Case Report

Mechanisms of mid-thoracic spine fracture/dislocation due to falls during horse racing: A report of two cases

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**A R T I C L E  I N F O**

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**A B S T R A C T**

We reported two cases of jockeys who sustained fracture/dislocation of the mid-thoracic spine due to traumatic falls during horse racing. We examined the injury mechanism based upon the patients' diagnostic images and video footage of races, in which the accidents occurred. Admission imaging of patient 1 (a 42 years old male) revealed T5 burst fracture with bony retropulsion of 7 mm causing complete paralysis below T5/6. There existed 22° focal kyphosis at T5/6, anterolisthesis of T5 relative to T6, T5/6 disc herniation, cord edema and epidural hemorrhage from T4 through T6, and cord injury from C3 through C6. Admission imaging of patient 2 (a 23 years old male) revealed T4/5 fracture/dislocation causing incomplete paralysis below spinal level. There existed compression fractures at T5, T6, and T7; 4 mm anterior subluxation of T4 on T5; diffuse cord swelling from T3 through T5; comminuted fracture of the C1 right lateral mass; right frontal traumatic subarachnoid hemorrhage; and extensive diffuse axonal injury. The injuries were caused by high energy flexion-compression of the mid-thoracic spine with a flexed posture upon impact. Our results suggest that substantially greater cord compression occurred transiently during trauma as compared to that documented from admission imaging. Video footage of the accidents indicated that the spine buckled and failed due to abrupt pocketing and deceleration of the head, neck and shoulders upon impact with the ground combined with continued forward and downward momentum of the torso and lower extremities. While a similar mechanism is well known to cause fracture/dislocation of the cervical spine, it is less common and less understood for mid-thoracic spine injuries. Our study provides insight into the etiology of fracture/dislocation patterns of the mid-thoracic spine due to falls during horse racing.

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**Introduction**

The most common region of the thoracolumbar spine sustaining fracture/dislocation is T12/L1 due to its transitional position from the stiff thoracic spine to the more flexible lumbar spine. An increased incidence of spinal fractures is also noted at the cervicothoracic junction, C7/T1. In their analyses of thoracolumbar spine injuries, Magerl et al. found that the majority of thoracic spine injuries (39%) occurred at T12. In comparison, the superior thoracic vertebrae each accounted for 0%–11% of the thoracic spine injuries. The ribs provide stiffness to the thoracic spine and absorb some energy during high energy traumatic loading thus reducing the loads transferred to the thoracic spine. The large traumatic load required to cause fracture/dislocation of the thoracic spine leads to neurological injury in the majority of cases.

Falls are the leading cause of injuries to jockeys during equestrian sports, particularly those that occur during jumping. Injuries to the head are more common than the spine, which may be associated with injuries to the extremities, thorax, face, pelvis, and/or abdomen. During racing, horses reach speeds up to 64 km/h which places both the jockey and horse at high risk of traumatic injury due to a fall. Triantafyllopoulos et al. reported three cases of fracture/dislocation to the mid-thoracic spine in jockeys due to falls occurring during horse racing. They reported burst fracture, bilateral facet dislocation, anterior dislocation, and compression fracture at T5 through T7 with neurological status ranging from grade A to grade E on the American spinal injury association impairment scale. They did not report head or cervical spine injuries. Apart from anecdotal clinical observations, little is known regarding the mechanism of fracture/dislocation to the mid-thoracic spine due to falls during horse racing.
The purpose of the present study was to report two cases of jockeys who sustained fracture/dislocation of the mid-thoracic spine due to traumatic falls during horse racing. We investigated the etiology and biomechanical mechanisms of injury based upon video footage of races in which the accidents occurred, examined the patients' medical records and diagnostic images, and synthesized prior case studies and biomechanical investigations.

Case reports

Injury causation

In both cases, video footage of races in which the accidents occurred was available. For case 1 (a 42 years old male), images from the rear camera demonstrated that the front of the horse went down first which caused the jockey to be thrown forward and downward. Front and side views demonstrated that the back of the jockey’s head and neck impacted and pocketed the ground first with his spine in a flexed position. During the initial impact, his head was oriented rearward relative to the direction of forward horse motion. Subsequently, his body rotated and his middle and back impact entered the ground, and he landed on his left side with his spine in a flexed position. The horse subsequently landed on the jockey. The jockey remained pinned underneath the horse for several minutes until the horse could be lifted up.

For case 2 (a 23 years old male), the front of the horse body went down first which caused the jockey to be thrown forward and downward. The jockey impacted the ground on his front with his spine in extension and his right leg initially straight and subsequently extended. Forward momentum caused his body to become inverted and his head and shoulders impacted and pocketed the ground with his spine flexed. He moved forward and upward, and landed on his right side and back.

Clinical imaging, diagnosis and treatment

When patient 1 arrived at the emergency room, he was unable to move his legs and complained of electricity in his upper arms, difficulty breathing and pain everywhere. Admission MRI demonstrated burst fracture of the T5 vertebra with a large fragment of the posterior T5 vertebral body retropulsed approximately 7 mm into the canal, which caused cord compression (Fig. 1). The T5/6 disc was herniated. Cord edema and epidural hemorrhage were observed from T4 through T6. There were fractures of the inferior endplate and T6 superior endplate with approximately 22° of focal kyphosis at T5/6 and anterolisthesis of T5 relative to T6. The fractures at T5/6 extended posteriorly into the posterior elements consistent with a Chance-type fracture. There were disrupted fractures of the spinous processes at T4, T5 and T6; laminar fractures at T5, T6 and T7; a left inferior facet fracture at T6, and right transverse process fractures at T6, T7 and T8. The height of the T7 vertebral body was decreased suggestive of compression fracture without retropulsion. Cervical spine injuries consisted of spinous process fractures at C3 and C4, extensive edema within the posterior paraspinal soft tissues from C1 through C5, and moderate edema of the prevertebral soft tissues. An abnormal T2/STIR hyperintense signal within the cervical cord was observed at C3 through C5. The patient had multiple rib fractures.

