Magnetic resonance imaging of crossed cerebellar diaschisis and bright pulvinar in status epilepticus

Bandar N. Al-Jafen, *MD, SSC-Med (Neuro),* ***Mohammed H. Alanazy,*** *MBBS, MD,* ***James N. Scott,*** *MD, FRCPC,* ***Neelan Pillay,*** *MD, FRCPC.*

ABSTRACT

يعد استفراق عابر المخيخ والوسادة المشرقة علامات نادرة في المرضى الذين يعانون من حالة صرعية. نستعرض في هذا المقال حالة مريض يبلغ من العمر 53 عاماً، عُثر عليه في حالة اختلال وخلط وفقدان للتحكم بالبول، بالإضافة إلى عدم قدرته على التلفظ. وقد اتفقت نتائج تخطيط الدماغ مع الحالة الصرعية الغير تشنجية. وأظهرت صور الرنين المغناطيسي للدماغ نتائج تتفق مع استفراق عابر المخيخ والوسادة المشرقة. ولقد قمنا بمناقشة هذه الحالة لزيادة الوعي بهذه العلامات في التصوير بالرنين المغناطيسي في المرضى الذين يعانون من اختلال وخلط.

Crossed cerebellar diaschisis and bright pulvinar are rare in patients with status epilepticus. We present a case of a 53-year-old man who was found confused, incontinent, and nonverbal. The EEG findings were consistent with non-convulsive status epilepticus. The brain MR images showed findings consistent with crossed cerebellar diaschisis and bright pulvinar. We report and discuss this case to increase the awareness of these MRI signs in confused and obtunded patients.

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From the Division of Neurology, Department of Clinical Neurosciences (Al-Jafen, Alanazy, Pillay), and the Department of Radiology (Scott), University of Calgary, Calgary, Alberta, Canada.

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Address correspondence and reprint request to: Dr. Bandar N. Al-Jafen, Epilepsy and EEG Fellow, Department of Clinical Neurosciences, Calgary Comprehensive Epilepsy Program, University of Calgary, Foothills Medical Center, 1403-29 St N. W., Calgary, Alberta T2N 2T9, Canada. Tel. +1 (403) 7085563. Fax. +1 (403) 2107507. E-mail: bandaraljafen@gmail.com

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In confused patients, neuroimaging is helpful to diagnose acute neurological insults. Transient and permanent thalamic changes have been noted in convulsive and non-convulsive seizures.[1] Crossed cerebellar diaschisis (CCD) has been described initially in stroke patients.[2] There are rare reports of thalamic and CCD in non-convulsive seizure patients. Our objective in reporting this particular case is to highlight the importance of these MRI changes in a confused patient. It is crucial to exclude non-convulsive status epilepticus by correlation with EEG in an acutely obtunded patient with these MRI signs.

Case Report. A 53-year-old man was found confused, incontinent of urine, and nonverbal. There was no evidence of major trauma at the scene. A limited emergency room examination revealed equal reactive pupils with no gaze preference, global aphasia, diffuse rigidity, bilateral symmetric brisk deep tendon reflexes, and right extensor plantar response. Initial laboratory work revealed elevated liver enzymes and creatine kinase of 42,000 IU\L (66-400 IU\L). The CSF examination revealed a protein of 0.68, and 1.1 white blood cells with no xanthochromia. Glucose was normal, and gram stain was negative. The CT head, and CT angiography scans were performed in the emergency room and showed a previous craniotomy for a remote left subdural hematoma, but no acute pathology. Initially, he was treated empirically with acyclovir, vancomycin, and ceftriaxone in addition to intravenous fluid hydration. On the second day of admission, he continued to be confused and showed twitching of his right leg. The EEG demonstrated evidence of left periodic lateralized epileptiform discharges consistent with non-convulsive status epilepticus (**Figure 1**). He was treated with a load of intravenous phenytoin and levetiracetam. Failure to control seizures and persistent altered level of consciousness prompted admission to the intensive care unit for intubation and propofol infusion under continuous EEG monitoring. An MRI of the brain

