ORIGINAL RESEARCH PAPER
Volume - 9 | Issue - 9 | September - 2020 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

ROLE OF MAGNETIC RESONANCE IMAGING IN DIAGNOSIS AND GRADING OF DIFFUSE AXONAL INJURY – A CASE SERIES

Radiology

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ABSTRACT

Traumatic brain injury is a major cause of death and disability worldwide. The most common cause is motor vehicle accidents, especially when the acceleration-deceleration force on the brain is massive. Traumatic brain injury can be divided into focal and diffuse injuries (diffuse axonal injury (DAI) and diffuse brain edema). Glasgow Coma Scale which measures level of consciousness at the emergency department has been the primary clinical variable to grade initial brain injury severity. The sites of predilection include the gray-white matter interfaces at the subcortical region, corpus callosum, brainstem and cerebellum. Computed tomography (CT) is the initial investigation of choice in unstable patients, but it underestimates the extent of parenchymal injury. Magnetic resonance imaging (MRI) is a more sensitive method to detect DAI.

We report four cases of diffuse axonal injury in patients who presented to the emergency department with traumatic head injury. All of these patients had low Glasgow coma scale score on admission and neurological deterioration of these patients led to evaluation with MRI.

KEYWORDS

Diffuse axonal injury, Glasgow Coma Scale, Brain injuries, Magnetic resonance imaging

CASE REPORTS

Case Report 1:

A 28 year old male was brought to the emergency room, with weakness of the left upper and lower limbs. The patient had history of trauma one month back. On examination, the Glasgow coma scale (GCS) was found to be 10. On examination power of the left upper limb and lower limb was found to be 2/5.

Magnetic resonance imaging (MRI) brain was done which showed multiple T2* weighted gradient echo (T2*GRE) blooming foci (Figure 1 A, B) and hypointense foci on susceptibility weighted imaging (SWI) (Figure 1 C, D) in grey white matter junctions of bilateral frontal and temporal lobes, right thalamus, midbrain, left hemi-pons, cerebellar vermis and left cerebellar hemisphere. Above findings represent diffuse axonal injury (DAI) grade III.

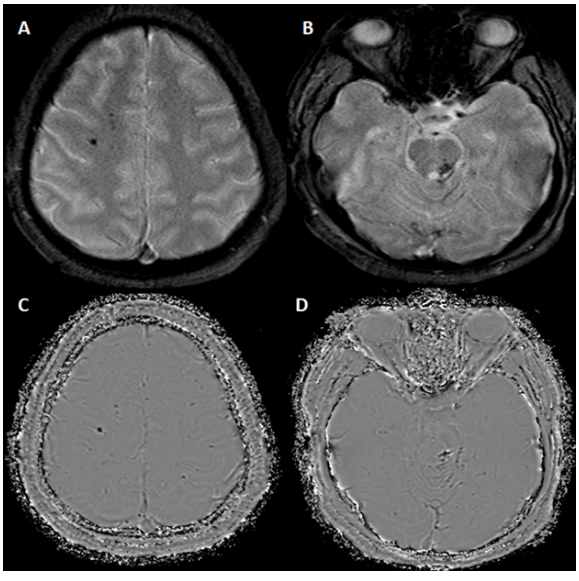

Figure 1: A 28 year old male with weakness of left upper and lower limbs and history of road traffic accident one month back. A, B) MRI Brain T2*GRE sequence axial sections show GRE blooming foci in subcortical grey white matter junction of right frontal lobe and left hemi-pons. C, D) MRI Brain SWI sequence axial sections show hypointense foci in subcortical grey white matter junction of right frontal lobe and left hemi-pons.

Case Report 2:

A 55 year old male was brought to the Emergency room after a road traffic accident. He had loss of consciousness with head injury. GCS was 7 on admission. CT Brain showed a small peri-ventricular hemorrhage of size 5 mm in the body of right caudate nucleus. Few petechial hemorrhages were seen in right frontal lobe grey white matter junction. Intra-ventricular hemorrhage in occipital horns of bilateral lateral ventricles was seen (Figure 2).

