



Case Report

Isolated Abducens Nerve Palsy Due to Anterolateral Pontomedullary Infarction in a Young Adult

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Abstract

A 24-year-old woman suffered from headache for several days, and then binocular diplopia occurred. On examination, she demonstrated signs of right abducens nerve palsy. General examination was unremarkable. A detailed neurological examination was normal, revealing no evidence of brain stem dysfunction. Brain magnetic resonance imaging (MRI) demonstrated hyperintensities in the right upper anterolateral medulla on T2-weighted images and diffusion-weighted images. Her diplopia resolved in 2 months. Ophthalmological examination revealed complete recovery. Follow-up MRI revealed hyperintensities in the right anterolateral lower pons and upper medulla. A complete MRI study should be considered in nontraumatic isolated abducens palsy, which includes thin sections through the nucleus and fascicle of the abducens nerve. (*Tzu Chi Med J* 2008; 20(2):136–139)

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1. Introduction

Isolated abducens nerve palsy is the most frequently encountered ocular motor nerve palsy (1,2). Although vascular causes of this palsy are suspected in a number of patients (1,2), focal brainstem infarction as a cause of isolated abducens palsy is rarely reported, with reported infarctions confined to the pontine tegmentum or anterolateral pons (3–7). We present a case of acute isolated abducens palsy due to anterolateral pontomedullary infarction, which was confirmed by brain magnetic resonance imaging (MRI).

2. Case report

A 24-year-old woman suffered from headache for several days, and then blurred vision developed, which made her unable to drive. One week later, binocular diplopia occurred. She denied having any other neurological symptoms. Her past medical history was unremarkable. Ophthalmological examination revealed normal visual acuities, color vision, visual fields, pupillary reactions, and eyelids. There was no proptosis. The patient demonstrated signs of a right sixth nerve palsy with rapid saccades in all directions except on

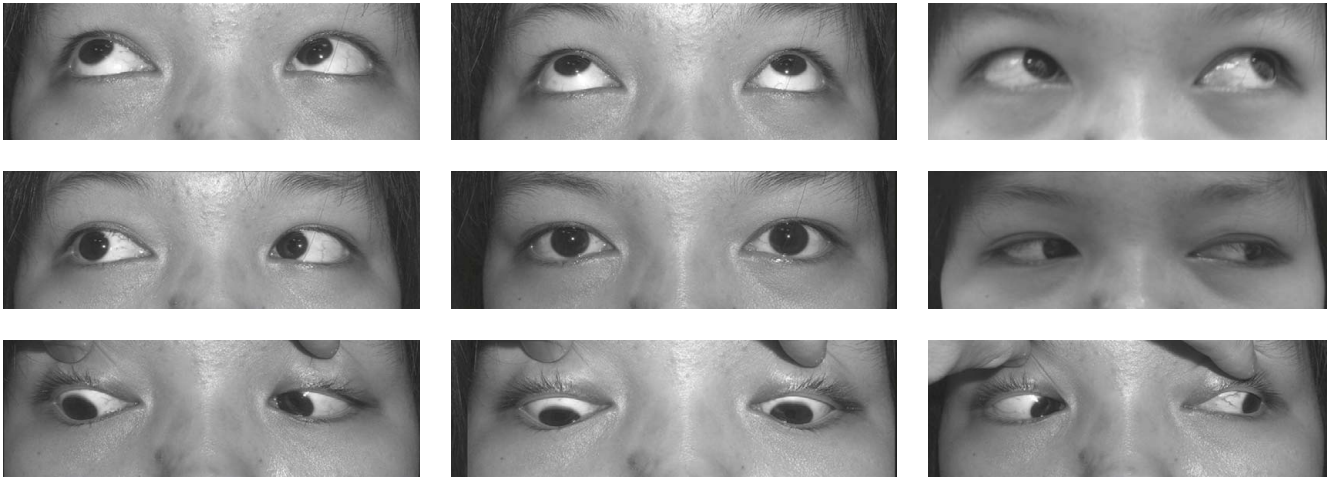


Fig. 1 — The patient demonstrated signs of a right sixth nerve palsy with partial limitation of abduction in the right eye during right lateral gaze. A prism cover test revealed an esotropia of 25 prism diopters in the primary position, which increased to 35 prism diopters on right gaze and decreased to 8 prism diopters on left gaze.