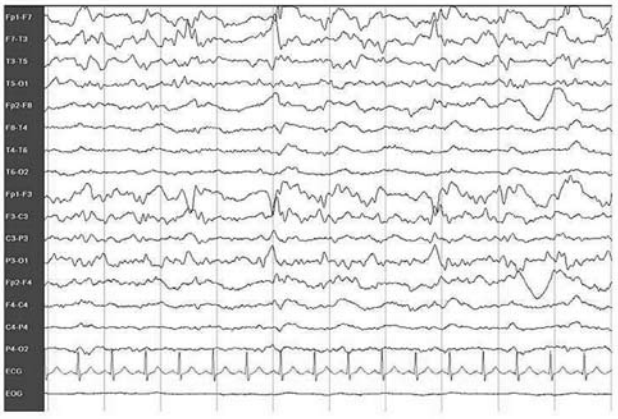

Figure 1 - Patient EEG demonstrated left-sided periodic lateralized epileptiform discharges indicating ictal activity.

revealed abnormal signal involving the left frontal and peri-insular cortex, left pulvinar, and the right cerebellar hemisphere consistent with crossed cerebellar diaschisis (**Figure 2**). Two days later, he showed marked clinical improvement and seizures were controlled on levetiracetam and topiramate. Follow-up MR imaging at one week showed the same findings as the first one. Since there was no clinical or laboratory evidence of acute viral encephalitis, the etiology of status epilepticus was probably due to the old left subdural hematoma.

Discussion. We report this case with these brain MRI findings (crossed cerebellar diaschisis and bright pulvinar) in a patient with non-convulsive status epilepticus. Diaschisis is defined as a reduction in

