Chapter 11
Sport-Related Structural Brain Injury

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Introduction

Sport-related structural brain injury (SRSBI) is defined as intracranial trauma seen on neuroimaging incurred by an athlete during sport or other life activities. Though sport-related concussion (SRC) receives much of the attention when discussing the topic of head injuries in sports and are significantly more common, SRSBI has potential to cause permanent neurologic deficits and/or death and may require emergent brain or spine surgery. Distinguished from SRC by the presence of positive imaging findings of intracranial trauma, SRSBI includes subarachnoid hemorrhage (SAH), epidural hemorrhage (EDH), subdural hemorrhage (SDH), intraparenchymal hemorrhage (IPH), diffuse cerebral edema (DCE), and diffuse axonal injury (DAI) [50]. Cases of SRSBI are medical emergencies, require hospital admission, often warrant intensive care unit (ICU) monitoring, with or without surgery.

Since SRSBI represent a more severe form of SRC, a general understanding of the epidemiology of SRC is worthy of discussion. SRC comprises up to 9% of all athletic injuries in the United States (U.S.), with approximately 3.8 million emergency department (ED) visits documented between 2001 and 2018 for sports and recreation-related traumatic brain injuries (TBI) [23, 46]. Following more than a

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decade of increasing rates of in-game SRC, rates have plateaued from 2014 to 2017, specifically a decrease in recurrent and practice SRC rates [27, 46]. Furthermore, in a study of 13,000 U.S. high school students, being male, white, in a higher grade, and participating in competitive sports were associated with a higher lifetime prevalence of reporting a diagnosed concussion, and participating in contact sports was associated with a higher odds of being diagnosed with more than one concussion [45]. Although much attention has been given to SRC stemming from collision sports such as American football (football), ice hockey, and lacrosse, lower contact sports, including international football (soccer), basketball, and cheerleading, have also been implicated [32, 34].

Given their low prevalence, a lack of information exists regarding pathophysiology, epidemiology, and outcomes following SRSBI [28]. Similarly, consensus regarding SRSBI management and return-to-play (RTP) is lacking, leaving neurosurgeons with little guidance to manage these complex situations. This chapter highlights the pathophysiology, epidemiology, treatment, and long-term outcomes in athletes suffering SRSBI.

Definition/Pathology

SRSBI is defined as intracranial trauma seen on neuroimaging incurred by an athlete during sport or other life activities. Athletes diagnosed with a concussion during play with subsequent neuroimaging findings positive for SDH, EDH, SAH, IPH, DAI, or DCE are considered to have suffered an SRSBI (Figs. 11.1, 11.2, and 11.3). Fractures alone without parenchymal damage are not typically considered SRSBI, as long as the brain is uninvolved [50]. The pathology of SRSBI is heterogeneous, and ranges from the well-described SDH to the relatively poorly understood and controversial Second Impact Syndrome (SIS) [1]. Common mechanisms of injury are similar to SRC, including head-to-head, head-to-opponent, and head-to-ground mechanisms [39, 49]. Though most SRSBI occur during sport, injuries that occur during other activities but involve an RTP decision are also grouped under SRSBI due to the ensuing discussion between neurosurgeon and athlete/family.

Subdural Hemorrhage

SDH is defined as intracranial bleeding between the dura and arachnoid layers of meninges. It is the most-reported form of SRSBI, as well as the most common cause of sport-related fatality [10]. The pathophysiology of subdural hemorrhage involves rupture of bridging veins traversing the subdural space, leading to intracranial hemorrhage not confined between suture lines, and potentially rapid increases in

