

## Paraplegia: An Uncommon Presentation of Parasagittal Contusions; A Case Series

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### ABSTRACT

**Background:** In our hospital setting, the presence of a sudden bilateral lower limb paraplegia following trauma directs the mind of the clinician towards injury to the spinal cord; however, cerebral causes of paraplegia do occur even though such presentations are rare entities after traumatic brain injury. Patients presenting with acute inability to move both lower limbs were diagnosed to have spinal cord injury following multiple trauma or road traffic accident by casualty staff.

**Methods:** We report three cases seen over a period of two years, with the patients presenting with a history of acute paraplegia following trauma: assaults or road traffic accident.

Further evaluation revealed alteration or loss of consciousness and evidence of injuries to the skull. Neurological examinations revealed flaccid weakness of the lower limbs with power of grade 0 (MRC grade: 0/5), all sensory modalities of the lower limbs were preserved. Normal anal tone and superficial abdominal reflexes; there were no areas of tenderness or deformity over the spine.

**Results:** Initial radiologic evaluations of the spine showed normal findings, and all patients had computed tomography (CT) of the brain that revealed acute bilateral parasagittal contusions in the fronto-parietal region. Following active physiotherapy over a period of three to seven (3-7) weeks, all patients regained muscle power of at least 4/5, ability to walk, and were later discharged from the hospital.

**Conclusion:** A high index of suspicion is needed in diagnosing paraplegia secondary to different aetiologies such as parasagittal contusion. A low threshold in requesting for neuroimaging scan of the brain is required, although, Computed tomography (CT) done within first 1-3 days may be falsely negative.

**Keywords:** Paraplegia, Contusions, Parasagittal

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### Introduction

In our hospital setting, the presence of a sudden bilateral lower limb paraplegia following trauma directs the mind of clinician towards injury to the spinal cord; however, the cerebral cause of paraplegia does occur even though such presentation is a rare entity after traumatic brain injury. Patients presenting with acute inability to move both lower limbs were diagnosed to have spinal cord injury following multiple trauma or road traffic accident by our casualty observation staff. In the developed world many patients attend trauma and accident and emergency departments with head injuries. Approximately 300/100,000 population/year required admission and out of this 9/100,000 die (5000/year in Britain). Most of the deaths are inevitable while some are potentially preventable; however, such cannot be said in our environment, since there is no adequate data to elucidate the exact number per population. The leading causes of head injuries include vehicular crashes, falls, and assaults which affects mostly young or working age groups.

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	DOI: 10.31173/bomj.bomj_105_15



Once head injury has occurred, nothing can alter the impact of the initial damage, and brain damage occurs both as effects and as a result of the development of secondary complications. Translation of kinetic energy into passive parenchymal damage which could be compressive, tensile and shearing strain contributes to the tissue damage in the form of mechanical loading of the brain and will result in a variety of effects which includes cerebral contusions, laceration of the brain, diffuse axonal injury, epidural, subdural and intra-cerebral hematoma.

### **Case presentation**

#### **Case 1**

A 20-year-old cattle rearer presented to us with a 5-day history of inability to walk following an assault by some men following an altercation earlier on.

He sustained multiple injuries to the head and other parts of his body, said to have had transient loss of consciousness, and no bleeding or cerebrospinal fluid leakage from the nose, ears, and mouth.

He noticed that he cannot move his lower limbs after that and had pains on his lower back. Other systems were normal.

On examination, he was found to be conscious, alert with a Glasgow coma scale score of 15/15 and pupils were both normal-sized and reacting to light, no cranial nerve deficits. The cardiovascular, respiratory and abdominal examination were normal.

Neurological examinations showed power on both upper limbs was grade 5/5 in all muscle groups, while both lower limbs were flaccid with a power of grade 0 (MRC grade: 0/5), all sensory modalities of the lower limbs were preserved. Normal anal tone and superficial abdominal reflexes, there were no areas of tenderness or deformity over the spine.

His thoraco-lumbar x-ray and Magnetic Resonance Imaging (MRI) of thoracolumbar spine revealed normal findings, which did not correlate with the clinical presentation of the patient, however, his Computed tomography of the brain showed bilateral frontoparietal

contusions with extensive oedema (figure 1). A final diagnosis of parasagittal contusion was made, and he was managed non-operatively with active physiotherapy.

He gradually regained the power on his lower limbs with the grade of 4<sup>+</sup>/5 and walked without support after six weeks on admission and was discharged home to see at outpatient clinic.

#### **Case 2**

A 45-year-old man farmer who was seen and later referred from another hospital to our centre with a diagnosis of a spinal cord injury. He presented with two-day history of inability to move both lower limbs after being attacked by armed robbers, there was no loss of consciousness, but had multiple lacerations on the legs and back, no fractures. Other systems were essentially normal.

