Case Report

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Firework discharge leading to cerebral contusion and motor paraplegia

Matthew Zeller[1], Paul Bluhm[1], Marika Gassner[1,2], Viviane Ugalde[3], Travis Abele[4] and Mary Condron[1,2,5]

Abstract

A 57-year-old male sustained a blunt head injury after discharging a mortar firework off the vertex of his head. Physical examination revealed a stellate scalp lesion and pure bilateral leg paraplegia. Initial spinal computed tomography and magnetic resonance imaging were negative for pathology. Initial head computed tomography revealed open, nondisplaced, frontal, and parietal skull fractures with underlying subdural and subarachnoid hemorrhage. Follow-up magnetic resonance imaging one week later showed bilateral precentral gyri frontal lobe contusions involving the lower extremity motor cortices and subcortical white matter extending anteriorly into the region of the supplementary motor areas. The patient's complete paraplegia informed the subsequent hospital rehabilitation. However, motor recovery was more rapid than anticipated, with the patient regaining ambulatory function before inpatient rehabilitation discharge after 27 days of hospitalization. He continued to have issues with spasticity after discharge. We discuss the current literature surrounding paraplegia secondary to head trauma and the recovery that follows. Firework misuse is a known cause of head injury but has not been recorded as a cause of isolated bilateral paraplegia. Isolated precentral gyri contusion must be considered in patients presenting with paraplegia following trauma to the vertex of the head and normal spinal imaging. We show the importance of repeat imaging to follow the evolving nature of traumatic head injuries presenting with paraplegia. We also illustrate the variability in rehabilitation planning and the need for adjustment in rehabilitation planning for paraplegic patients following head trauma.

Keywords

Traumatic paraplegia, bilateral precentral gyri contusion, firework injury, pure motor paralysis, traumatic brain injury, rehabilitation

Introduction

While bilateral lower extremity paralysis is common following spinal cord injury, isolated bilateral lower limb paralysis following cerebral vertex contusions is less commonly diagnosed and scarcely reported in the literature. The case presented here is one of isolated acute bilateral leg paralysis following blunt vertex injury and subsequent precentral gyri contusion from discharging a mortar firework. Ambulatory recovery following the injury was more rapid and complete than would have been expected from a spinal cord injury with the same level of deficit.

Case presentation

A 57-year-old Caucasian male presented to the emergency department (ED) with a stellate scalp laceration and complete bilateral lower extremity paraplegia after discharging a mortar firework while holding it over his head. On admission, his Glasgow Coma Scale (GCS) was 14 (E4V4M6) with a blood alcohol level of 115 mg/dL. The patient denied loss of consciousness. No additional significant laboratory findings were present. He

[1]College of Osteopathic Medicine of the Pacific-Northwest, Western University of Health Sciences, Portland, USA
[2]St. Charles Medical Center, Trauma and Acute Care Surgical Services, Bend, USA
[3]The Center, Orthopedic & Neurosurgical Care & Research, Bend, USA
[4]Central Oregon Radiology Associates, Bend, USA
[5]Department of Surgery, Oregon Health & Science University, Portland, USA

Corresponding author:
Matthew Zeller, College of Osteopathic Medicine of the Pacific-Northwest, Western University of Health Sciences, Portland, OR, USA.
Email: matthew.zeller@westernu.edu

showed extreme anxiety prior to the recognition that he could not move his lower limbs. Initial non-contrast computed tomography (CT) of the brain revealed open, non-displaced, frontal, and parietal skull fractures (Figure 1) with underlying subdural and subarachnoid hemorrhage (Figure 2). Complete imaging of the spine with CT and magnetic resonance imaging (MRI) revealed no abnormalities.

On neurological examination, the upper extremity motor and sensory exam were normal, and the lower extremity sensory exam was intact, including proprioception, light touch, temperature, vibration, and pinprick. Motor examination showed 0/5 muscle strength across all muscles of the lower extremity. Deep tendon reflexes (DTRs) were 2+/4 at patellar tendon and Achilles tendon bilaterally. Babinski exam showed right toe upgoing and left toe mute. On hospital day two, he developed 2–3 beat clonus that persisted throughout his acute hospital stay. Initially, he displayed normal bowel function but experienced urinary retention that led to Foley catheter placement with resolution after four days. Although not a spinal cord injury, his exam would have been consistent with an American Spinal Injury Association (ASIA) Impairment Scale score of B thoracic level and incomplete B due to sensory sparing. ASIA score was performed for baseline assessment of his deficit and to follow the progression of injury and recovery. CT with contrast performed on hospital day two showed small bilateral parietal vertex parenchymal contusions with vasogenic edema but no contusions within the premotor cortex. Brain MRI was not obtained at this time due to the patient's severe anxiety related to his injury. His intensive care unit stay was extended due to his requirement for intravenous dexmedetomidine and ketamine to control his anxiety. It was notable that his anxiety did not appear to be related to alcohol use disorder or withdrawal.

