lesions in mesial temporal regions for differential diagnosis and outcome prediction.

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