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Case report

Two cases of traumatic head injury mimicking acute encephalopathy with biphasic seizures and late reduced diffusion

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Abstract

Acute encephalopathy with biphasic seizures and late reduced diffusion (AESD) presents a distinct clinical course of biphasic seizures and impaired consciousness. These symptoms are followed by reduced diffusion in the subcortical white matter on magnetic resonance imaging that is typically observed between 3 and 9 days after illness onset. Here, we report two cases of traumatic head injury with clinical and radiological features similar to those for AESD. The similarities between our cases and AESD may be useful in understanding the pathogenesis of AESD.

Keywords: AESD; Head injury; Acute encephalopathy

1. Introduction

Acute encephalopathy is a generic term for acute brain dysfunction that is usually related to viral infection. The most frequent acute encephalopathy syndrome in Japan is acute encephalopathy with biphasic seizures and late reduced diffusion (AESD) [1,2]. The clinical course and magnetic resonance imaging (MRI) findings in AESD show unique features. These include a prolonged febrile seizure developing within 24 h of fever onset and a second seizure with consciousness disturbance occurring 4–6 days after recovery from the initial seizure. MRI usually shows no abnormalities during the first 2 days following injury, but reduced diffusion in the subcortical white matter appears around the time of the second seizure [3]. On the other hand, it is well established that children with mild head injuries sometimes deteriorate following a lucid interval [4]. Some case reports including head injuries describe late reduced diffusion upon MRI [5,6]. However, cases with head injury showing both a clinical course of second seizures and radiological features similar to those for AESD have not yet been reported. Here, we report two cases of traumatic brain injury with both clinical and radiological features similar to those for AESD.

2. Case reports

2.1. Case 1

A previously healthy 9-month-old boy fell backwards and sustained a head injury. He experienced a generalized convulsion for 1 min and subsequently became unconscious. Upon arrival to a local hospital, he showed severe consciousness disturbance (Glasgow Coma Scale [GCS] of 4; E1, V1, M2) and was intubated. Brain computed tomography (CT) showed an acute subdural hematoma around the cerebral falx, but the hematoma was not treated surgically because a subsequent CT

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failed to indicate hematoma expansion. The patient's consciousness (GCS of 13) improved 6 h after the injury, and therefore, he was extubated. He did not fully recover, however, and his convulsions continued in clusters, which were controlled by intravenous phenytoin administration. On day 2, he was transferred to our hospital because of prolonged unconsciousness.

On admission, his body temperature was 38.6 °C and heart rate was 155/min. His respiratory state was stable. He showed no bruises, burns, or fractures on whole-body examination. A laboratory examination revealed mild inflammation (white blood cell, 15 500/μL; C-reactive protein, 2.82 mg/dL) with anemia and disordered coagulation (hemoglobin, 5.9 g/dL;

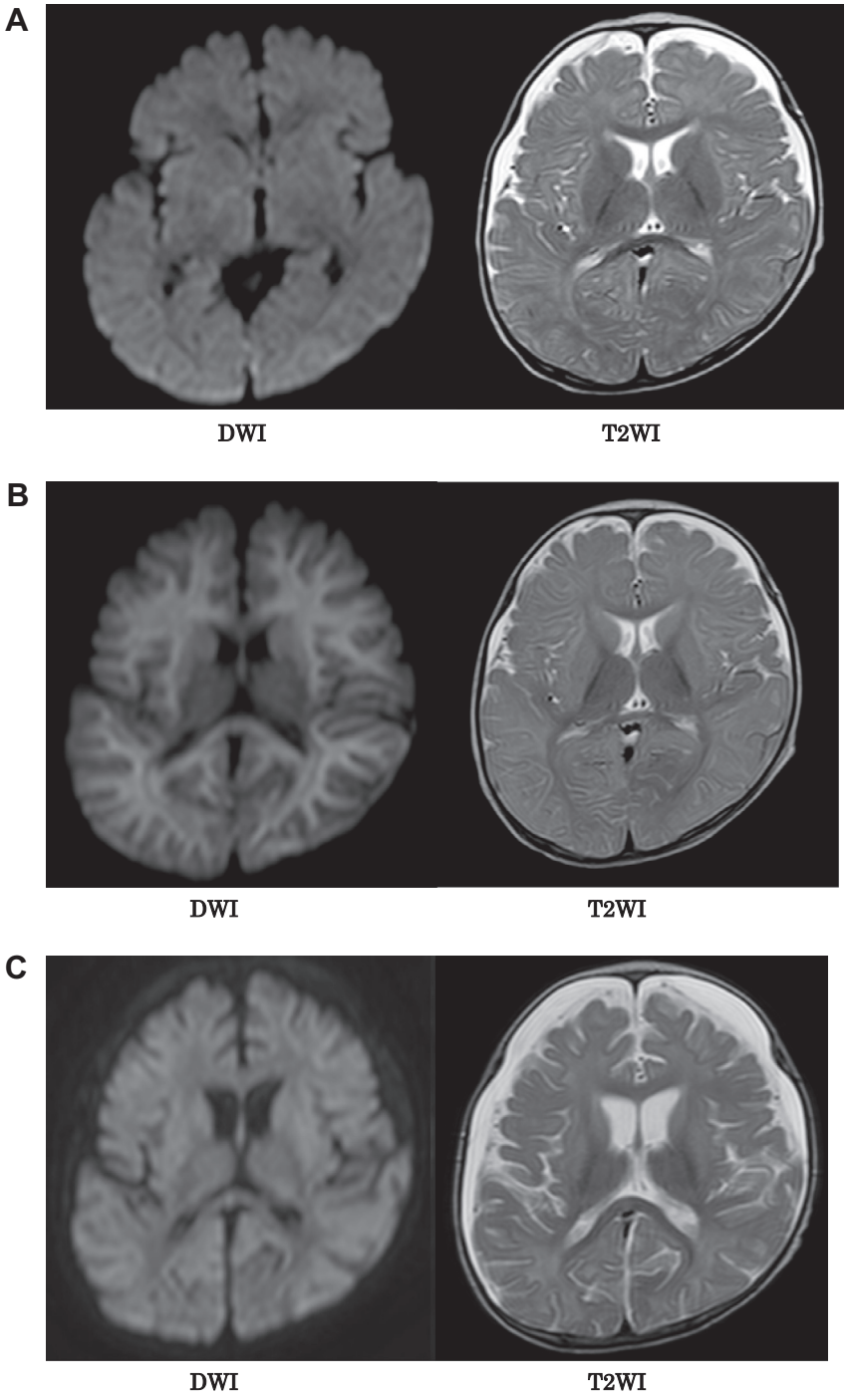

Fig. 1. (A) Brain magnetic resonance imaging (MRI) performed on admission failed to identify parenchyma injury. DWI, diffusion-weighted image; T2WI, T2 weighted image. (B) MRI performed on day 5. Note the hyperintensity in the deep subcortical white matter. (C) MRI performed on day 19. The hyperintensity on DWI resolved, but diffuse cerebral atrophy was observed.