MRI Findings of Acute Hippocampal Disorders: Pictorial Essay

급성기 해마 질환의 자기공명영상 소견: 임상화보

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The hippocampus is one of the most metabolically active regions of the brain; therefore, it may be affected by various acute disorders. This study aimed to introduce and categorize various acute conditions that can involve the hippocampus and explain the findings of MRI, especially diffusion-weighted imaging (DWI). Acute hippocampal disorders are divided into six categories: infection, inflammation, metabolic, ischemic, traumatic, and miscellaneous. In this study, patients were retrospectively reviewed based on clinical findings and MRI, especially DWI. All diseases had been confirmed clinically or pathologically. Many acute hippocampal disorders overlap with the clinical manifestations. Thus, it is necessary to categorize acute hippocampal lesions and understand their specific imaging findings for differential diagnosis.

Index terms Hippocampus; Acute Disease; Magnetic Resonance Imaging; Diffusion Magnetic Resonance Imaging

INTRODUCTION

The hippocampus, a section of the limbic system, plays a critical role in learning, memory, and emotional behavior (1) and is vulnerable to acute neurological disorders.

The hippocampus is located in the inner limbic gyrus and consists of three segments: the head, body, and tail. The hippocampus is formed by two layers of gray matter in the coronal section: the cornu ammonis and dentate gyrus.

The posterior cerebral artery (PCA) mainly supplies arterial blood to the hippocampus. Three major arteries, including the anterior, middle, and posterior hippocampal arteries, arise from PCA. The hippocampal head receives arterial supply from the branches of the anterior choroidal artery (2).

This study will introduce and categorize various acute disorders that can involve the hippo-campus and explain the findings of MRI, especially with diffusion-weighted imaging (DWI). This study was approved by our Institutional Review Board, and the requirement for informed consent was waived (IRB No. EMC 2020-05-011-001).

INFECTION

HSV ENCEPHALITIS

Herpes simplex virus (HSV) encephalitis is the most common lethal sporadic encephalitis. Most patients are infected with HSV-1 (> 90%) (3). Patients often show nonspecific symptoms, such as fever, headache, nuchal rigidity, altered mental status, and focal or generalized seizures (4). The common sites of involvement are the anterior and medial temporal lobes, frontal lobe, and insula. Extratemporal involvement, including the limbic system, cingulate gyrus, and thalami, occurs in up to 55% of cases (Fig. 1). In the early stage, hyperintensity on DWI and hypointensity on apparent diffusion coefficient (ADC) maps can be observed due to cytotoxic cell swelling. Patchy areas of diffusion restriction indicating cytotoxic injury, particularly of the cortex and deep gray matter structures, are usually present and are a key feature of viral encephalitis. On T2-weighted and fluid-attenuated inversion recovery (FLAIR) images, hyperintensity caused by edema and inflammation can be observed within 48 hours (5). Leptomeningeal and underlying cortical contrast enhancement are commonly associated features (6). Recently, a real-time polymerase chain reaction for detecting HSV-DNA in the cerebrospinal fluid (CSF) has replaced routine brain biopsy for diagnosis.

JAPANESE ENCEPHALITIS

Japanese encephalitis, which is spread by mosquitoes, is one of the various viral encepha-

Fig. 1. A 62-year-old male diagnosed with herpes simplex virus encephalitis.
A. Diffusion-weighted image shows patchy hyperintense lesions in the right hippocampus (arrow) and right parahippocampal gyrus.
B. The fluid-attenuated inversion recovery image also shows patchy hyperintense lesions in the right hippocampus (arrow), right parahippocampal gyrus, frontal lobe, and temporal lobe.
**C.** The enhanced T1-weighted image shows prominent leptomeningeal enhancement along the pial surface of the right temporal cortical sulci (arrow).
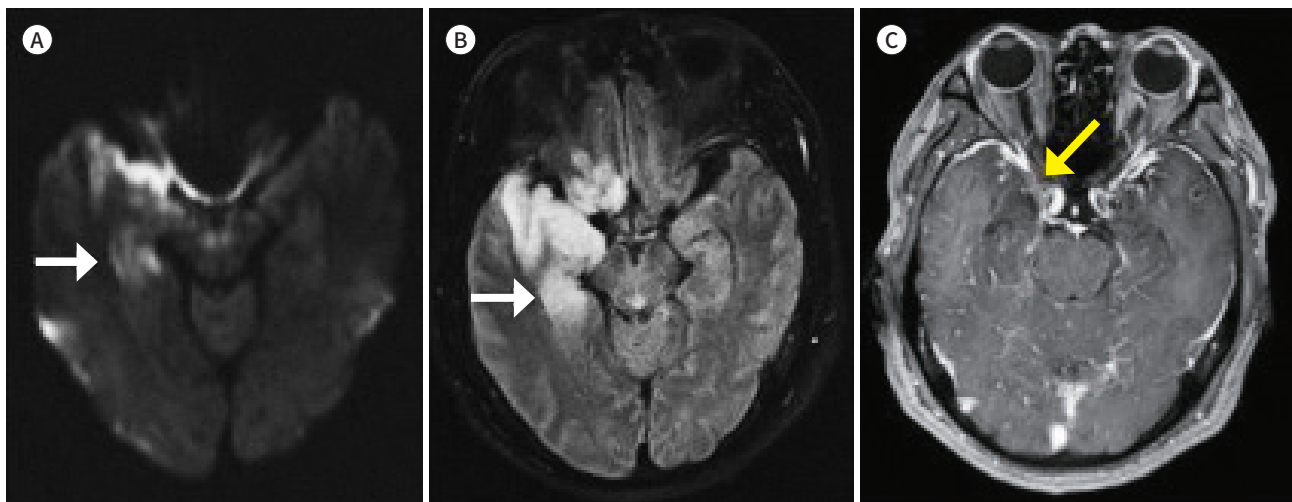
litides. It is especially prevalent in Korea, Japan, Southeast Asia and India. Patients may present with fever, headache, and focal neurological signs. The overall mortality rate is approximately 25% (7). On MRI, bilateral T2 thalamic hyperintensity is the most characteristic finding. Other areas of involvement include the pons, basal ganglia, hippocampus, and cerebral cortex (Fig. 2) (8). Findings on DWI are variable, and one report noted the value of this sequence in identifying abnormal lesions that were not evident on T2-weighted imaging, while other reports described some but not all lesions that showed evidence of restricted diffusion (9). Enhancement on contrast-enhanced imaging is uncommon, suggesting only a minor deficit in the blood-brain barrier. Japanese encephalitis is definitely confirmed by the antibody detection of immunoglobulin M (IgM) in serum and CSF.

