Exceptional Neurologic Recovery in a Teenage Football Player After Second Impact Syndrome With a Thin Subdural Hematoma

Michelle A. Potts, MD, Eric W. Stewart, MD, Michael J. Griesser, MD, Joshua D. Harris, MD, Carl D. Gelfius, MD, Karl Klamar, MD

INTRODUCTION

Second impact syndrome (SIS) occurs when a person sustains a second brain injury before the symptoms of an initial brain injury resolve [1,2]. Severe brain edema develops due to cerebral vasculature dysautoregulation and causes significant neurologic deficits [1-3]. SIS is a rare condition that occurs primarily in adolescents and young adults. Severe neurologic deficits and death typically occur with SIS [1,2,4]. This report describes an exceptional neurologic recovery in a teenage football player after SIS with a thin subdural hematoma (SDH). Institutional review board approval was obtained for this case study.

CASE PRESENTATION

A 13-year-old previously healthy male football player sustained a concussion during a football game. He reported that he was tackled with significant force. He had no loss of consciousness but did state that he had a headache after the game and felt quite fatigued over the next 2-3 days, sleeping most of the time. He did not inform his coach or athletic trainer of his symptoms, and he continued to participate in football practices and games. Approximately 3 weeks after the initial injury, the patient was playing the corner back position and tackled another player head on. There was no loss of consciousness. He reported that it was not a hard hit but that he did not feel "quite right." He stayed in the game for another play but continued to not feel well and walked to the sidelines, where he collapsed. He became unresponsive, with a Glasgow Coma Scale score of 3, and was intubated and treated with midazolam at the scene; he then was transported to a tertiary pediatric hospital. He was noted to have posturing en route to the hospital, but no seizures. While in the emergency department, his pupils were unequal, left 6 mm and right 2 mm, and nonreactive. In the emergency department, he was given mannitol, midazolam, fentanyl, and fosphenytoin. Mild hyperventilation was also performed. With these interventions, his left pupil changed from 6 mm to 2 mm. Computed tomography of the head (Figure 1) revealed a left frontoparietotemporal heterogenous SDH, 6 mm in width, with associated effacement of the left lateral ventricle and shift of the midline structures toward the right. The basal and cerebral sulci were not well seen, which is evidence of extreme swelling. There was no evidence of uncal or diencephalic herniation. There was no evidence of intra-axial hemorrhage. The gray–white matter differentiation was preserved. Hemispheric asymmetry was noted. Multifocal posttraumatic ischemic infarction was not noted. There was a hypodensity within the left frontoparietal parenchyma consistent with edema. He was emergently taken, within hours of the injury, to the operating room for a left craniectomy and clot evacuation.

His residual deficits after decompression included mild right hemiparesis, with the upper limb more involved than the lower limb. He was able to ambulate with physical therapy 500 feet with contact guard assistance (CGA) but had several episodes of loss of balance. He was able to go up and down steps with CGA but became very fatigued. He needed CGA to minimal assistance with activities of daily living such as bathing and dressing. On testing with speech therapy, he demonstrated deficits with immediate memory, short-term memory, problem solving, and receptive language. Once medically stable after his surgical

M.A.P. Division of Physical Medicine and Rehabilitation, Nationwide Children's Hospital, 700 Children's Dr, Columbus, Oh 43205. Address correspondence to: M.A.P.; e-mail: Michelle.Potts@nationwidechildrens.org
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E.W.S. Department of Physical Medicine and Rehabilitation, Ohio State University Medical Center, Columbus, OH

M.J.G. Department of Orthopaedic Surgery, Ohio State University Medical Center, Columbus, OH
Disclosure: nothing to disclose

J.D.H. Department of Orthopaedic Surgery, Ohio State University Medical Center, Columbus, OH
Disclosure: nothing to disclose

C.D.G. Division of Physical Medicine and Rehabilitation, Nationwide Children's Hospital, Columbus, OH; Department of Physical Medicine and Rehabilitation, Ohio State University Medical Center, Columbus, OH
Disclosure: nothing to disclose

