INTRODUCTION
Diffuse axonal injury is defined as widespread damage to the brain caused by trauma and not presenting as localized injuries or as a consequence of herniation and perfusion failure. In diffuse axonal injury (DAI), the brain shows minimal gross alterations. However, classic microscopic findings include reactive axonal swellings secondary to tearing of nerve fibers, scattered microglial accumulations and debris-laden macrophages. In addition, many patients with DAI have hemorrhagic or necrotic lesions in the corpus callosum and the dorsolateral quadrant of the rostral pons.

There are significant differences in the pathophysiology and mechanics of damage between DAI and focal brain lesions. Rotational acceleration is the most common cause of DAI. Compared with focal brain lesions, in DAI skull fractures are much less commonly seen. There is no lucid interval associated with DAI whereas nearly half of the patients with focal brain lesions present with lucid interval. There is no difference between the two groups in terms of the amount or prevalence of hypoxic brain damage or...
presence of brain swelling\(^{(6,9)}\). However the intracranial pressure elevation is significantly higher in patients with focal brain lesions. Of the three characteristic lesions seen in diffuse impact injury, focal necrosis or hemorrhage in the corpus callosum is the most clearly delineated. The presence of gross callosal damage in DAI has been reported from 15 to 30\%\(^{(10,13)}\). Microscopic lesions in the corpus callosum are far more common having been reported in 100 of these patients. These callosal lesions are considered to be caused by lateral stretching of the corpus callosum owing to dorsoventral flattening of the head at the moment of the impact\(^{(2,7,9)}\). In addition focal contusion and laceration type injures are commonly seen in adjacent structures like fornix, cingulated gyri, septum pellucidum, caudate nuclei and dorsal thalamus\(^{(8,13)}\).

The second classic lesions in DAI is the hemorrhagic necrosis on the dorsolateral quadrant of the rostral pons next to the brachium conjunctivum\(^{(2,9,13)}\). This lesion is not due to impaction of pons against the incisura of the tentorium (an idea once widely accepted) and is an indirect lesion seen to be associated with DAI. These brain stem lesions are always hemorrhages in those who survives and sometimes appear as discrete hematomas\(^{(2,6,7)}\). With time, the hemorrhages are resorbed leaving behind the shrunken and hemosiderin-stained areas.

The third characteristic lesion in DAI is the reactive axonal swelling from tearing of the axons. Reactive axonal balls are best seen with silver impregnation stains and increase in prominence with prolonged survival time. Traction balls are seen as early as three hours post DAI\(^{(9,12)}\). Degeneration of the corticospinal and corticobulbar tracts may become so advanced that these tracts appear very pallid when stained with silver or Luxon fast.

In DAI, stresses induced by movement of the head are sufficient to injure numerous axons throughout the brain. However contact phenomena are not essential in causing these injuries\(^{(4,8)}\). Rather the head must undergo acceleration in particular direction to cause DAI. If the magnitude of the injury is enough, many axons are physically disrupted and small vessels torn. Later microglia migrate into the areas of axonal damage\(^{(7,8)}\). With destroyed axons and degeneration of myelin, lipid is produced which is stainable with Marchi techniques and Sudan black\(^{(10,12)}\). Total brain mass in eventually decreased resulting in atrophic appearance of the brain.

**CASE PRESENTATION**

The patient is a 22 year old male who was on a scooter and hit by a car. The patient sustained a right tibial plateau fracture for which he underwent an open reduction and internal fixation and also a left femur fracture for which he had a rod placement. Upon his arrival patient was GCS of 15, responding and moving all extremities. A day after his orthopedic operation, the patient suddenly deteriorated into GCS of 3. On neurological exam the patient had decorticate posturing. He was unresponsive to pain stimuli. The pupils are enlarged bilaterally but partially reactive to light. Patient was subsequently intubated and sedated. Three serial CTs of the brain were all negative (Figure 1 and 2).

The diagnosis of diffuse axonal injury was made. There is evidence of hemorrhage within the corpus callosum classic for DAI. The patient also has the diagnosis of factor IX deficiency for which he is receiving appropriate treatment. Figure 1 and 2: normal appearance of brain in diffuse axonal injury at the time of injury (Figure 1 ) and three days after the trauma (figure 2). Note minor diffuse edema three days post trauma.

The patient was managed conservatively and placed on a low dose steroid. He remained unresponsive and with a GCS of

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4-5 for 10 days. After those 10 days, the patient became arousable. Within the next day, his GCS score returned to a 15. He still continues to have multiple focal deficits including 0/5 motor in his left arm and a feeling of weakness in his legs, but he is able to follow commands and respond to questions.

DISCUSSION
There is controversy surrounding the role of factor IX deficiency in sudden onset of diffuse axonal injury in this patient. There is paucity of studies about the role of coagulation pathway deficiency and diffuse axonal injury. On the other hand this may be an atypical variation of DAI presenting with a lucid interval. Perhaps the period during which the patient was responsive with GSC of 15 was a form of lucid interval. However lucid interval is much more commonly associated with focal brain lesions and has never been documented with DAI. The precipitating factors to sudden onset of deterioration in ICU patients and delayed DAI are yet to be investigated.

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