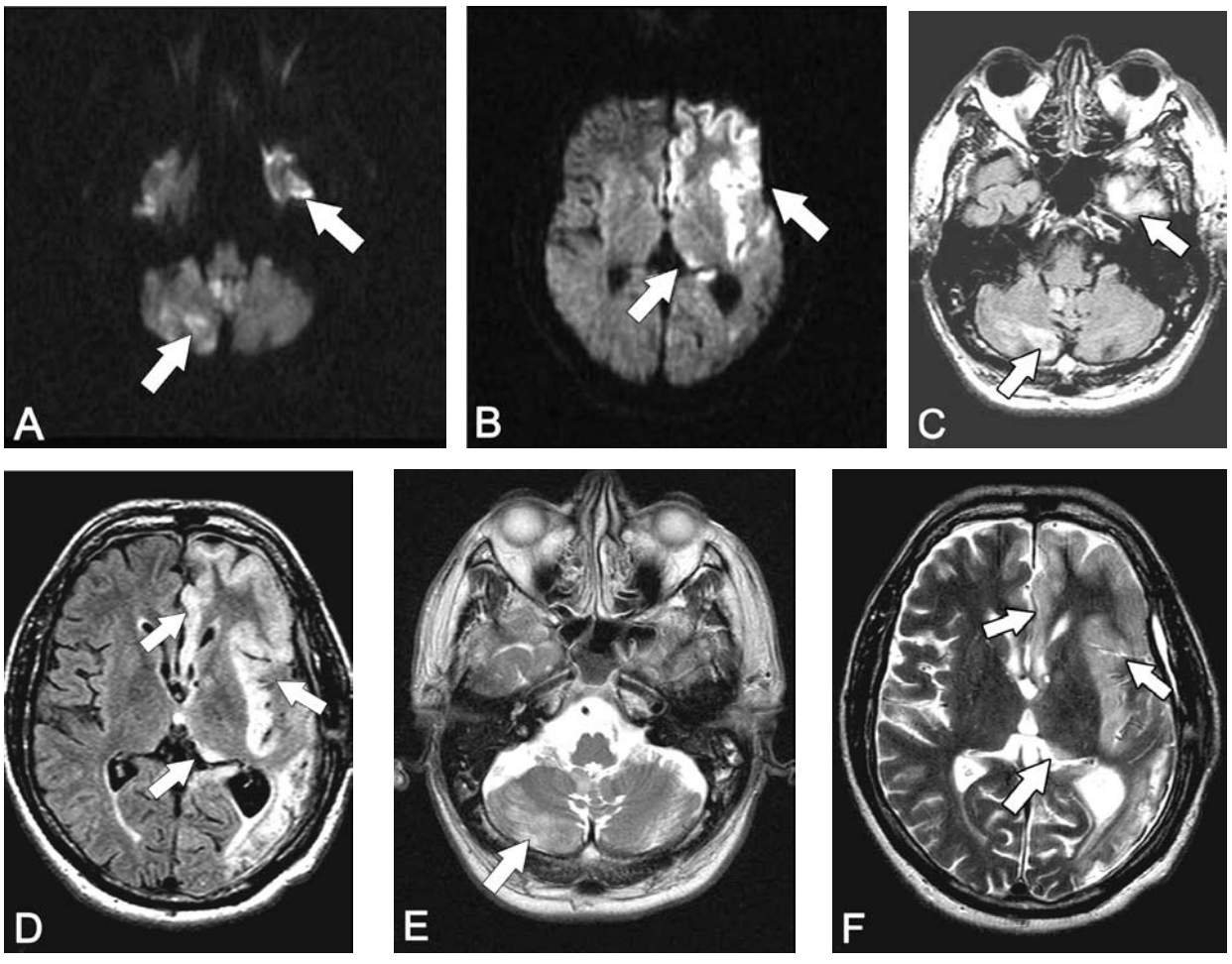

Figure 2 - The MR imaging shows restricted diffusion on axial diffusion-weighted images **(A, B)**, and increased signal on axial FLAIR (**C, D**), and T2 weighted images (**E, F**) in the left frontal, peri-insular, and temporal regions, left medial pulvinar nucleus, as well as involvement of the right cerebellar hemisphere indicating crossed cerebellar diaschisis.

blood flow and metabolism remote from a diseased cortical area.[2] The most likely mechanism of CCD is the interruption of the afferent corticopontocerebellar pathways, resulting in reduction in blood flow and oxygen uptake in the cerebellar hemisphere contralateral to a supratentorial lesion.[2] The CCD has been initially identified on positron emission tomography scans in stroke patients.[2,3] It has also been reported in brain tumors,[4] Rasmussen's encephalitis,[4] herpes simplex encephalitis,[5] post-intracarotid amytal injections,[6] and ictal single-photon emission computed tomography.[7] Partial status epilepticus can cause a restricted diffusion in diffusion-weighted images,[8] which could be related to a mismatch between metabolism and cerebral blood flow resulting in tissue hypoxia, and subsequently cytotoxic injury and edema.[9] The MRI changes in CCD are generally reversible, however frequent and prolonged seizures may push the process beyond reversibility and cause excitotoxic cell damage leading to cerebellar atrophy.[9]

Involvement of the pulvinar is postulated to be due to the propagation of temporal lobe seizures, and the finding of MR signal abnormality within it in patients with non-convulsive partial status epilepticus has only recently been recognized.[10,11] These MRI changes may be reversible, but some may show permanent atrophy.[1] Pulvinar and involved cortical area are presumably normal tissues, which are subsequently affected by seizure spread during frequent and prolonged seizure.[10,11]

In conclusion, CCD and involvement of the pulvinar have been rarely reported in patients with prolonged and uncontrolled seizures.[1,5] For patients in prolonged confusional states, such signal changes are important diagnostic clues that seizure may be occurring. However, clinicians should not rely on diagnostic imaging. The EEG is required in all patients with decreased level of consciousness in the absence of a clear diagnosis.

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