K.K. Division of Physical Medicine and Rehabilitation, Nationwide Children's Hospital, Columbus, OH; Department of Physical Medicine and Rehabilitation, Ohio State University Medical Center, Columbus, OH
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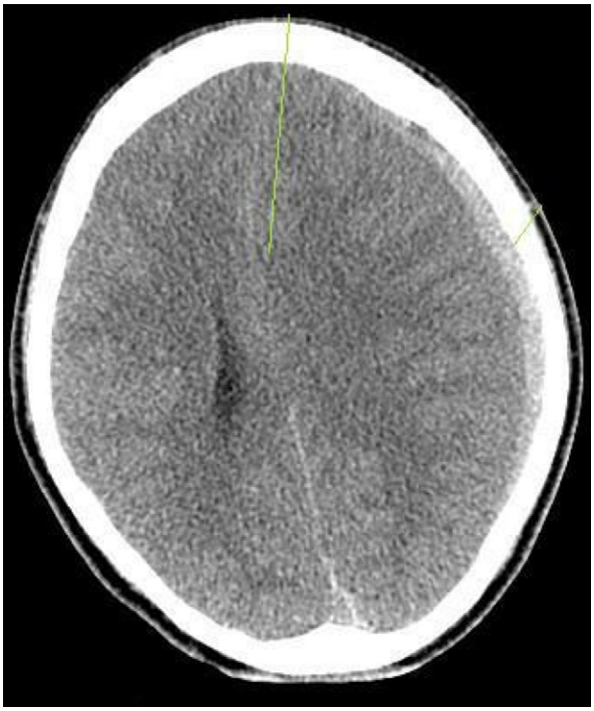

Figure 1. Computed tomogram of the head obtained after second injury, showing a large midline shift and subdural hematoma formation.

procedure, he was admitted to acute inpatient rehabilitation at a tertiary care pediatric hospital.

Before rehabilitation admission, on hospital day 6, the Wee Functional Independence Measure (Wee-FIM) revealed deficits in self-care, sphincter control, mobility transfers, locomotion, communication, and social cognition that improved to supervision or modified independent level before discharge (Table 1). Expected Wee-FIM scores for a healthy, typically developing, 13-year-old boy would be 7 for each domain, with a total of 126. Neuropsychological testing, performed on hospital day 18, revealed deficits with executive functioning, fine motor speed, and dexterity.

Eight weeks after discharge from acute inpatient rehabilitation, he was seen in the physical medicine and rehabilitation outpatient clinic. He was reassessed by a physiatrist and a physical therapist who reported that his strength was 5/5 throughout. He was ambulating community distances independently without an assistive device. Balance, range of motion, and endurance were documented as normal. He was reassessed by an occupational therapist who reported that his handwriting was functional and that he was independent with all activities of daily living, including dressing, bathing, and feeding himself. He was seen by a speech therapist who reported that he had improved processing speed and organizational skills and that his problem-solving skills were age appropriate. He was also seen by a neuropsychologist who did not repeat testing but, on interviewing him, thought that he was functioning with very minimal limitations. The neuropsychologist believed that the patient would benefit from extra support from the school, such as an Individualized Education Plan under the traumatic brain injury designation or a 504 plan due to his neuropsychologic testing, which revealed deficits with executive functioning, fine motor speed, and dexterity. She encouraged the patient and his parents to share a copy of his inpatient neuropsychology test results with the school. He had returned to school and, per patient and parental reports, he was at his baseline academic level of functioning. He had not returned to any contact sporting activities at follow-up per physiatric recommendations.

DISCUSSION

Given the significant neurologic decline after mild impact and the radiologic findings of a thin SDH with a large midline shift, this patient's injury is consistent with SIS. SIS was initially described in 1973 by Schneider [5], and the term "second impact syndrome" was coined by Saunders and Harbaugh, as noted by Moori et al [4] in 1984. Since its description, there have been multiple case studies that characterize individuals after SIS [1,2,4]. Classic SIS develops from significant brain edema without intracranial bleeding, but there are 18 case reports that describe SIS with thin SDH.