Fig. 2. A 56-year-old male diagnosed with Japanese encephalitis.
A-D. Diffusion-weighted (A, B) and fluid-attenuated inversion recovery (C, D) images show patchy hyperintense lesions in both hippocampi (arrows) and thalami (arrowheads).
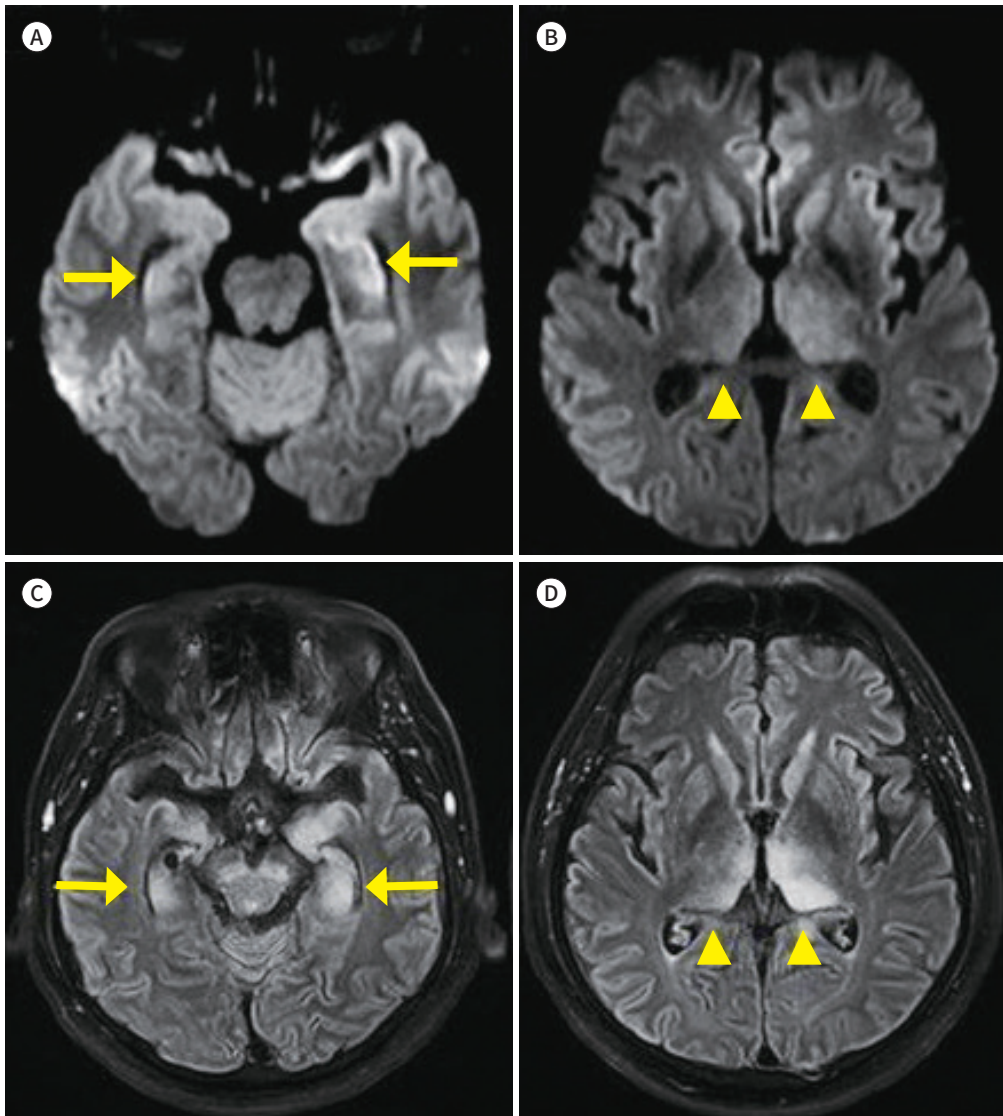
INFLAMMATION

