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Rare Case of Bilateral Cerebellar Hemorrhage in a Male Boxer

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Abstract The sport of boxing carries with it the risk of brain injury with acute and chronic neurological injuries such has contusion, concussion and intracranial bleeding has been reported. The force sustained from a boxing blow can be as powerful as being hit with a 6-kg wooden mallet striking at 20 mph. Bilateral cerebellar hemorrhage secondary to trauma is a rare entity that has not been reported in the literature previously. One can only speculate as to how a boxer could potentially develop a cerebellar bleed. Regardless of whether a very powerful punch or the act of falling back and hitting the occipital region directly on the floor, it is very likely that a predisposing factor exist. We present you a rare case of a 26 year-old male boxer who was knocked out in a boxing match and was noted to have acute parenchymal and subarachnoid hemorrhage bilaterally in the cerebellar hemispheres. Our report highlights the need to consider cerebellar bleed in boxers who present after ataxia after boxing/knocked out.

Keywords: *bilateral cerebellar hemorrhage, boxing*

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1. Introduction

The sport of boxing carries with it the risk of brain injury [1]. Cerebral microhemorrhages [2], brain stem hematoma [3], subdural hematoma [4] with boxing have been previously reported. Boxing related neurological damage can be acute or chronic [5]. Acute neurological injuries include concussion, intracranial hemorrhage, brain contusion and traumatic brain injury is among the chronic neurological changes from boxing [6]. Bilateral cerebellar hemorrhage secondary to trauma is a rare entity that has not been reported in the literature previously. We present you a rare case of a 26 year-old male boxer who was knocked out in a boxing match and was noted to have acute parenchymal and subarachnoid hemorrhage in bilateral cerebellar hemispheres.

2. Case Presentation

A 26 year old male presented with unsteady gait after he was knocked out in a boxing match. He was wearing a protective headgear however it slid during the match and subsequently, multiple strikes were sustained to the face and head. On arrival to the emergency department he was afebrile, heart rate was 123 beats/minute, blood pressure was 123/78 mmHg, and respiratory rate was 18 breath per minute. Physical examination of chest, heart and abdomen was within normal limits. Neurological examination has been summarized in Table 1. He was awake with Glascow Coma Scale score (GCS) of 14 but had some memory impairment. His metabolic panel revealed Blood Urea Nitrogen (BUN) of 26 mg/dl, Creatinine (Cr) of 4.39 mg/d, creatinine phosphokinase (CPK) > 160,000U/L, potassium (K) of 8.1mEq/L and lactate of 5.8 mmol/L.

Electrocardiography (EKG) revealed normal sinus rhythm with first degree atrioventricular block. EKG later revealed atrial fibrillation. Computed tomography (CT) of the head revealed acute parenchymal and subarachnoid hemorrhage in bilateral cerebellar hemispheres; left greater than right with surrounding edema (Figure 1). Magnetic resonance imaging of brain revealed subarachnoid hemorrhage in the cerebellar hemispheres bilaterally with moderate surrounding edema (Figure 2). Patient was subsequently intubated due to worsening mental status and acute respiratory failure. Continuous veno-venous hemodialysis (CVVHD) was initiated to manage acute renal failure, hyperkalemia and rhabdomyolysis. Neurosurgery performed ventriculostomy with drain which revealed clear cerebrospinal fluid that jetted out under pressure. Following ventriculostomy, the patient was noted to have absent brain reflexes with fixed and dilated pupils. Neurology evaluated the patient and declared him brain dead after positive apnea test.

Table 1. Neurological examination at the time of presentation

Cranial nerves II-XII		Grossly intact
Muscle Strength:		
	Right	Left
Deltoid	3/5	3/5
Biceps	3/5	3/5
Triceps	3/5	3/5
Grip	3/5	3/5
Hand intrinsic muscle	3/5	3/5
Psoas	4/5	4/5
Quadriceps	4/5	4/5
Hamstring	4/5	4/5
Dorsiflexion	4/5	4/5
Plantar flexion	4/5	4/5
Extensor hallicis longus	4/5	4/5
Sensory	Normal in the C4-T1 distributions bilaterally, and the L2/S1 distributions bilaterally	