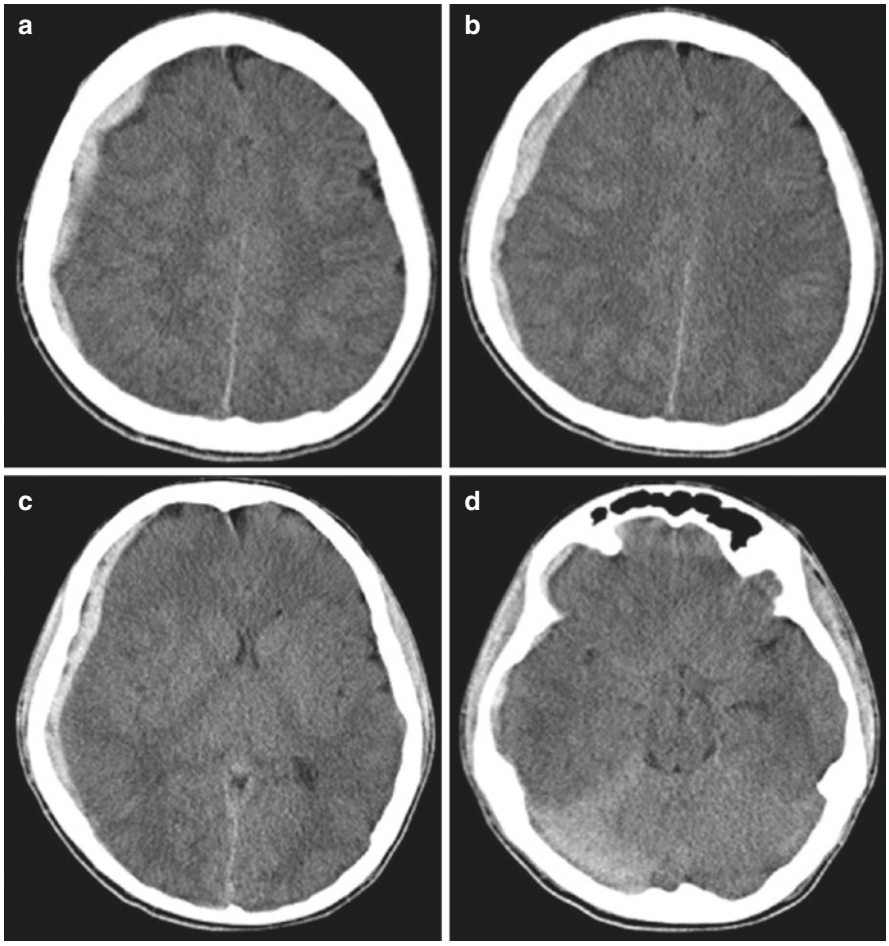

Fig. 11.1 Non-contrast CT of head for SDH SRSBI. (**a**, **b**) 9-mm acute right convexity subdural hemorrhage with associated effacement of ipsilateral sulci and midline shift; (**c**) 8-mm of midline shift related to hemorrhage with significant effacement of lateral ventricles; (**d**) extension of subdural towards floor of anterior and middle fossa with further layering along right tentorium, early right uncal herniation with effacement of crural cisterns. (With permissions from Yengo-Kahn et al. [49])

intracranial pressure leading to subsequent neurological deficit or death. SDH can be subcategorized by the period of time since bleeding as: minutes to hours being acute SDH (aSDH), or days to weeks being chronic SDH (cSDH). Radiographically, aSDH appears as an extra-axial crescent-shaped hyperdensity on non-contrast head computed tomography (CT), most common along the convexities, tentorium, or falx [44]. In contrast, cSDH typically presents as a hypodense extra-axial collection

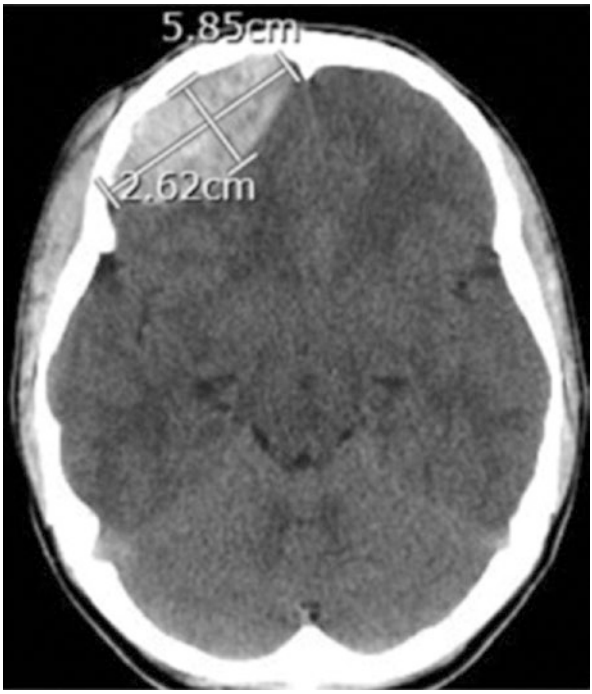

Fig. 11.2 Non-contrast CT of head for EDH SRSBI. Scan shows a 2.6-cm right, frontal epidural hematoma after heading resulting in a head-to-head collision in a soccer game. (With permissions from Mummareddy et al. [39])

along the cerebral convexity [47]. Both aSDH and cSDH presentations have been observed related to sports, ranging from collision-heavy sports such as football to low-impact sports such as race walking [10, 11]. Although the majority of SDH in SRSBI are aSDH, requiring immediate hospitalization and potential neurosurgical intervention, cSDH should not be ignored as a potential etiology as a cause of persistent and progressive symptoms in athletes following head trauma.