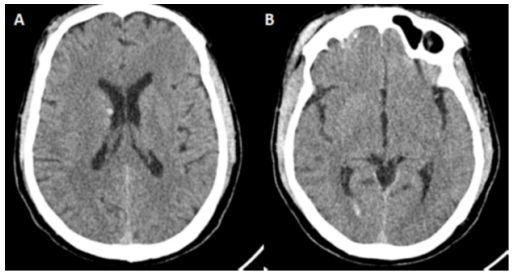

Figure 2: A 55 year old male with loss of consciousness after a head injury due to road traffic accident. A) CT Brain axial section shows a petechial hemorrhage in body of right caudate nucleus. B) CT Brain axial section shows intra-ventricular hemorrhage in the

MRI brain was advised as there was no significant improvement in the patient's condition and low GCS. MRI Brain showed hypointense foci on SWI and T2*GRE blooming foci suggestive of petechial hemorrhages in right frontal white matter, right centrum semiovale, right corona radiata, body of right caudate nucleus, genu, body and splenium of corpus callosum (Figure 3). Above features are suggestive of DAI grade II.

CASE REPORT 3:

A 20 year old male was brought to the Emergency room after a road traffic accident with head injury. GCS was 7 on admission. CT Brain showed contusional hemorrhages in right temporal lobe (Figure 4 A). MRI Brain was done 4 days after the initial injury, as the GCS of the patient did not improve. MRI Brain showed bilateral diffuse cerebral edema. T2*GRE blooming focus of size 2 mm in the right temporal lobe periventricular region (temporal horn) was seen (Figure 4 B).

Above findings likely represent diffuse axonal injury grade I.

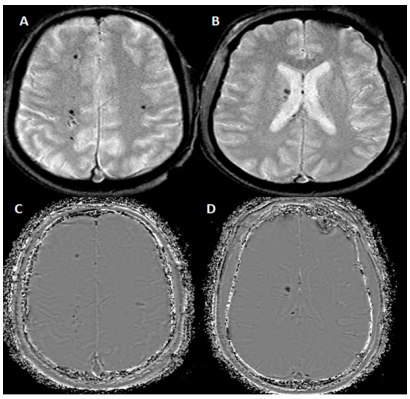

Figure 3: A, B) MRI Brain T2*GRE sequence axial sections show subcortical grey white matter junction of bilateral frontal lobes and body of right caudate nucleus. C, D) MRI Brain SWI sequence axial sections show hypointense foci in subcortical grey white matter junction of right frontal lobe and body of right caudate nucleus.

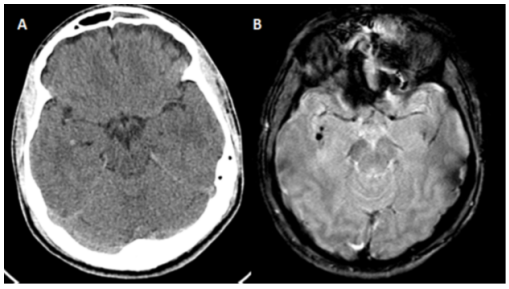

Figure 4: A) CT Brain axial section shows a tiny petechial hemorrhage in peri-ventricular region of right temporal lobe. B) MRI Brain T2*GRE sequence axial section shows a GRE blooming focus in peri-ventricular region of right temporal lobe.

DISCUSSION:

INTRODUCTION:

Traumatic brain injury (TBI) is a major cause of death and disability worldwide [2]. TBI occur most often due to motor vehicle accidents, especially when the acceleration-deceleration force on the brain is massive [5]. TBI can be divided into focal (fracture, contusion, subdural or epidural hematoma) and diffuse injuries (DAI and diffuse brain edema) [2]. DAI is now widely accepted as one of the most common type of injury in patients with severe head trauma [4].

ETIO-PATHOGENESIS:

DAI is a type of brain damage which occur secondary to rotational acceleration / deceleration. Centripetal forces generated by high-speed impact, lead to cortico-subcortical shearing. The damage is axonal resulting in tearing/stretching of axons causing impairment of axoplasmic transport and electrical network [1, 11].

There is subsequent swelling of the axons, resulting in discrete bulb formations or elongated varicosities that accumulate organelles. Calcium entry into damaged axons occurs, which initiates further damage by activation of proteases and induction of mitochondrial swelling and dysfunction. Ultimately, swollen axons become disconnected and contribute to neuropathologic changes in brain tissue [9]. Grossly, lesions range in size from 1 mm to 15 mm. They are ovoid in shape, with their long axis parallel to the direction of the involved axonal tracts [4].