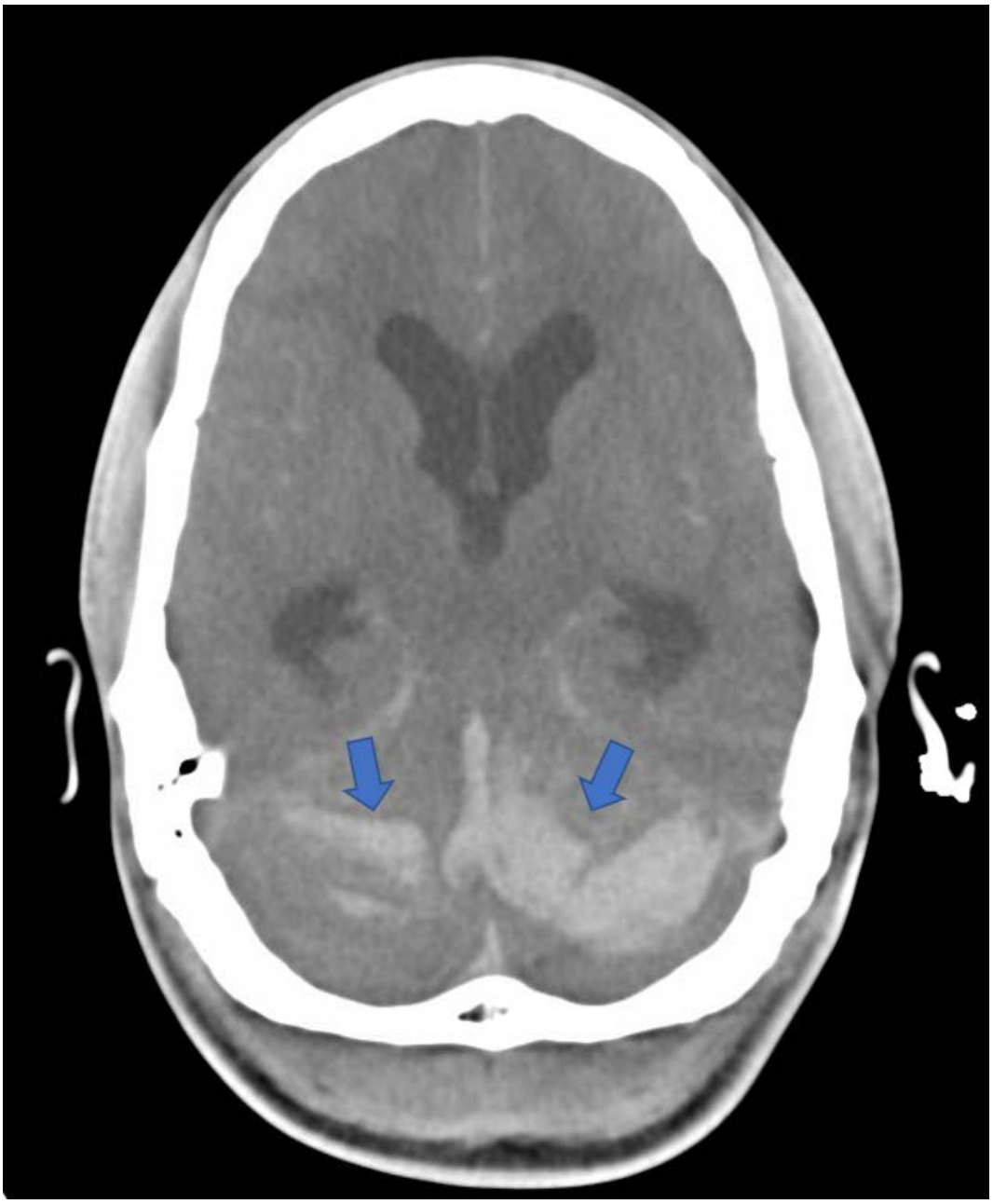

Figure 1. CT brain demonstrating bilateral cerebellar hemorrhages with extension into the Vermis

