Case Report

Journal of Emergency Medicine Case Reports

Diffuse Axonal Damage and Status Epilepticus

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Abstract

Diffuse axonal injury (DAI), microscopic damage to axons in the brain neuralpath ways, corpus callosum, and brain stem, is associated with significant mortality and morbidity. Thetreatment of patients with DAI is geared to ward spreventing secondary injuries and facilitating rehabilitation. A 57-year-old male patient was brought to the emergency room by the EMS team with the complaint of seizures in the form of an incision on the scalp, change in consciousness, confusion and convulsions after he lost his head to a cutting tool at work. In his neurological examination, it was found that the pupils were isochoric, the patient had seizures repetitively every two minutes, his consciousness was confused, and he could not obey orders. The patient was intubated with the diagnosis of status epilepticus due to diffuse axonal damage after a sharp object injury and was followed up in the intensive care unit. The patient improved clinically after the intensive care unit and was discharged with a follow-up recommendation. The possibility of diffuse axonal damage due to the mechanism of the trauma should be considered in cases that are not of high severity and no etiology has been detected and presenting with post-traumati cunconsciousness.

Keywords: Diffuse axonal injury, trauma, seizure

Introduction

Epilepsy is one the diseases that frequently leads to disability and can affect individuals of all ages, races, social classes, and geographical regions (1). Diffuse axonal injury (DAI), microscopic damage to axons in the brain neuralpath ways, corpus callosum, and brain stem, is associated with significant mortality and morbidity. The occurrence of DAI depends on the mechanism of injury; it is more common in high-energy traumas, especially in traffic accidents (2-4). Diffuse axonal injury is defined clinically by comalasting 6 hours or longer after traumatic brain injury (TBI), excluding swelling or ischemic brain lesions (2). DAI is considered the most important factor in determining morbidity and mortality in TBI survivors and is the most common cause of post-traumatic coma, disability, and persistent near-vegetative state (2,3).

Outcome of patients after DAI correlated with the number of lesion sidentified by imaging. A longitudinal study analyzing the evolution of traumatic axonal injuryusing magnetic resonance imaging (MRI) in 58 patients with moderateor severe TBI showed that as the number of lesions observed early after trauma increases, so does impairment in functioning after 12 months (5). A study of 26 DAI patients showed that the volume and number of MRI-identified lesions performed within 48 hours of hospitalization were strongly associated with the level of disability observed at hospital discharge (6). Treatment of patients with DAI is geared towards preventing secondary injuries and facilitating rehabilitation. Secondary injuries and hypotension, edema, intracranial hypertension, and hypoxia are the leading causes of increased mortality. Therefore, emergency care is recommended to prevent hypotension, hypoxia, cerebral edema, and high intracranial pressure (ICP) (7).

Case

A 57-year-old male patient was brought to the emergency room by the EMS team with the complaint of seizures in the form of an incision on the scalp, change in consciousness, confusion and convulsions after he lost his headto a cuttingtool at work. The vital signs of the patient at the time of admission were fever 36.4 °C, pulse 110/min, respiratory rate 18/min, systolic blood pressure 180 mmHg, diastolic blood pressure 126 mmHg, blood sugar 136 mg/dL. The Glasgow Coma Score (GCS) of the patient at the time of admission was 12 (M:4 M:4 V:4). In the anamnesis, it was learned that the EMS team was informed about the patient,

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Received: 10.06.2024 • **Revision:** 02.07.2024 • **Accepted:** 16.07.2024
DOI: 10.33706/jemcr.1498661

Cite this article as: Ugur Y, Ozcan AB. Diffuse Axonal Damage and Status Epilepticus. Journal of Emergency Medicine Case Reports. 2024;15(3): 63-65

DOI: 10.33706/jemcr.1498661
Journal of emergency medicine case reports. 2024;15(3): 63-65

who caught his hair in a cuttingtool at work and sat on the ground after an incision on the scalp, and that he was conscious and normal at that time. The ambulance team reported that diazepam was administered to the patient, who started to have altered consciousness and seizures in the ambulance during his transfer.

In the physical examination of the patient, incisions containing the skin and subcutaneous tissue of medium depth, approximately 5 cm in length, with irregular paralel edges, were observed in the scalp area of the left parietal bone close to the occipital bone. No acute traumatic lesion was detected in any other part of the body in his external examination. In his neurological examination, it was found that the light reflex was positive bilaterally, the pupils were isochoric, the patient had convulsive seizures recurring every two minutes, his consciousness was confused, and he could not obey orders. When the GCS was re-evaluated, this time was calculated as 10 (E:4 M:4 V:2). No pathological findings were found in other system examinations (respiratory, cardiac, abdominal, extremity).