AUTOIMMUNE ENCEPHALITIS

Autoimmune encephalitis is an antibody-mediated inflammatory disease and the most common inflammatory disease that affects the limbic system. Autoimmune encephalitis is subdivided into two groups based on the presence of antibodies associated with underlying tumors (10). Paraneoplastic autoimmune encephalitis is related to onconeuronal antibodies such as Yo, Hu, Ri and MA2 antibodies (11). However, non-paraneoplastic autoimmune encephalitis is related to voltage-gated, potassium channel (VGKC) antibodies, as well as antibodies directed against glutamic acid decarboxylase (GAD), N-methyl-D-aspartate (NMDA) receptor/channel or to unknown antigens (12). Typical clinical features of autoimmune encephalitis include behavioral and cognitive deterioration and epileptic seizures, among other clinical presentations (movement disorders, dysautonomia, etc.). In paraneoplastic and non-paraneoplastic autoimmune encephalitis, only a few case reports (11, 13) and a single case series (10) detected hippocampal hyperintensities on DWI, and it remained unclear whether these represented cytotoxic changes in water distribution or mainly a T2 shine-through effect. Moreover, swelling and hyperintensivity of uni- or bilateral temporomesial structures and limbic systems in FLAIR and T2-weighted sequences can be detected, and persist over months to years (Fig. 3). Diffuse or patchy contrast enhancement suggestive of inflammation is observed in a few patients while intense enhancing lesions are unlikely in autoimmune encephalitis (14). The frequent involvement of the basal ganglia helps distinguish it from HSV encephalitis (15).

METABOLIC

HYPOGLYCEMIC ENCEPHALOPATHY

Hypoglycemic encephalopathy is associated with severe hypoglycemia in patients with dia-