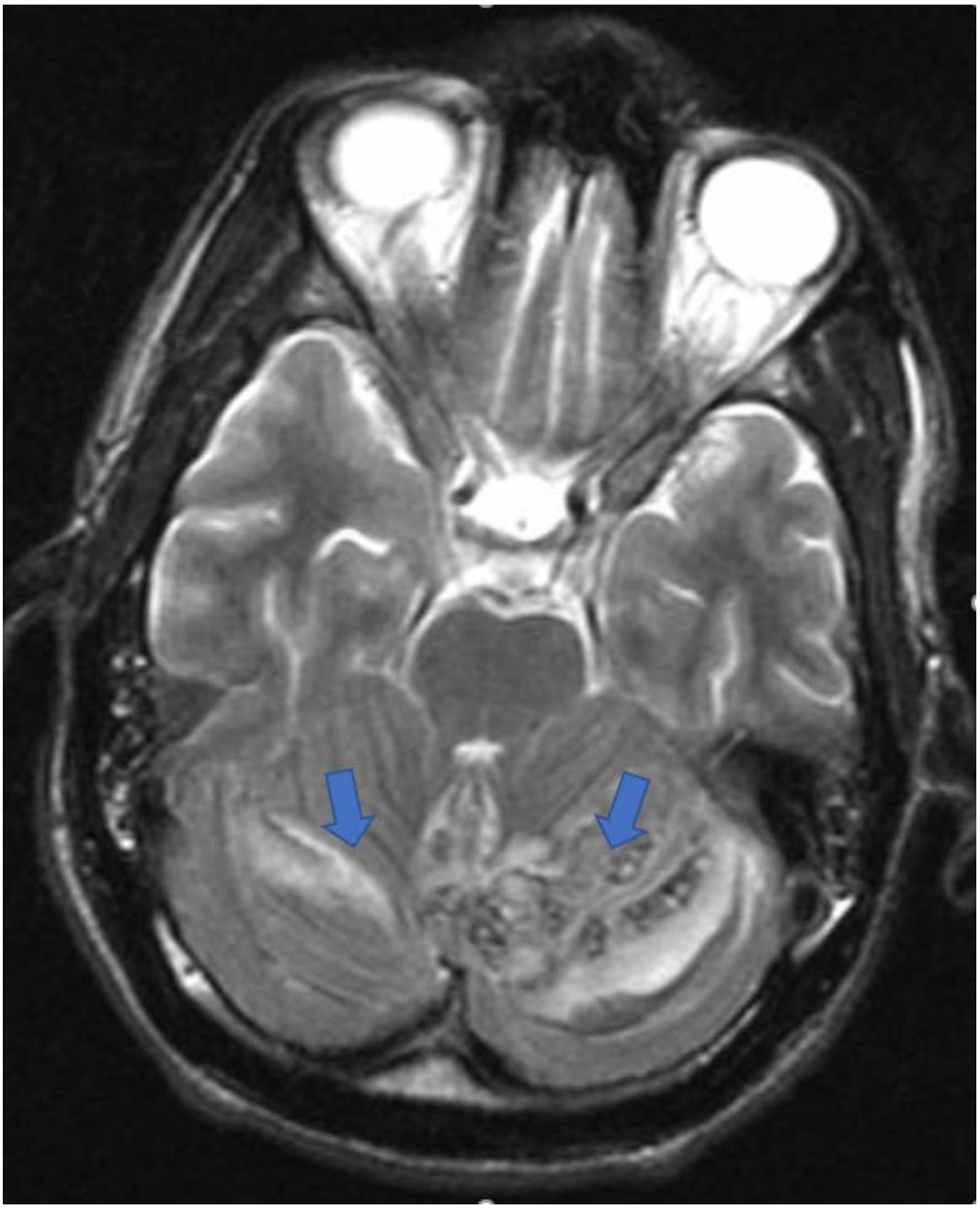

Figure 2. MRI brain demonstrating subarachnoid hemorrhage in the cerebellar hemispheres bilaterally with moderate surrounding edema