The incisions of the patient were closed with stapler and dressing was applied. Tetanus and antibiotic prophylaxis were performed. Brain computed tomography (CT) imaging was performed in the patient who was re-treated with diazepam and given a loading dose of levatiracetam despite his recurrent seizures. The patient, who seseizures continued after imaging, was intubated on a planned basis due to the risk of status epilepticus and aspiration. Diffusion magnetic resonance (MR) imaging and SWI imaging were applied to the patient who was followed up under phenytoin and midazolam infusion after intubation. No acute traumatic pathology was observed in the brain CT examination, except subcutaneous hematoma in the area of the incision (Figure-1). On the other hand, in diffusion MR imaging, an increase in intensity thickness compatible with posttraumatic hematoma in the subcutaneouss of planes in the left parietal and millimetric-sized no specific signal increases in both frontal subcortical white matter were detected (Figure-1). In the cranial SWI MR examination, linear hemorrhagic signal losses in the right frontobasal white matter were evaluated in favor of grade 1 diffuse axonal injury (Figure -2).

The patient was intubated and followed up in the intensive care unit with the diagnosis of status epilepticus due to diffuse axonal damage after a sharp object injury. The patient improved clinically after four days in the intensive care unit, and was discharged with a follow-up recommendation after being followed up in the service for four days.

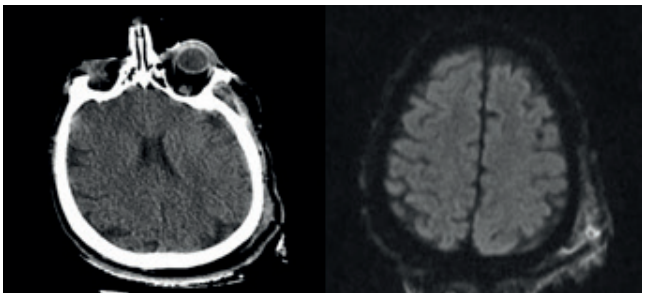

Figure 1. Subcutaneous hematoma in left parietooccipital on brain CT image, intensity thickness increase consistent with post-traumatic hematoma in the subcutaneouss oftplanes in the left parietal in diffusion MR examination

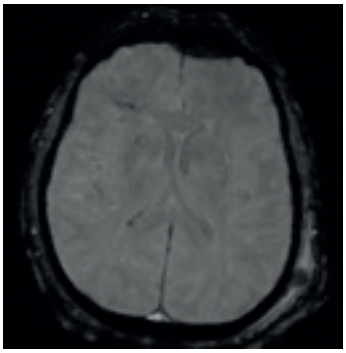

Figure 2. Cranial SWI MRI inthe right frontobasal linear hemorrhagic in white matter signal losses grade 1 diffuse compatible with axonal injury hypointense area

Discussion

Diffuse axonal injury (DAI) is a “hidden” pathology of traumatic brain injury (TBI). Although found throughout the white matter, it mainly contains microscopic damage, making it nearly invisible to current imaging techniques. As a classification, two general categories of brain trauma have emerged, defined as “focal” and “diffuse” brain injury (3,8,9). In our case, according to this classification, a focal brain injury can be mentioned when the results in the imaging findings and theclinic are examined.

In approximately 50% of cases, DAI occurs as a result of high-speed vehicle crashes, falls, and assaults (10,11,12). According to Moe et al, the same mechanisms of injury are observed in low-speed traumatic accidents such as sports injuries, falling from stairs or standing up, which can also lead to DAI (13). In our case, the etiology developed due to a sharp instrument injury, apart from other common causes. This showed that we should consider diffuse axonal damage in unexpected clinical situations.

According to the histopathological findings, Adams et al. classified DAI into three grades: Grade I–DAI with axonal lesions in the cerebral hemispheres; Grade II–DAI with focal axonal lesions in the corpus callosum; Grade III–DAI with focalor multiple axonal lesions in the brain stem (14). In the MRI examination of our case, it was evaluated as Grade I because axonal injury was detected in the right frontobasal white matter.

CT examination is stil the gold Standard for imaging DAI from an emergency stand point. CT scan may be negative or show typical DAI findings including multiple hemorrhagic lesions 5 to 15 mm in diameter at the gray-white matter

interface (15). MRI is the recommended tool for imaging DAI, but its usability is limited comparedto CT, especially in emergencies. According to Gentry et al, MRI may show diffuse, small, focal abnormalities confined to the white matter tracts. They tend to be multiple and non-hemorrhagic when present (16). In our case, there was no DAI finding on CT imaging, but axonal damage was detected on MRI imaging.

Conclusion

High-speed motor vehicle accidents are often involved in the etiology of diffuse axonal injury. The most common mechanism involves an accelerating and decelerating movement in the white matter path ways of the brain. It was note worthy that diffuse axonal damage, which we usually follow after or in association with hemorrhagic or ischemic cerebrovascular accident, did not accompany these diagnoses in our case. It comestomind that the patient in our case caused diffuse axonal damage during his rescue effort with a throwing motion after his hair was caught in the cuttingtool. As a result, the possibility of diffuse axonal damage due to the mechanism of the trauma should be considered in cases that are not of high severity and no etiology has been detected and presenting with post-traumatic unconsciousness.

Ethics

The case report has written in an anonymous characteristic, thus secretand detailed data about the patient has removed. Editor and reviewers can know and see these detailed data. These data are backed up by editor and by reviewers.

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