Of note, there is early evidence that cSDH potentially occurs more frequently in athletes with pre-existing arachnoid cysts, congenital cerebrospinal fluid collections enveloped in an arachnoid membrane [38, 51]. In athletes with existing arachnoid cyst who suffer acute or chronic SDH, evacuation of the hemorrhage is performed in conjunction with cyst fenestration [51]. Presently, there is no absolute contraindication for patients with an arachnoid cyst to participate in sports, given the unlikelihood of a hemorrhage occurring. Furthermore, no data exists to establish the true incidence and risk of SRSBI with an arachnoid cyst. Though reports of cSDH and arachnoid cysts exist, aSDH can certainly occur in the setting of arachnoid cysts, but is easily missed in the setting of an emergent craniotomy [51]. Furthermore, publication bias is likely at play, since a cSDH in a young athlete is more academically interesting than a traumatic aSDH. Overall, multi-institution studies are needed to establish prevalence of these rare injuries in the setting of arachnoid cysts.

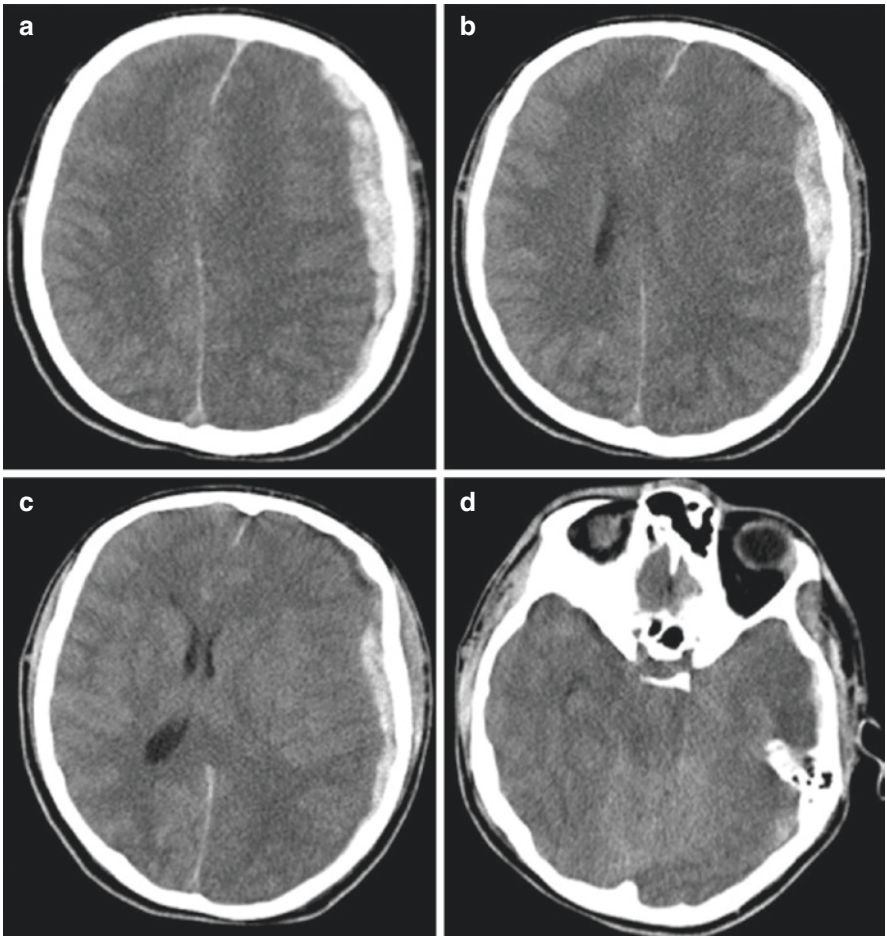

Fig. 11.3 Non-contrast CT of head for SDH SRSBI with DCE. (**a**) 12-mm acute left convexity subdural hemorrhage with effacement of bilateral sulci and clear midline shift; (**b**) acute hemorrhage again demonstrated with near total effacement of left lateral ventricle; (**c**) midline shift measured at 10-mm; (**d**) complete effacement of basal cisterns with uncal herniation. (With permissions from Yengo-Kahn et al. [49])