Following a week of hospitalization in the acute care setting, the patient was transferred to inpatient rehabilitation, at which time he required maximum assistance for bed mobility, transfers dependent on Hoyer lift bed to wheelchair. Gait and stairs were not tested. He displayed no active range of motion of the lower extremities, retained sensation throughout, with some hypertonia at the ankles with 2–3 beat clonus. MRI obtained while in inpatient rehabilitation showed bilateral precentral gyri frontal lobe contusions involving the lower extremity motor cortices and subcortical

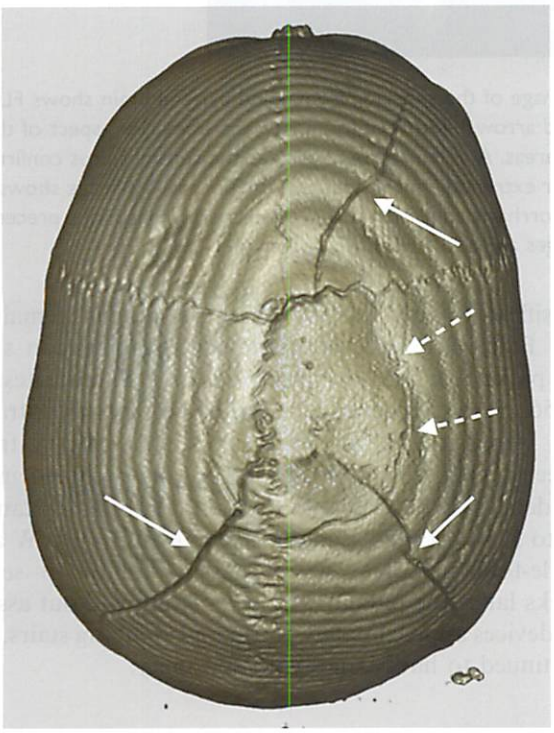

Figure 1. Initial non-contrast CT of the brain. A 3D surface shaded image of the vertex of the skull demonstrates linear fractures involving the frontal and parietal skull (solid arrows) and an ovoid fracture of the parietal bone (dotted arrows).

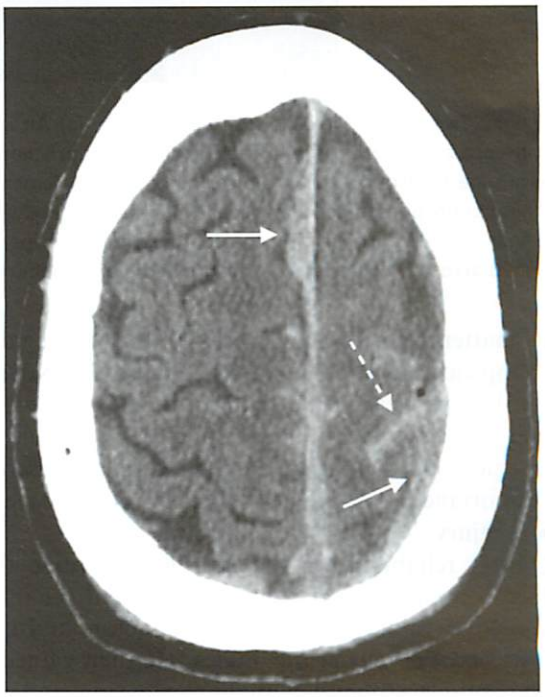

Figure 2. Initial non-contrast CT of the brain. An axial CT image in soft tissue algorithm shows left frontal subarachnoid hemorrhage (dotted arrow) and right parafalcine and left superior convexity subdural hemorrhage (solid arrows). Note the absence of contusion evident in the superior frontal brain parenchyma.