**Fig. 3.** A 46-year-old male diagnosed with autoimmune encephalitis.
A, B. Diffusion-weighted (A) and fluid-attenuated inversion recovery (B) images show a patchy hyperintense lesion in the left hippocampus (arrows).
C. Coronal enhanced T1-weighted image shows a patchy enhancing lesion in the left hippocampus (arrowhead).
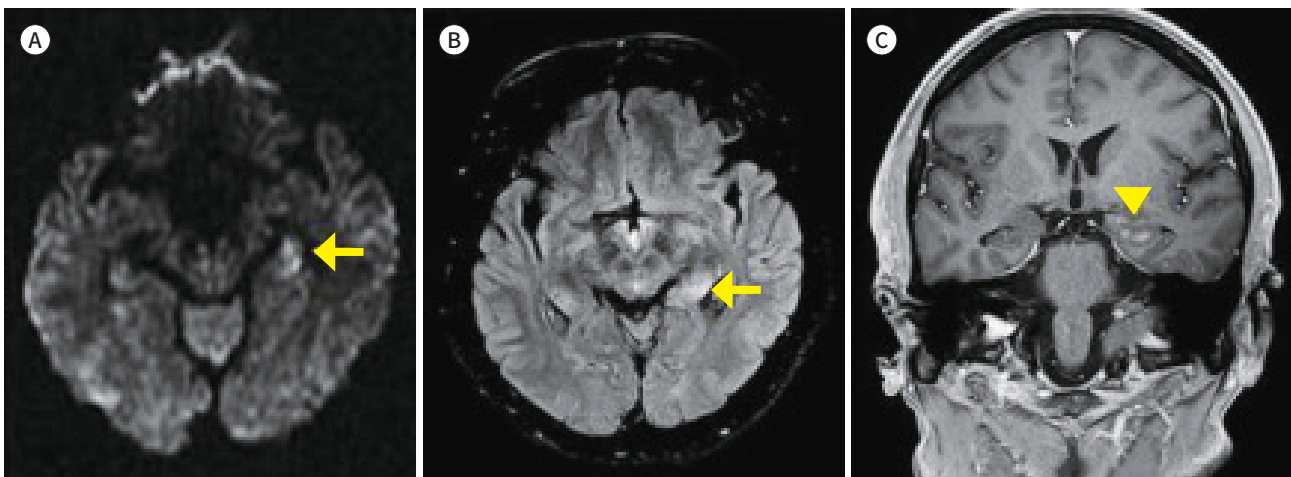
Fig. 4. A 66-year-old male diagnosed with hypoglycemic encephalopathy.
A-C. Diffusion-weighted images show patchy hyperintense lesions in both hippocampi (arrowheads), posterior limbs of the internal capsule (arrows), and high frontal white matter and cortex areas.
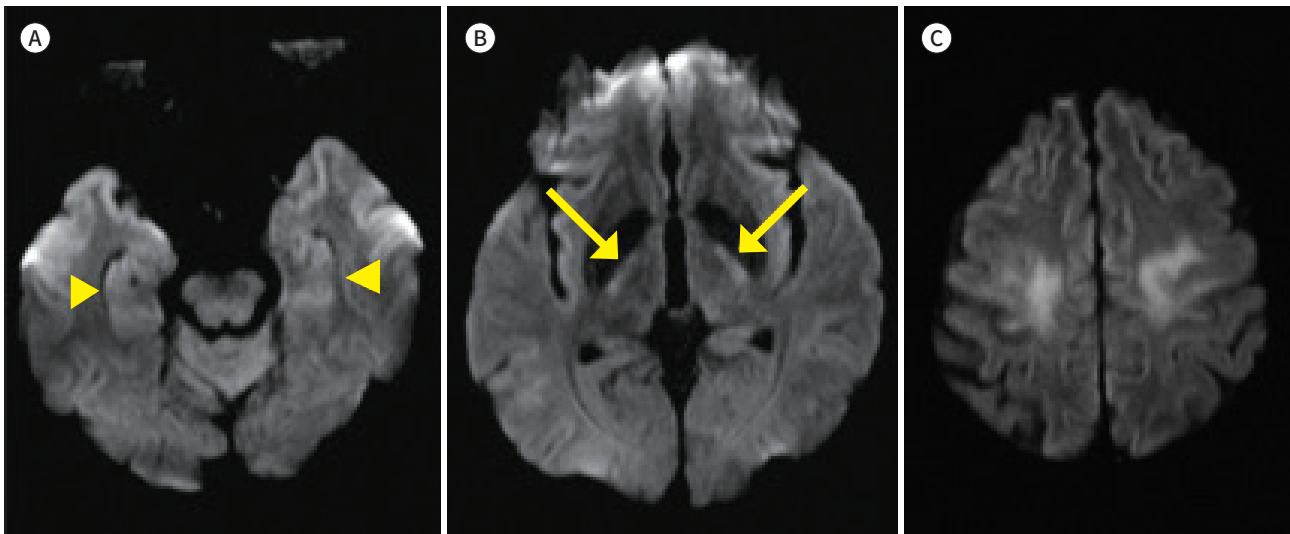
betes. The symptoms of hypoglycemia include unconsciousness, altered mentality, and seizures (16). Bilateral involvement of posterior limbs of the internal capsule, hippocampus, basal ganglia, and cerebral cortex is characteristic. Lesions can show hyperintensity on DWI and T2-weighted images (Fig. 4). The lesions may show reversible restricted diffusion. The pathomechanism of hypoglycemic encephalopathy still remains unclear. Some pathogenetic mechanisms for diffusion restriction in hypoglycemic encephalopathy that have been proposed include the following: 1) energy failure, 2) excitotoxic edema, and 3) asymmetric cerebral blood flow (17). Hemorrhages have not been reported in hypoglycemia, although contrast enhancement may occur (18). Fujioka et al. (19) reported that patients with basal ganglia involvement survived with persistent neurological impairment. Involvement of the basal ganglia indicates a poor prognosis.
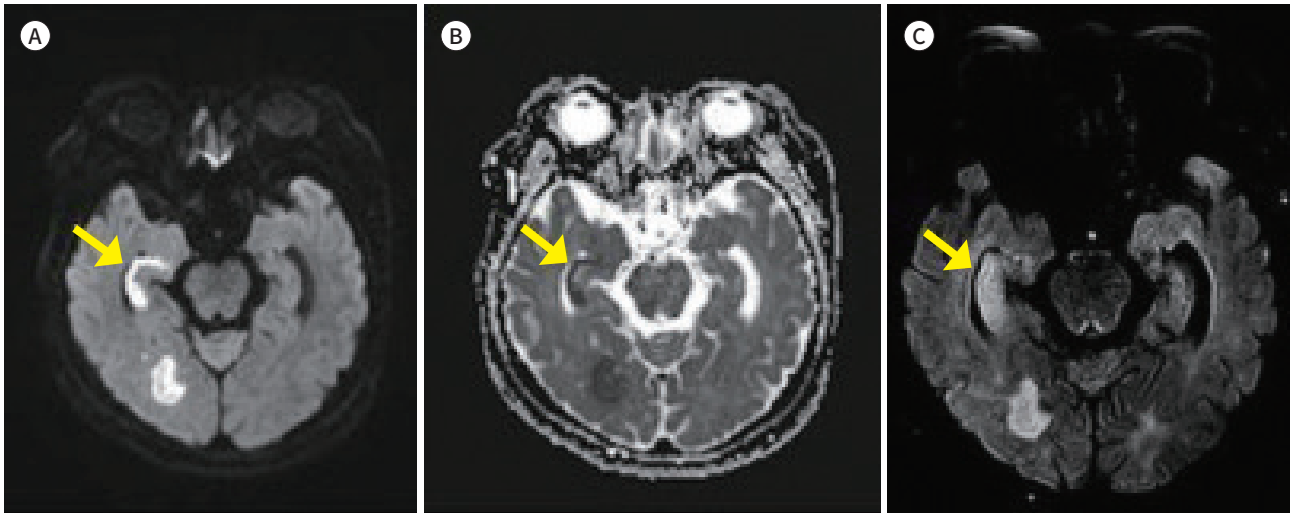
ISCHEMIC