The histopathological grading of DAI, proposed by Adams and associates, into grades 1 - 3 is based on the presence of axonal injury in the cerebral hemispheres with a predilection for the grey-white matter interface (Grade 1), the corpus callosum (Grade 2) and the dorso-lateral, rostral brainstem (Grade 3) [2].

CLINICAL FEATURES:

DAI is difficult to diagnose in acute phase. Clinical signs and imaging may suggest the diagnosis of DAI, but confirmation is only possible postmortem [5].

GCS, which measures level of consciousness at the trauma scene or at emergency department, has been the primary clinical variable to grade initial brain injury severity into mild (GCS 13–15), moderate (GCS 9–12) or severe (GCS ≤ 8) [7].

Depending on the severity and extent of injury, patients can present acutely with immediate loss of consciousness or confusion and persist as coma and cognitive dysfunction. The diagnosis is often only suspected when the patients do not make a neurological recovery [6]. Axonal injury in the brainstem appears to be a primary factor in the generation of coma in DAI patients [8].

IMAGING:

CT is the initial investigation of choice for the evaluation of unstable patients [4]. CT findings in DAI are typically limited to petechial hemorrhages in the white matter and traumatic edema of the brain [2]. However, CT underestimates the extent of parenchymal injury as less than 20% of DAI are macroscopically hemorrhagic [4].

MRI is a more sensitive method to detect DAI and is capable of visualizing even microscopic amounts of blood products. MRI sequences sensitive for hemorrhage are T2*GRE and SWI [2].

Lobar White Matter Shearing Lesions:

DAI lesions most commonly occur at the subcortical grey-white mater junction in the cerebral hemispheres, which constitute 67% of DAI lesions. This anatomical region is more vulnerable because of its peripheral location and because of the abrupt transition in tissue density between gray and white matter [4].

Corpus Callosum Shearing Lesions:

When portions of the accelerating and rotating brain, lag behind the adjacent faster moving areas, shear stress develops between tissues. This happens mainly in the callosal region because the independently mobile cerebral hemispheres are connected by the less mobile corpus callosum. The corpus callosum is the second most commonly involved area in DAI, constituting 21% - 47% of DAI lesions. Splenium is more frequently affected. Sometimes intra-ventricular hemorrhage can be seen, which is caused due to shearing of sub-ependymal veins [4].

Brainstem Shearing Lesions:

Midbrain and upper brainstem is the third common location for shearing injuries. The postero-lateral quadrant of the mesencephalon, adjacent to the superior cerebellar peduncle, is the most frequently affected. Brainstem injuries are only seen in more severely injured patients [4].

TREATMENT:

Treatment of patients with DAI is directed towards the prevention of secondary injuries and facilitating rehabilitation. It is the secondary injuries that lead to increased mortality. These include hypoxia with coexistent hypotension, edema and intracranial hypertension. Therefore, prompt care should be taken to avoid hypotension, hypoxia, cerebral edema and elevated intracranial pressure. Initial treatment priority in traumatic brain injury patients is focused on resuscitation [10]. Park et al. demonstrated a positive correlation between MRI grade and duration of unconsciousness. They found that grade I patients had a 1 week recovery time, whereas the recovery of grade II and grade III patients was 2 weeks to 2 months [3].

DIFFERENTIAL DIAGNOSIS:

The main differentials of diffuse axonal injury include cortical contusions, diffuse vascular injury, amyloid angiopathy and chronic hypertensive encephalopathy [10]. The differential diagnosis of brainstem DAI lesions includes duret hemorrhage in trans-tentorial herniation, peri-mesencephalic sub-arachnoid hemorrhage and vascular malformations [4].

CONCLUSION:

Even with closed head injury, the brain can suffer severe damage from diffuse axonal injury due to shearing forces. The sites of predilection include the gray-white matter interfaces at the subcortical region, corpus callosum, basal ganglia, brainstem and cerebellum. Conventional neuro-imaging studies underestimate the amount of damage because non-hemorrhagic lesions are difficult to identify in CT. MRI is more sensitive in detecting DAI [4].

ABBREVIATIONS:

CT – Computed Tomography, DAI – Diffuse Axonal Injury, GCS –

Glasgow Coma Scale, GRE – T2* weighted Gradient Echo, MRI – Magnetic Resonance Imaging, SWI – Susceptibility Weighted Imaging

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