Emergency spinal surgery was performed on patient 1, consisting of decompressive laminectomies at T5 through T7, bilateral pediculectomy and decompression at T6, posterior fusion at T3 through T5, and decompressive laminectomies and posterior fusion at C3 through C6. Following surgery and rehabilitation, patient 1 continued to have complete paralysis below the T5/6 spinal level, pain at the level of T5/6, and occasional numbness in his arms.

Patient 2 was unconscious upon arrival at the emergency room. Reports indicated that he was able to move his hands following the accident but not his legs. Admission MRI demonstrated fracture/dislocation at T4/5 with compression fractures at T5, T6 and T7 (Fig. 2). Diffuse cord swelling was observed from T3 through T5 consistent with cord contusion. Anterior subluxation of T4 on T5 of approximately 4 mm was observed with anterior wedge compression deformity and comminuted fractures of the T5 vertebral body. There was mild retropulsion of the posterior T4 cortical margin on the right which caused canal narrowing. Posterior ligamentous injuries were observed most pronounced at the T3/4 interspinous ligament. Admission CT of his cervical spine demonstrated a comminuted fracture to the C1 right lateral mass. CT and MRI of his head and brain revealed a right frontal traumatic subarachnoid hemorrhage and extensive diffuse axonal injury. A fracture of the left medial fourth rib was observed.

Spinal surgery was performed on patient 2 the day following the accident, consisting of decompressive laminectomy at T4, T5 and T6 and posterolateral fusion at T2 through T8. During the surgery, the T5 lamina was observed to be free and connected only by soft tissues. Following the surgery and rehabilitation, patient 2 continued to have incomplete paralysis and tingling sensation but not feeling below the T4/5 spinal level, pain at the level of T4/5 and short-term memory loss.

Discussion

Prior clinical and epidemiological studies have identified T12/L1 as the most common region of the thoracolumbar spine sustaining fracture/dislocation. Magerl et al. found that the
The biomechanical mechanisms of these injuries are not well described in the literature. Prior biomechanical studies have found that cord compression is greatest transiently during traumatic loading as compared to the subsequent recoil position of the spine.\textsuperscript{11,12} This suggests that the transient cord compression during trauma may substantially exceed that documented clinically from admission imaging. Admission imaging is not able to detect any secondary cord compression that may have occurred during patient evaluation and transport. Our two cases support the prior biomechanical findings.\textsuperscript{11,12} The average anteroposterior diameter of the human thoracic spinal canal is 16.2 mm.\textsuperscript{13} Animal studies suggest that 40\%–60\% canal occlusion caused impaired neuronal conduction at compression velocities below 3 m/s, and that no recovery of somatosensory-evoked potentials occurred following 75\% canal occlusion.\textsuperscript{14,15} Admission imaging of our cases demonstrated 7 mm retropulsion of a T5 bony fragment in patient 1 indicating 43\% canal occlusion and mild retropulsion of the posterior T4 cortical margin in patient 2. Although the peak velocity of transient cord compression during trauma was unknown in our cases, their clinical outcomes suggest that substantially greater cord compression occurred during trauma as compared to that documented from admission imaging.

The mechanism of injury in our cases consisted of high energy flexion-compression of the mid-thoracic spine with the spine in a flexed posture at the time of impact. The injuries were caused by abrupt pocketing and deceleration of the head, neck and shoulders upon impact with the ground combined with continued forward and downward momentum of the torso and lower extremities. A similar mechanism is well known to cause compression fractures of the cervical spine due to head-first falls, spear tackling in football or diving head-first into shallow water.\textsuperscript{16–18} The injury mechanism is less common and less understood for causation of mid-thoracic spine fracture/dislocation. Availability of video footage of races in which the accidents occurred combined with examination of the patients’ diagnostic images in our cases were valuable in deducing the injury mechanism. Stop-frame analyses of the videos led to identification of mid-thoracic spine buckling with the jockey in inverted posture. In this inverted posture, the head, neck and shoulders impacted the ground, while the thoracic spine was preflexed and legs were bent forward.

We reported two cases of jockeys who sustained fracture/dislocation of the mid-thoracic spine due to falls while horse racing. The injury mechanism consisted of high energy flexion-compression of the preflexed thoracic spine. Studies which describe mechanisms of mid-thoracic spine fracture/dislocation during sports accidents may help guide and inform future biomechanical investigations for creating realistic injuries. These data may ultimately lead to: (1) increased awareness of the injury among athletes, coaches, trainers, referees, and judges; (2) improved training techniques; (3) rule modifications; and (4) design of safer protective equipment and athletic facilities. The data may also provide guidance to clinicians when: performing patient maneuvers during transport, performing reduction and decompression, and choosing the optimal fixation technique.

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Ethical statement

Patient health information has been secured. All procedures involving human patients were in accordance with ethical standards of the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Declaration of competing interest

The author consults on personal injury cases related to the topic of this research.
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