ACUTE ISCHEMIC STROKE

PCA mainly supplies arterial blood to the hippocampus. Although PCA territory infarction is common, involvement of the hippocampus is relatively rare. Aphasia is common in hippocampal infarction as a typical sign of stroke with one-sided weakness. Moreover, the memory deficits may be detected by neurological examination (20). On DWI, hippocampal infarction is distinguished by four typical patterns: complete, dorsal, lateral hippocampus, and small circumscribed lesions in the lateral hippocampus (Fig. 5). It is associated with the vascular anatomy of the hippocampus (21). Acute ischemic stroke disrupts the cerebral energy metabolism, leading to failure of the Na^+/K^+ adenosine triphosphatase pump, loss of ionic gradients, and net translocation of water from the extracellular to the intracellular compartments. This is now the commonly accepted theory of diffusion restriction in acute stroke. According to the

Fig. 5. A 48-year-old female diagnosed with acute ischemic stroke.
A. The diffusion-weighted image shows patchy hyperintense lesions in the right hippocampus (arrow) and occipital area.
B. The apparent diffusion coefficient map shows patchy hypointense lesions in the right hippocampus (arrow) and occipital area.
C. The fluid-attenuated inversion recovery image shows patchy lesions with a slightly increased signal intensity in the right hippocampus (arrow) and occipital area.


literature (22), the signal intensity on FLAIR images varies after stroke. However, most of the literature indicates that in most patients with ischemic stroke, the findings on FLAIR images are positive 6–12 hours after the onset of symptoms. In ischemic stroke, enhancement may be arterial or parenchymal. Arterial enhancement usually occurs first and may be seen as early as 0–2 hours after onset of stroke. It fades about 1 week after stroke, around the time parenchymal enhancement begins. It is important key to note that isolated infarction of the hippocampus is rare. It may be helpful to distinguish it from other neurological disorders involving the hippocampus.

HYPOXIC ISCHEMIC INSULT

Hypoxic ischemic insult is the brain dysfunction resulting from insufficient oxygen or blood flow for a period of time. Cardiac arrest and cerebrovascular disease are common causes in adults. Generally, patients undergo acute events (i.e., drowning, asphyxia, cardiac arrest) and delayed resuscitation (23). Initially, hypoxic-ischemic insult affects gray matter structures, such as the thalami, basal ganglia, hippocampus, and cerebral cortex. Gray matter structures have high metabolic requirements for oxygen and glucose. Therefore, these structures are susceptible to hypoxic ischemic damage. DWI is a sensitive modality for detecting lesions in the acute period. In acute phase (< 24 hours), the lesions demonstrate widespread diffusion restriction in the thalami, basal ganglia, hippocampus, and cerebral cortex (Fig. 6). Ischemic brain damage results in restricted diffusion of free water, principally due to the failure of the energy-requiring active water transport mechanism (24). In early subacute phase (1 day to 2 weeks), the lesions show increased T2-signal intensity due to vasogenic edema (25). Contrast enhancement may be observed in the cortex, basal ganglia, and thalami, and it is due to blood–brain barrier breakdown. The contrast enhancement is usually ob-