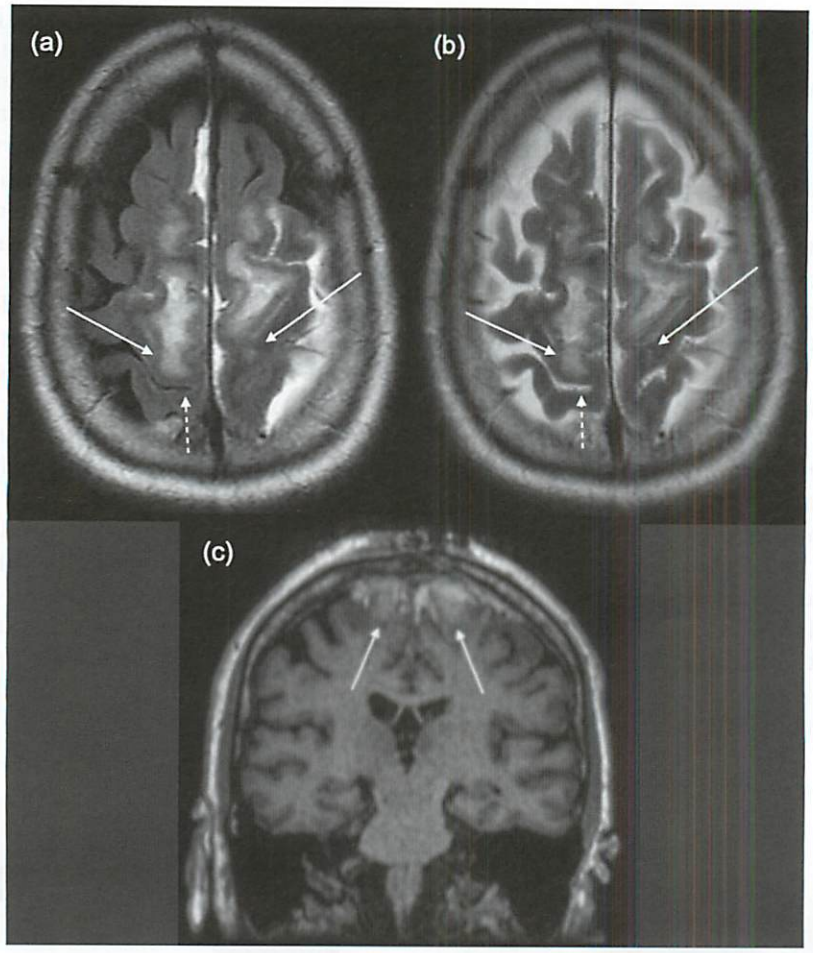

Figure 3. Two-week follow-up MRI of the brain. (a) An axial FLAIR image of the superior frontal and parietal brain shows FLAIR hyperintense contusion in the bilateral frontal lobe precentral gyri (solid arrows) with extension into the posterior aspect of the bilateral superior frontal gyri in the region of the supplementary motor areas. A dotted arrow indicates the central sulcus confirming that these contusions affect the precentral gyri in the region of the lower extremity motor cortices. (b) An axial T2 image shows the same findings (c) A coronal T1 image demonstrates T1 hyperintense hemorrhagic contusion within the bilateral frontal lobe precentral gyri (solid arrows). Hyperintense subarachnoid and subdural hemorrhages are also present in images (a–c).

white matter and extending anteriorly into the region of the supplementary motor areas (Figure 3). No additional lesions were found within the rest of the intracranial motor system. These findings were not as conspicuous on the patient's earlier CTs, underscoring the importance of MRI in assessing traumatic neurological injury.

During rehabilitation, he first gained trace thigh abductor strength on day five of inpatient rehabilitation, along with 3+ DTRs at the adductors and knees with 2–3 beat clonus at the ankles. He then gained 2/5 graded muscle strength with hip extension and knee flexion, and ankle DTRs were graded 4+ with sustained clonus bilaterally. After just over two weeks in inpatient rehabilitation, he progressed to standing with the use of parallel bars and was making progress on sit-to-stand transfers. Hip flexion measured 3−/5, knee flexion measured 3/5, and knee extension measured 5/5. Ankle dorsiflexion and extensor hallucis longus remained 0/5. Following a 27-day inpatient rehabilitation stay, the patient showed an improved Berg Balance test of 56/56. He had independent supine bed mobility; transfers were sit-to-stand with supervision and a front wheeled walker (FWW), sit pivot was modified independent, and he was able to ambulate multi-distances up to 50 m between seated rest breaks using FWW and ankle-foot orthoses. At outpatient follow-up seven weeks later, the patient was ambulating without assistive devices approximately 130 m and climbing stairs. He continued to have issues with spasticity.