Subarachnoid Hemorrhage

SAH is defined as bleeding into the subarachnoid space between the arachnoid and pia layers enveloping the brain [50]. It can be further subcategorized by etiology and origin of bleeding. Traumatic SAH, as seen in SRSBI, is due to momentary brain oscillation secondary to rotational or linear acceleration of the head, and subsequent shearing of small cortical vessels [17]. CT imaging reveals isolated hyperdense foci along the cerebral sulci, and traumatic SAH can present concurrently with diffuse axonal injury, SDH, or other structural injuries [18]. Outside traumatic SAH,

athletes rarely present with large-vessel damage or aneurysm rupture secondary to a traumatic event. Vertebral artery injury can be precipitated by trauma to the skull base or neck, caused by impact with a hockey puck, collision secondary to tackle in rugby, and direct blow impact from an opponent's foot in martial arts and kickboxing [12, 33, 37, 42]. These patients can present with symptoms such as headache, neck pain, or Horner's syndrome if the sympathetic plexus along the carotid artery is disrupted. Aneurysm rupture has been reported in a variety of sport settings, including soccer, hockey, and weightlifting, with several cases reporting fatalities [3, 24, 43]. In these cases, a pre-existing aneurysm and/or vascular malformation becomes disturbed by an external trauma or hemodynamic force, leading to aneurysmal SAH. Aneurysmal or AVM-related SAH are complex injuries and require primary treatment of the vascular abnormality by an open or endovascular approach. Like non-sport-related trauma, detection of an underlying aneurysm often portends a poor prognosis, compared to isolated traumatic SAH from a cortical vessel rupture alone [50]. As the literature for sport-related SAH is limited to case reports and case series, trends and outcomes must be extrapolated from the general neurosurgical literature rather than sport-specific data [26, 40, 42]. Overall, as long as no underlying lesion is found, acute management of sport-related, traumatic SAH is similar to non-sport SAH. However, the RTP discussion is more nuanced and requires resolution of any previous hemorrhage, along with an in-depth discussion of the risks/benefits of continued sport participation.

Epidural Hemorrhage

EDH is defined as bleeding between the skull and the dura mater. Most often, epidural hemorrhages occur due to high-impact trauma and are associated with skull fractures in the area of the middle meningeal arteries, although the venous sinuses may be implicated occasionally. EDH classically presents with acute loss of consciousness, followed by a lucid phase and subsequent rapid decompensation. EDH is classically identified as a biconvex, lens-shaped hyperdensity limited by cranial suture lines on head CT. It is considered a neurosurgical emergency. EDHs have been described in a variety of sport settings, including impact injuries in basketball and simultaneous head-to-head and ball-to-head contact during contested headers in soccer [14, 39]. Management is again similar to non-sport-related EDH, and often requires emergent craniotomy and evacuation.

Intraparenchymal Hemorrhage

IPH is defined as bleeding within the brain parenchyma. The spectrum of etiologies causing IPH is vast, ranging from trauma (brain contusions) to hypertension to tumors or vascular malformations. Within sport, IPHs have been documented most

often when occurring concurrently with other forms of intracranial bleeding, such as traumatic SAH and SDH in soccer players, boxers, and ice hockey players, among other sports [16, 20–22]. Furthermore, traumatic IPH frequently occurs at bone-brain interfaces in the form of contusions. Small, deep-seated IPH or punctate IPH within the corpus callosum, hippocampi or the grey-white junction frequently reflects DAI rather than true contusions, and these injuries are rarely related to sports. Most important in any sport-related IPH is ruling out a primary vascular lesion through CTA and/or MRI.

Diffuse Cerebral Edema and Second-Impact Syndrome

Diffuse cerebral edema (DCE) is a commonly fatal condition that results from a failure of cerebral hemodynamic autoregulation [50]. DCE in the context of SRSBI can occur after any high-energy TBI. The proposed mechanism involves a rapid loss of cerebral autoregulation with a concurrent rise in catecholamines, effectively leading to an inability of the cerebral vasculature to accommodate the rapid rises in blood pressure [50]. However, some patients with DCE seem to fit a clinical entity known as Second Impact Syndrome (SIS). SIS occurs when an athlete who has suffered a recent SRC is returned to play prematurely, and sustains a second blow to the head or body. The second, inciting impact may be low energy and not head-related, including blows to the torso, and occurs while the athlete is still recovering from the first SRC. After the second impact, the brain loses its ability to autoregulate, DCE ensues, and the athlete loses consciousness quickly [50]. DCE can present in isolation as part of SIS, with no intracranial hemorrhages or mass effect observed, or concurrently with other structural brain injury, such as SDH, often worsening the first injury [41]. It must be noted that SIS is a controversial topic, with continued debate over its existence [1]. Some report its existence only in young, football athletes, while other researchers refute its existence at all [7, 35]. Regardless, it is critical for providers to both prophylactically caution against premature RTP and recognize the signs of an athlete presenting with DCE following an initial insult.