Fig. 6. A 50-year-old male diagnosed with hypoxic ischemic insult.
A-D. Diffusion-weighted images (A, C) show multifocal patchy hyperintense lesions in both hippocampi (arrows), basal ganglia (arrowheads), and cerebral cortex areas. The apparent diffusion coefficient maps (B, D) show hypointense lesions in both hippocampi (arrows), basal ganglia (arrowheads), and cerebral cortex areas.
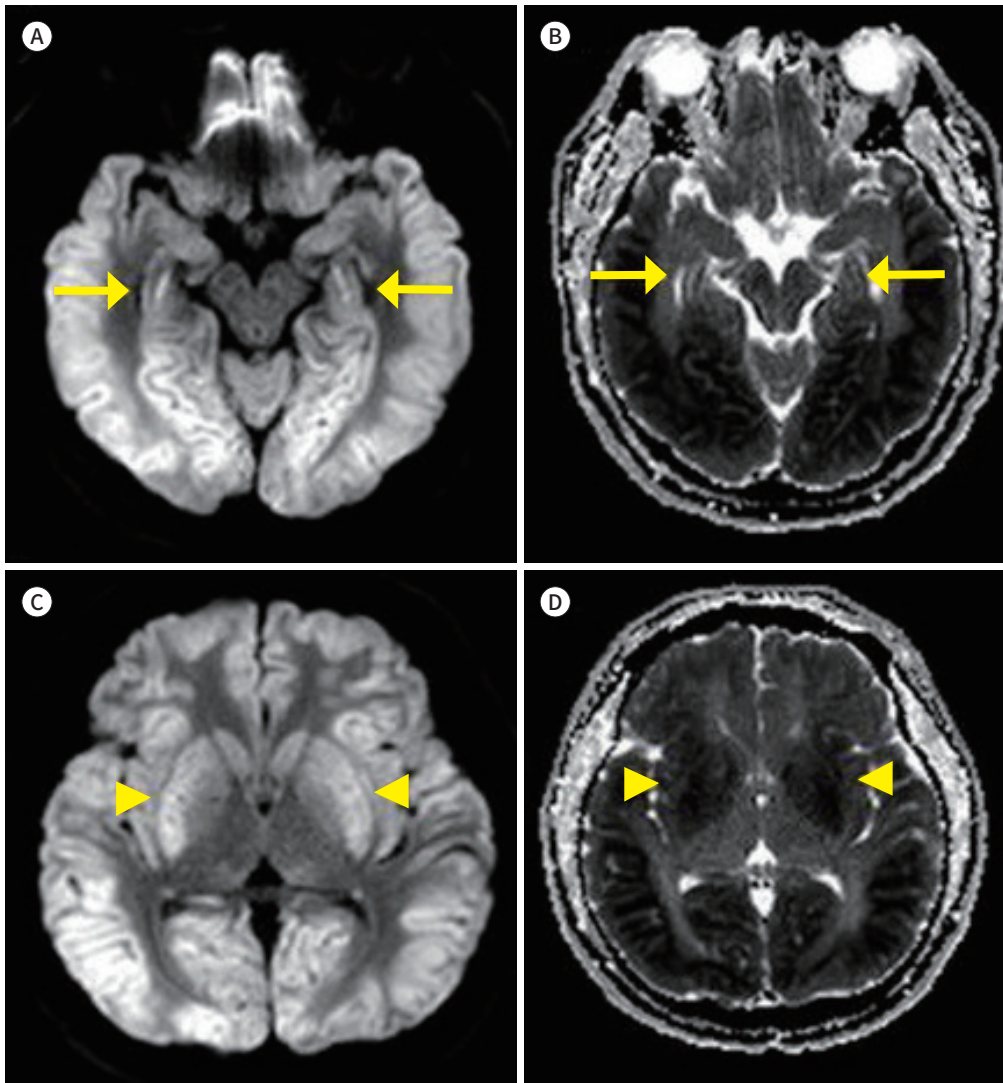
served after 1–2 weeks, peaks after 1–2 months, and commonly resolves after 6 months (26).
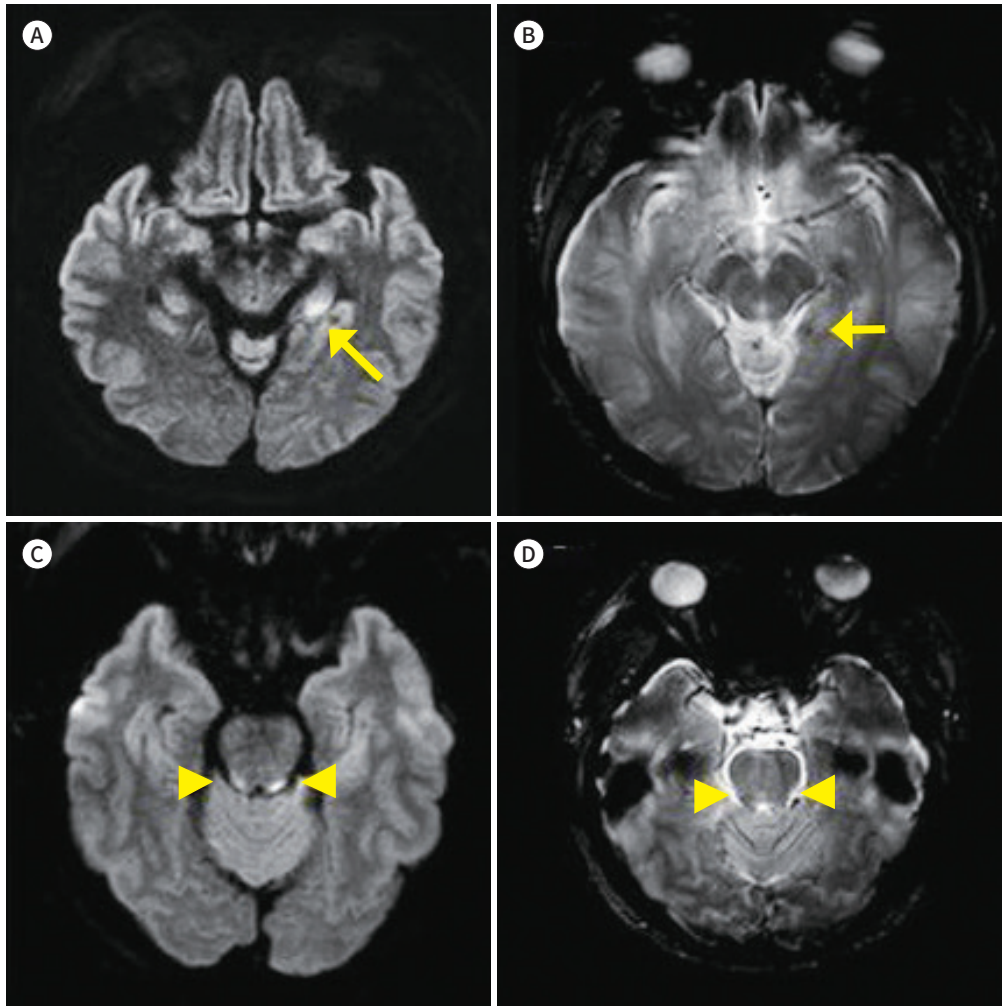
TRAUMATIC