Discussion

Only two other case reports exist describing traumatic non-spinal non-hemorrhagic paraplegia.[1,2] Due to the corticospinal motor tracts coalescing at the pyramids of

the spinal cord, bilateral paraplegia is more commonly seen in traumatic spinal cord injuries or from spinal cord compression from hemorrhage.[3,4] In any adult trauma patient with paraplegia or quadriplegia and negative spinal imaging, spinal cord injury without radiographic abnormality (SCIWORA) must be considered. Although prior to diagnosis of SCIWORA, attention should be placed on eliminating brain lesions with further imaging. The mechanism of injury is an essential factor in the development of a differential diagnosis and plan for patients with isolated paraplegia following trauma. In the initial workup of a patient with vertex head trauma, non-spinal causes of paraplegia must be investigated. Injury to the posterior frontal cortex following head trauma is increasingly reported as a cause of loss of motor function in the lower extremities. Monoplegia, as a result of hematoma or cerebral contusion following motor vehicle accident head trauma, has also been well-described in the literature.[5,6]

Non-spinal causes of paraplegia following trauma, while less commonly diagnosed, are important to investigate following negative spinal imaging. A cranial cause of paraplegia was considered early in our patient's clinical course due to the presence of a parietal fracture and negative spinal CT and MRI. In cases where cranial fracture is not present, consideration of cerebral causes of paraplegia is often delayed.[6] In our case, initial head CT showed subdural and subarachnoid hemorrhage but did not delineate precentral parenchymal involvement, consistent with previous reports of head trauma leading to paraplegia.[1,2] Continued imaging including CT in the acute course, followed by brain MRI, was essential to differentiate the presence of bilateral superior frontal lobe contusions in addition to subdural, subarachnoid, and surrounding vasogenic edema.

Head injury following the misuse of fireworks is a common cause of firework-related ED visits and can lead to death.[7] Head and neck injuries account for up to 42% of all firework injuries, with burns being the most common injury pattern.[8] Projectile trauma from military combat is the closest mechanism of injury to this case and has been reported to cause motor paralysis with sensory deficits.[9] To our knowledge, this is the first case report describing firework misuse as the cause of paraplegia.

This case also considers the variability in rehabilitation planning for the patient. The rehabilitation course was initially determined based on his complete paraplegia, with expected recovery being unknown.[10] The patient was expected to need full assistance with sit-to-stand transfers and a power wheelchair due to his body habitus. Continued outpatient rehabilitation would focus on returning to ambulatory status after discharge. Motor recovery in patients with traumatic brain injuries (TBIs) have shown to be greater than in a comparative stroke population and can occur up to six years after injury.[11,12] Initial severity of motor weakness has been tied to longer recovery time, while meaningful clinical recovery can continue to occur several years following a severe TBI. Our patient displayed rapid improvement in lower extremity muscle strength during inpatient rehabilitation, ambulating prior to discharge. This refocused discharge planning on further ambulatory function, despite the patient's initial complete paralysis, which suggested ambulation would take more prolonged rehabilitation.

Conclusion

We report the case of a 57-year-old male who presented to the ED with isolated lower extremity motor paralysis after discharging a firework off the vertex of his head. The blast effect resulted in a bilateral precentral gyri contusion causing isolated paraplegia. Presenting with an unusual injury with an unclear prognosis at admission, our patient with pronounced injury on MRI and complete paraplegia demonstrated nearly full motor recovery. This case report adds to the body of knowledge regarding motor recovery after TBI.

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None.

Contributorship

All authors contributed to data collection and study design. MG, VU, MC, and TA interpreted the data. MZ and PB completed the literature review. All authors reviewed and edited the manuscript and approved the final version of the manuscript.

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ORCID iDs

Matthew Zeller https://orcid.org/0000-0002-6061-2215
Paul Bluhm https://orcid.org/0000-0002-3642-1224
Marika Gassner https://orcid.org/0000-0002-9285-3064
Viviane Ugalde https://orcid.org/0000-0003-2747-5842
Travis Abele https://orcid.org/0000-0002-3891-3687

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