3. Discussion

Boxing is a sport that consists of two opponents fighting with padded gloves that weighs between 8-10 ounces with the ultimate goal of either knocking out the opponent or delivering more punches before reaching the allotted time. Per the World Boxing Federation, allotted time for a boxing bout is described as a total of 10-12 rounds depending on the event with each round consisting of a 3-minute duration, with a 1-minute rest period between rounds. Fighters are grouped in different classes depending on their weight to assure a comparable fight [6]. Due to the nature of the sport, injuries are quite common and according to Unterharnscheidt et al, intracranial injuries in boxing can affect any area of the brain. Especially involved are the large neurons of different layers of the cerebral cortex, and the purkinje cells of the cerebellum [7]. In one study, the overall incidence rate of injury in boxing was 17.1 per 100 boxer-matches or 3.4 per 100 boxer-rounds. Even more a high rate of injuries in boxing like lacerations to soft tissues or contusions are the most common [8] despite wearing padded gloves. Hand injuries have been found to be the second most common type of injuries. The force sustained from a boxing blow can be as powerful as being hit with a 6-kg wooden mallet striking at 20 mph [9]. Even though head injuries with neurological symptoms occur at a rate of 4.2% [10], it is no surprise that significant consequence even fatalities can happen if neurological dysfunction is sustained given how forceful and impactful a punch can be. Furthermore, fatigue and cognitive awareness starts to decline as a match goes on. This in turn leads to relaxation of cervical muscles, lowering the effective head mass resisting a head blow. The consequence of a lowered resistance to head blows puts a boxer at a high risk for head injuries due to increase in translational and rotational accelerations with each

subsequent blow [11,12]. Intraparenchymal hemorrhage injuries are associated with linear acceleration [13] with subdural hemorrhage rates as high as 75% of all acute brain injuries and are the most common cause of death in boxing [14,15]. Unterharnscheidt further supports this finding in that the most common cause of death due to boxing injury is a subdural hemorrhage [7,11]. Cerebellar hemorrhages seem to be very rare and are hardly reported in the literature. One can only speculate as to how a boxer could potentially develop a cerebellar bleed. Regardless of whether a very powerful punch or the act of falling back and hitting the occipital region directly on the floor, it is very likely that a predisposing factor was the culprit. In a paper that examined the brain of 15 boxers, it would seem as though the most common cerebellar injuries were scarring, atrophy, fine fibrous gliosis and demyelination with considerable Purkinje and granule cell loss [16]. These ongoing changes in a boxer's cerebellar region with continuous repeated blows throughout one's career can predispose to an acute bleed if the right circumstances are met. In earlier works by Linderberg and Freytag, they described hemorrhage in the subarachnoid space between the folia of the tonsils. This damage has been ascribed to the brain being forced into the Foramen Magnum, which along the way caused shearing in the medulla oblongata and cerebellar tonsils [17]. Furthermore, authors believe that the origin of glial scarring and the loss of Purkinje cells probably transpired in a similar manner. While there isn't a lot of data in terms of management of acute cerebellar bleed sustained during or shortly after a boxing match, the management and outcome of subdural bleeds can be considered. Whilst following the American Association of Neurologic Surgeons guidelines for the management of severe head injury in a study evaluating acute subdural hematoma caused by boxing, 10 boxers were compared against 26 non-boxers with acute subdural hematoma were classified into three groups in accordance to their consciousness level. Transient unconsciousness type consisted of boxers who had returned to alertness within an hour from the time of injury. Lucid interval type entailed boxers who presented with neurological deterioration with a lucid interval from ten minutes to an hour after knockout and thirdly a deterioration of consciousness type. After resuscitation measures, stabilization efforts and evaluation by a neurologist, all patients underwent an axial cranial CT. If acute neurological deterioration or increased intracranial pressure was subsequently noted on examination or ensuing CT, operative management was initiated. All 10 boxers in the study underwent extensive decompressive craniotomy within four hours of surgery and the mortality rate was reported to be 30% [17,18]. Correspondingly, Koyama et al reported a mortality rate of 33% when craniotomy was done within 2 hour [19].

4. Conclusion

We presented a rare case of bilateral cerebellar bleeding in a 26 year old boxer complicated by multi-organ failure and subsequently brain death. The case highlights the need to consider cerebellar bleeding in the differential diagnosis of boxing brain injury and the institution of prompt therapeutic interventions as the case requires.

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