DIFFUSE AXONAL INJURY

Diffuse axonal injury (DAI) is mainly caused by rotational acceleration or deceleration and subsequent brain injuries, such as axonal stretching, sequential separation, and disruption of nerve fibers (27). The most frequently affected anatomical sites are the corticomedullary junctions, located in the corpus callosum, upper brainstem and deep gray matter (28). DAI occurs mainly in the splenium of the corpus callosum, and may also occur in the mesial temporal lobe, including the hippocampus and parahippocampal gyrus (Fig. 7) (29). Restrict-

Fig. 7. A 57-year-old male diagnosed with diffuse axonal injury.
A-D. Diffusion-weighted images **(A, C)** show small patchy hyperintense lesions in the left hippocampus (arrow) and both posterolateral pons (arrowheads). Gradient-recalled echo images **(B, D)** show a few foci of dark signal intensity in the left hippocampus (arrow) and both posterolateral pons (arrowheads).

ed diffusion on DWI can be present in affected areas with low ADC values up to 18 days after the initial traumatic event. According to a previous study (30), this finding may represent a reflection of cellular swelling or cytotoxic edema. Small lesions with dark signal intensity can be detected on susceptibility-weighted imaging or gradient-recalled echo due to hemorrhage. Over the first few days, these lesions can show hyperintensity on FLAIR images. Contrast enhancement does not improve the conspicuity of acute traumatic brain injury; however, a few small studies have shown that posttraumatic contusions enhance in their subacute stages, similar to infarctions, most likely related to blood-brain barrier disruption and inflammation (31).

## MISCELLANEOUS

TRANSIENT GLOBAL AMNESIA

Transient global amnesia (TGA) is a clinical syndrome characterized by anterograde and

Fig. 8. A 58-year-old female diagnosed with transient global amnesia.
A. Diffusion-weighted images show several punctate lesions with high signal intensities in both hippocampi (yellow lines).
**B.** Apparent diffusion coefficient maps show a few foci of low signal intensity in both hippocampi (white lines).
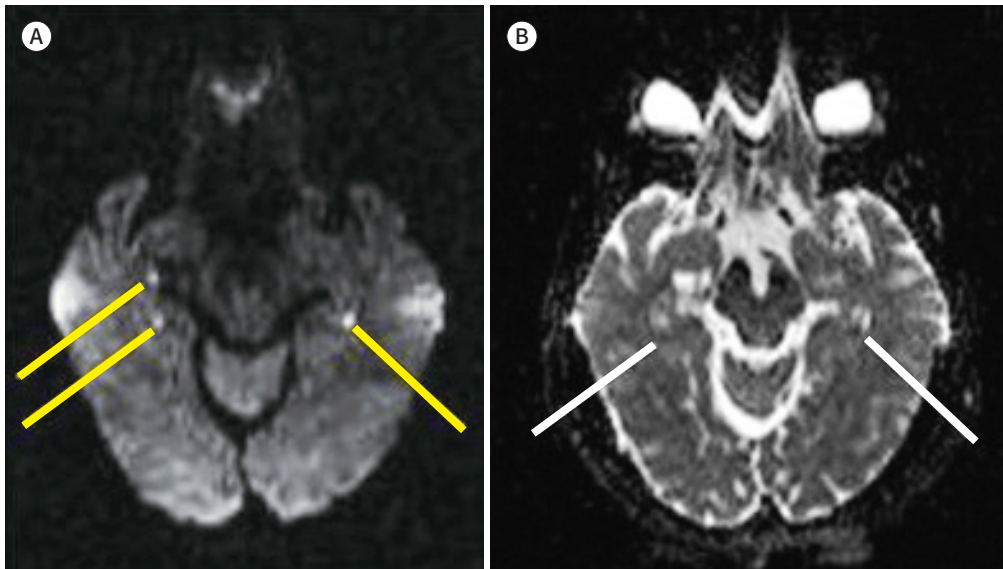
Fig. 9. A 54-year-old male diagnosed with seizure-related changes.
A. The diffusion-weighted image shows a patchy hyperintense lesion in the gray matter of the right hippocampus (arrow).
B. The fluid-attenuated inversion recovery image shows patchy lesions with slightly increased signal intensities in the gray matter of the right hippocampus (arrow).
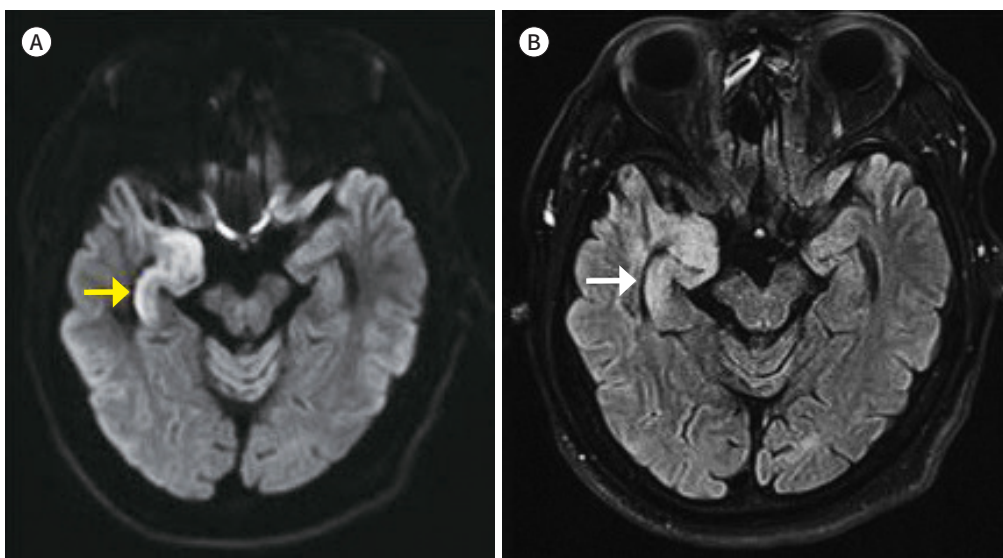
retrograde amnesia lasting less than 24 hours with complete resolution (32). The etiology of TGA remains unclear. However, several hypotheses associated with stroke, spreading depression and venous congestion have been suggested. No significant lesion can be detected on DWI and T2-weighted images in the acute phase, but serial DWI can show tiny, punctate DWI lesions in the lateral hippocampus after 48 hours (Fig. 8) (33). TGA lesion represents a tiny focus of diffusion restriction from ischemia with subsequent cytotoxic edema, rather than an