Table 1. *Wee functional independence measure (FIM) scores: pediatric FIM**

	Hospital Admission, Day 6	Hospital Discharge, Day 20
Eating	7	7
Grooming	4	5
Bathing	1	5
Upper-body dressing	4	5
Lower-body dressing	3	5
Toileting	1	6
Bowel	1	6
Bladder	5	6
Chair mobility	4	5
Toilet mobility	4	5
Tub mobility	4	4
Walking	4	5
Wheelchair mobility	1	1
Stairs	4	5
Comprehension	5	6
Expression	5	6
Social interaction	5	6
Problem solving	4	5
Memory	3	4
Total	69	97

*Wee-FIM score description: 7, independent; 6, modified independent; 5, supervision; 4, minimal assistance; 3, moderate assistance; 2, maximal assistance; 1, dependent. For ages 83 mo and more, the mean total score is 119.74, the median (SD) total score is 122 ± 5.40.

It is thought that the thin SDH does not contribute to the midline shift or herniation but that the massive brain edema is responsible for these changes. All the patients in the literature had sustained severe neurologic injury or had died [1,2,4].

This case is unique in that it represents one of the best neurologic outcomes after SIS [1,2,4]. Presently, there are no imaging or laboratory studies to confirm that the brain has completely healed from a concussion. Return to play after concussion is currently based on resolution of symptoms and neuropsychological testing if available. Most return to play recommendations are based upon the Consensus Statement on Concussion in Sport: the Third International Conference on Concussion in Sport held in Zurich in 2008, which states that the recommendations could be applied to adolescents and children down to the age of 10 years; there are no guidelines specific to the pediatric population under 10 years of age [6]. Once a patient has sustained a concussion, physical and cognitive rest is recommended until the patient is symptom free. The individual is returned to activity via a stepwise return to a participation program where the intensity of physical activity is gradually increased over several days as long as the patient remains symptom free [6]. After a concussion, most children recover completely within a few days to weeks [7]; however, it has been shown that children do require a longer time period than adults to recover from postconcussive symptoms [8]. Heightened awareness of the seriousness of a concussion and of the importance of preventing a second concussion while the brain is healing is paramount in preventing SIS. With the rarity of reported SIS cases, and with all having devastating results, this case represents the best neurologic recovery reported to date. It is imperative to collect cases such as ours in an effort to establish brain injury guidelines. In addition, this case should reinforce to physicians, parents, and athletes that mild symptoms after a concussion should not be ignored.

REFERENCES

1. Cantu RC. Second-impact syndrome. Clin Sports Med 1998;17:37-44.
2. Cantu RC, Gean AD. Second-impact syndrome and a small subdural hematoma: An uncommon catastrophic result of repetitive head injury with a characteristic imaging appearance. J Neurotrauma 2010; 27:1557-64.
3. Wetjen NM, Pichelmann MA, Atkinson JL. Second impact syndrome: Concussion and second injury brain complications. J Am Coll Surg 2010;211:553-7.
4. Mori T, Katayama Y, Kawamata T. Acute hemispheric swelling associated with thin subdural hematomas: Pathophysiology of repetitive head injury in sports. Acta Neurochir Suppl 2006;96:40-3.
5. Schneider RC. Head and Neck Injuries in Football. Mechanisms, Treatment, and Prevention. Baltimore, MD: Williams & Wilkins; 1973.
6. McCrory P, Meeuwisse W, Johnston K, et al. Consensus Statement on Concussion in Sport: The 3rd International Conference on Concussion in Sport held in Zurich, November 2008. Br J Sports Med 2009;43(Suppl 1):i76-90.
7. Kirkwood MW, Yeates KO, Wilson PE. Pediatric sport-related concussion: A review of the clinical management of an oft-neglected population. Pediatrics 2006;117:1359-71.
8. Field M, Collins MW, Lovell MR, Maroon J. Does age play a role in recovery from sports-related concussion? A comparison of high school and collegiate athletes. J Pediatr 2003;142:546-53.