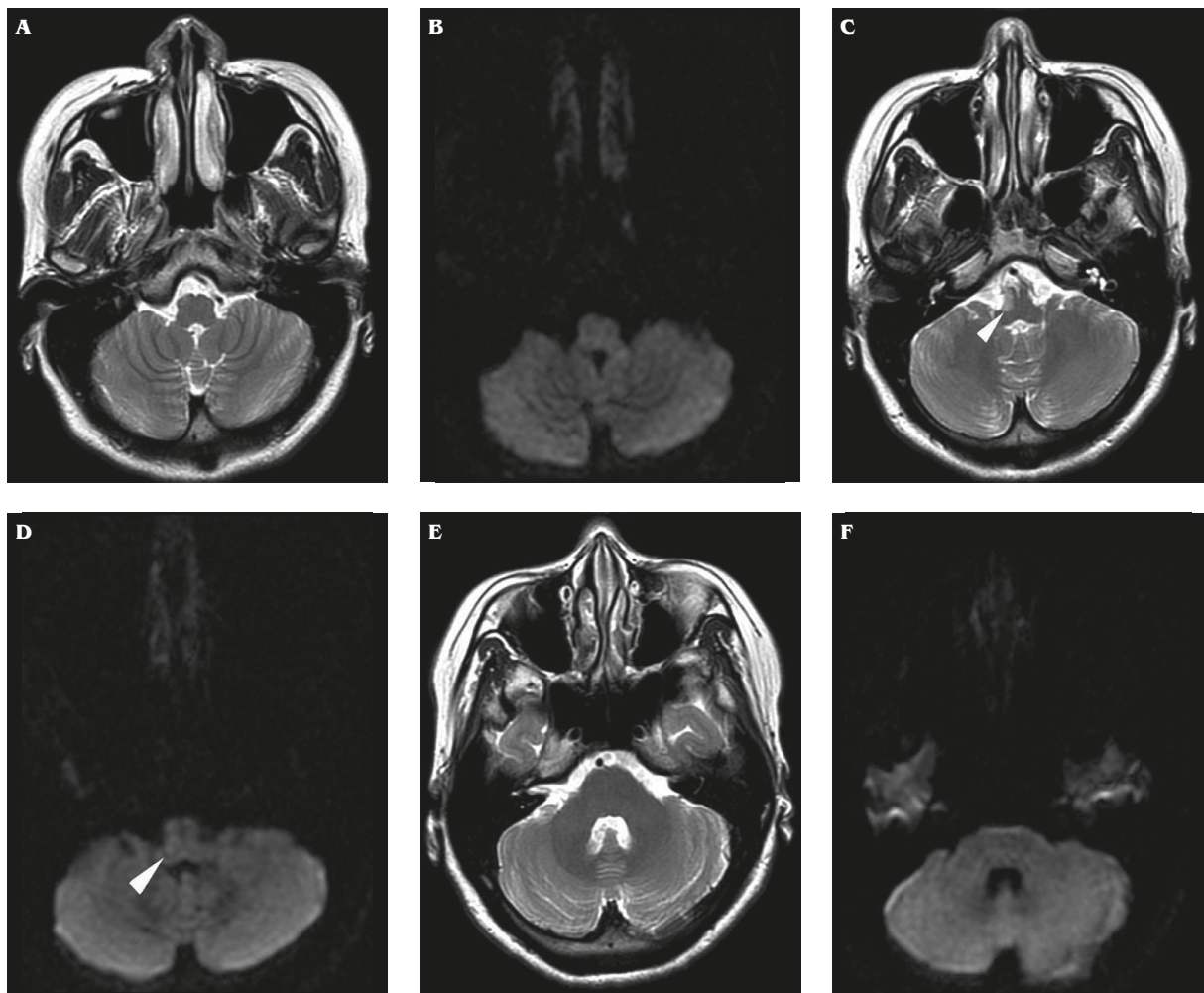


Fig. 2 — Brain magnetic resonance imaging (axial section, 5 mm in thickness) demonstrated hyperintensities in the right upper anterolateral medulla on T2-weighted images and diffusion-weighted images (arrowhead on Figs. C and D). The previous and next sections did not show any abnormalities (A, B, E, F).