Table 1. Characteristic Findings of Acute Hippocampal Disorders for Differential Diagnosis

	Characteristic Finding	Etiology
DWI	Tiny, punctate lesions	Transient global amnesia
ADC map	Normal or high signal intensity	Autoimmune encephalitis
SWI/GRE	Small lesions with dark signal intensity	Diffuse axonal injury
Common sites of involvement	Anteromedial temporal and frontal lobes, and insula	HSV encephalitis
	Bilateral thalami	Japanese encephalitis
	Bilateral involvement of posterior limbs of internal capsule and cerebral cortex	Hypoglycemic encephalopathy
	PCA territory (occipital and posteromedial temporal lobes)	Acute ischemic stroke
	Widespread involvement of cerebral cortex, and basal ganglia	Hypoxic ischemic insult
	Corpus callosum and upper brainstem	Diffuse axonal injury
CE-T1WI	Diffuse or patchy parenchymal enhancement	Autoimmune encephalitis
	Arterial enhancement	Acute ischemic stroke
	Leptomeningeal enhancement	Seizure related change, HSV encephalitis

ADC = apparent diffusion coefficient, CE-T1WI = contrast-enhanced T1-weighted imaging, DWI = diffusion-weighted imaging, GRE = gradient-recalled echo, HSV = herpes simplex virus, PCA = posterior cerebral artery, SWI = susceptibility-weighted imaging

acute infarction, as suggested by previous studies that performed follow-up MRI of the brain (34). No abnormal contrast enhancement is usually observed (35).

SEIZURE-RELATED CHANGES

Seizure-related changes are widely variable and can be observed in the area of epileptic focus or at distant sites, reflecting the transmission of seizures along neuronal networks. The most commonly affected areas are the temporomesial structures. In particular, temporal lobe and hippocampus are the most common areas of abnormality in the literature (36). Diffusion-restricted lesions can be detected on DWI and ADC maps (Fig. 9). The pathophysiology of DWI abnormalities in seizures is believed to be that impaired energy metabolism caused by prolonged ictal activity is known to stimulate glucose utilization, which is not correlated with enhanced blood flow (37). Thus, cytotoxic and vasogenic edema may result from this imbalance in demand and supply (38). In contrast to stroke, signal changes on DWI and T2-weighted images may develop simultaneously in status epilepticus, which implies a different pathophysiology associated with energy metabolism. Seizure-related gadolinium enhancement has been described in several reports and case series. The most commonly described pattern is a discrete area of leptomeningeal enhancement overlying a focal area of the ictal cortex (36).

CONCLUSION

It is necessary to understand the anatomy and blood supply of the hippocampus. Many acute hippocampal disorders overlap with the clinical manifestations. In addition to clinical manifestations, other studies, including laboratory tests, electroencephalogram and MRI, especially DWI, can assess a definitive diagnosis and adequate treatment. Thus, it is important to categorize acute hippocampal disorders and we have to understand their specific imaging findings (Table 1) for differential diagnosis.

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급성기 해마 질환의 자기공명영상 소견: 임상화보

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해마는 뇌에서 가장 대사가 활발한 부위 중 하나이다. 그러므로 해마는 다양한 급성기 질환으로부터 영향을 받을 수 있다. 이 연구의 목적은 해마와 관련된 다양한 질환들을 소개 및 분류하며 특히 확산강조영상을 중심으로 자기공명영상 소견에 대해 설명하는 것이다. 급성기 해마 질환은 감염, 염증, 대사성, 허혈성, 외상성 그리고 기타 총 6가지로 분류하였다. 환자들은 임상적 소견 그리고 확산강조영상 중심의 자기공명영상을 토대로 후향적으로 검토되었다. 모든 질환들은 임상적 또는 병리학적으로 진단되었다. 급성기 해마 질환들은 임상 양상이 겹치는 경우가 많다. 그러므로 감별 진단을 위해서 급성기 해마 질환들을 분류하고 각각의 특징적인 영상 소견을 이해하는 것이 중요하다.

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