On examination, he was fully conscious with a Glasgow coma score of 15/15, pupils were normal. There was normal muscle power in both upper limbs, and 0/5 in both lower limbs that were flaccid, sensory modalities of the lower limbs were preserved, he had normal anal tone and superficial abdominal reflexes. There were no areas of tenderness or step-in deformity over the spine. Other systemic examinations were normal.

The patient had thoracolumbar x-ray and MRI which were normal. His computed tomography (CT) of the brain showed bilateral fronto-parietal contusion with oedema and a diagnosis of parasagittal contusion was made.

The patient was managed non-operatively with physiotherapy. He gradually regained the power on his lower limbs 4<sup>+</sup>/5 in all muscles groups and walked after five weeks on admission.

#### **Case 3**

A 25-year-old trader who was referred from a peripheral hospital with a 5-day history of inability to walk following road traffic accident.

He was knocked down by a fast-moving car (hit and run) while riding on a motorbike and said to have had transient loss of consciousness and inability to walk after that. No bleeding from craniofacial orifices or convulsion and had no significant premorbid condition.



The patient had thoracolumbar x-ray and MRI which were normal. He had brain computed tomography (CT) scan which showed bilateral fronto-parietal contusion with oedema.

The patient was managed non-operatively with physiotherapy. He gradually regained the power on his lower limbs 4/5 and walked with support after seven weeks on admission.

### Discussion

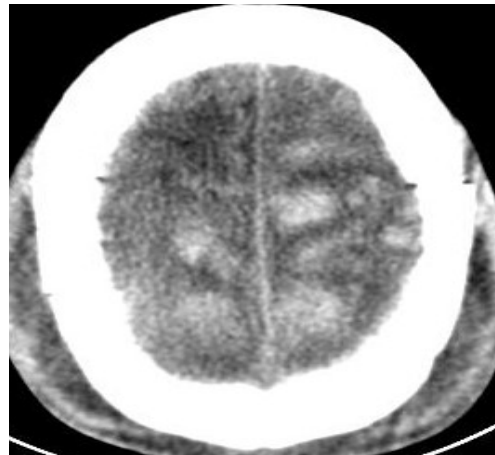
Focal contusions usually occur in the form of high-density lesions and may occur as coupe or counter coupe. They most commonly occur/involve the frontal and temporal lobes and do not occupy much space in the beginning but may increase in size afterward within days to cause significant intracranial hypertension. Depending on the mechanism, they may be multiple or occur bilaterally.<sup>1</sup>

Gliding contusions are a type of focal contusion that occur in the parasagittal region involving the cortex and adjacent white matter of the superior margin of the cerebral hemisphere usually along the falx cerebri which arises due to rotational mechanism rather than contact forces and associated with diffuse axonal injury.<sup>2,3</sup>

Paraplegia can result from a disruption at any level of the neural motor pathway, starting from the frontal parasagittal cortical neurons<sup>4</sup>, motor neurons from the motor cortices with some originating from the sensory cortices including the supplementary areas travel down to the thoracolumbar regions of the spinal cord as upper motor neurons, the peripheral lumbar spinal cord roots and peripheral nerves. Acute paraplegia most commonly results from a thoracic spinal cord column fracture and spinal cord injury or vascular injury supplying the cord<sup>4</sup>.

Paraplegia from cerebral contusion is extremely rare, though there were reported cases of this rarity, approximately 4% of all cortical contusion occurs in the superior fronto-parietal cortex.<sup>2,8</sup> Fewer would happen in the specific parasagittal cortex responsible for lower extremity movements.<sup>5,9</sup> Some also reported mono-paresis of the lower limb due to traumatic intraparenchymal contusions.<sup>7,9</sup> It is of interest to note that all the three cases were initially diagnosed to

have spinal cord injury and not parasagittal contusions leading to bilateral lower limb paraplegia mimicking acute spinal cord injury. Their initial lumbosacral Magnetic Resonance Imaging were negative and computed tomography of the brain in all the patients showed parasagittal contusions hence leading to the diagnosis of paraplegia due to parasagittal contusions (see figures 1 and 2). We also observed that the patients after going through the MRI of the spine and CT brain were unable to do repeat CT brain after several weeks of physiotherapy having had a useful recovery of motor functions in the lower limbs.



**Figure 1:** Computed Tomography (CT) Brain of a patient with bilateral fronto-parietal contusion



**Figure 2:** Computed Tomography (CT) Brain of a patient with left-sided contusion and oedema on the right side

### Conclusion

A high index of suspicion is required in diagnosing paraplegia secondary to different aetiology such as parasagittal contusion. A low

threshold in requesting for neuroimaging scan of the brain is needed, although, CT done within first 1-3 days may be falsely negative.

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**Cite this Article as:** Babagana Mohammed, Nasiru Ismail Jinjiri, Mohammad R. Mahmud, Ali Lassieni. Paraplegia: An Uncommon Presentation of Parasagittal Contusions; A Case Series. **Bo Med J** 2018; 15(2):64-67 **Source of Support:** Nil, **Conflict of Interest:** None declared.

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