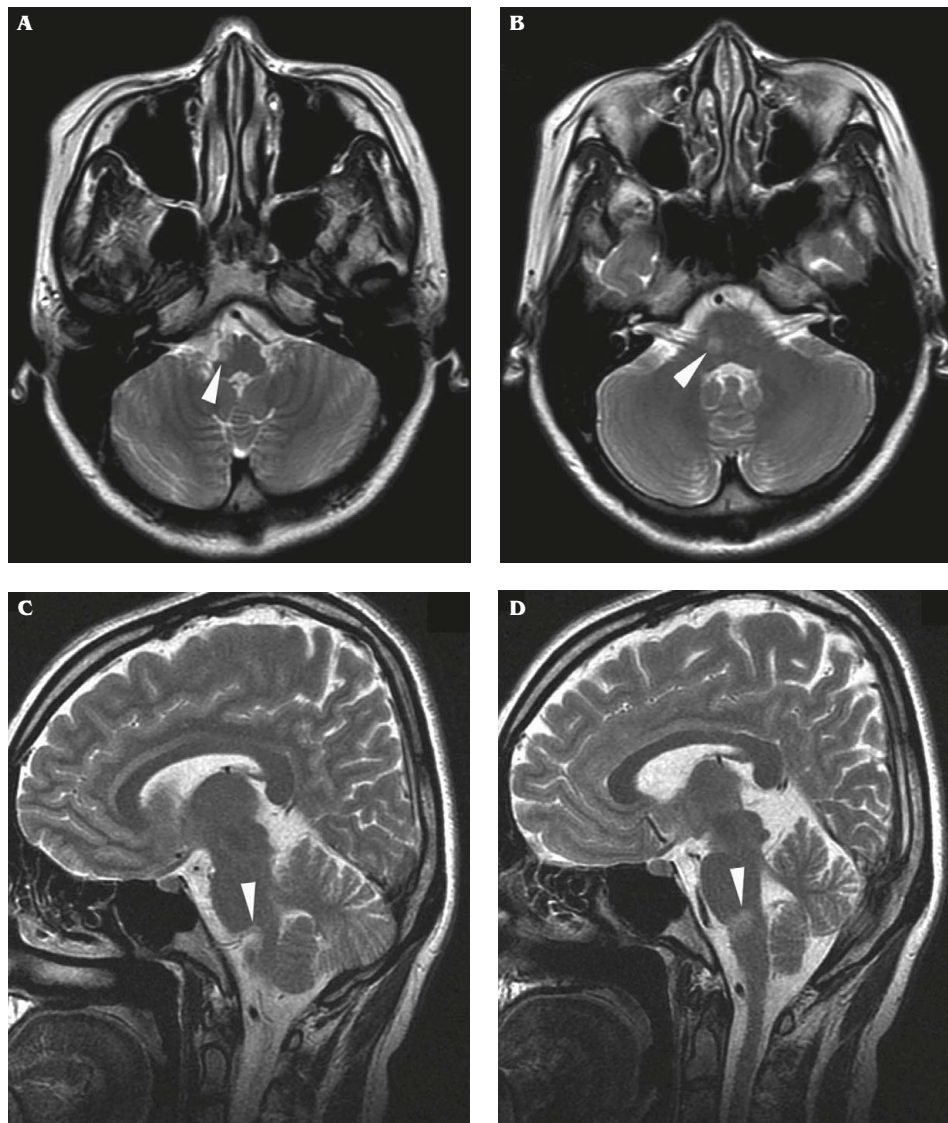


Fig. 3 — Follow-up magnetic resonance imaging revealed hyperintensities in the right anterolateral upper medulla (arrowhead on Fig. A) and lower pons (arrowhead on Fig. B), which explained the abducens palsy. T2-weighted sagittal sections helped illustrate the true infarction area (arrowhead on Figs. C and D).

right gaze, in which abduction of the right eye was slow. A prism cover test revealed an esotropia of 25 prism diopters in the primary position which increased to a 35 prism diopter esotropia on right gaze. There was only an 8 prism diopter esotropia on left gaze (Fig. 1). There was no pain on ocular movement. General and nasopharyngeal examinations were unremarkable. A detailed neurological examination was normal, revealing no evidence of brainstem dysfunction. Brain MRI demonstrated hyperintensities in the right upper anterolateral medulla on T2-weighted images and diffusion-weighted images (Fig. 2). Magnetic resonance angiography, electrocardiography, echocardiography and laboratory investigations were negative.

Her diplopia resolved in 2 months. Ophthalmological examination revealed complete recovery. Follow-up MRI

revealed hyperintensities in the right anterolateral lower pons and upper medulla (Fig. 3). She was followed up for 1 year and free of symptoms.

3. Discussion

Most previously reported cases of isolated abducens palsy show a small infarction in the tegmental pontine region (3–6); only one reported case illustrated an anterolateral pontine syndrome, presumably affecting the intrapontine abducens nerve fascicle (7). In our patient, MRI revealed hyperintensities in the right anterolateral pontomedullary area. Although with such areas involved, our patient presented with isolated abducens palsy. The common symptoms of rostral lateral

medullary infarction or olivary infarction, such as cerebellar ataxia, Horner's sign or sensory symptoms (8,9), were not seen in our patient.

Several reports described sudden deaths after medullary brain lesions, and some patients were considered neurologically normal at the time of sudden death (10–12). Since respiratory and cardiovascular "centers" are commonly believed to be in certain regions in the reticular formation of the brainstem, there is a possibility that an ischemic penumbra might affect cardiac and respiratory centers. It is therefore important to check for brainstem infarction and be aware of the possible severe complications. In our patient, the dimension of the stroke was not fully revealed on the first MRI study because of thicker axial sections and the lack of a T2-weighted sagittal view. If not for the involvement of such a large area, the infarction might have been missed. Follow-up MRI demonstrated the pontomedullary infarction which caused the abducens palsy. Therefore, we suggest that a complete MRI study should be considered in nontraumatic isolated abducens palsy, which includes thin sections through the nucleus and fascicle of the abducens nerve.

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