Epidemiology

With the little data that has been published, the overall incidence of SRSBI is low; the National Center for Catastrophic Sport Injury (NCCSI) noted that SRSBI was present in 14.7% (n = 11) of all reported sport-related catastrophic injuries [8]. Cantu and Mueller recorded 497 catastrophic brain injuries between 1945 and 1999; death was brain-related in 49% of these cases, with 16% related to cervical spine injuries, and 15% undefined [10].

Although the majority of attention in the discussion of SRSBI revolves around collision and high-impact sports, SRSBI is commonly observed in sports that are

not considered high-impact. The NCCSI defines catastrophic injuries as fatalities, non-fatal (permanent severe-functional disability), and serious (no permanent functional disability but severe injury) [8]. Though the NCCSI data include more than just SRSBI, such as sudden cardiac death, the statistics are nonetheless relevant to a discussion of SRSBI. According to the NCCSI's report on high school sports, gymnastics had the highest rates of fatal (0.84) injuries per 100,000 participants, while cheerleading had the highest rate of both non-fatal (1.71 in males, 0.98 in females) and serious (1.71 in males, 1.80 in females) injuries per 100,000 participants. Of note, football remained a significant cause for fatal (0.38), non-fatal (1.14) and serious (1.12) catastrophic injuries in male high school athletes. At the collegiate level, skiing had the highest rate of direct fatal injuries per 100,000 participants in both males and females (4.62 and 5.59 per 100,000, respectively). Both gymnastics (5.37 in males, 3.72 in females) and hockey (3.56 in males) had a higher incidence of non-fatal injury than football (2.28) per 100,000 participants [9]. Again, though we cannot tease out which of these are specifically due to SRSBI, one can imagine that many are due to neurologic causes.

Collision sports such as football and rugby have also been implicated in a wide variety of SRSBI, ranging from intracranial bleeds to DCE. While football results in the most cases of SRSBI, several cases of SRSBI have also been reported in high-impact sports such as soccer and basketball. Specifically, headers in soccer have been implicated in SRSBI, typically resulting from head-to-head collision or head-to-elbow/body collisions, rather than the actual head-to-ball contact. Finally, individual combat sports such as martial arts and kickboxing have also reported cases of SRSBI [11, 12].

Diagnosis and Management

Many healthcare providers, including certified athletic trainers (ATs), primary care and emergency medicine providers, neuroradiologists, neuropsychologists, and neurosurgeons, are involved in the diagnosis and management of SRSBI. While SRC is a clinical diagnosis, involving somatic, cognitive, and behavioral symptoms, along with physical signs, sleep/wake disturbance, and balance impairment, the gold standard for SRSBI diagnosis remains head imaging. The challenge may be the decision of *when* to image. Though obvious, certain "red flag signs/symptoms" require emergent transfer to the ER and immediate imaging. These include somnolence, unequal pupils, progressive headache, unremitting nausea/vomiting, prolonged or delayed loss of consciousness, focal neurologic deficit (one-sided facial droop, weakness, numbness), significantly altered mental status, progressive alteration in behavior or mental status, and delayed seizure. Providers potentially caring for athletes with head injuries should be familiar with and be able to recognize these signs and symptoms. Specific additional education in this area may be necessary. Also, having a neurosurgeon accessible to covering certified athletic trainers (ATs)

for urgent consultations can facilitate rapid triage and transport to a hospital setting with appropriate level of care. Furthermore, decision support tools such as validated prediction rules identifying clinically important TBI with imaging exist to support clinical decision-making [29].

Just as the case with SRC, once a diagnosis of SRSBI is suspected, an athlete must be immediately removed from play. Whereas an athlete with SRC can be monitored on the sideline, suspicion for an SRSBI requires immediate transfer to a hospital for head CT. If any of the "red flag signs/symptoms" are seen, calling emergency service dispatch (i.e., 9-1-1 in the USA) is most appropriate. Acute management hinges on prompt diagnosis, as the difference between fatal and positive outcomes may depend on early surgical intervention [49]. In evaluating an athlete for potential SRSBI in the emergent setting, it is critical to remain true to the tenets of acute trauma care: airway, breathing, and circulation. Positive neuroimaging of SRSBI should be further triaged as conservative management with close monitoring or immediate operative intervention, depending on the status of the patient.

Presently, there are no sport-specific guidelines or indications for surgical intervention in SRSBI, but we can rely on the general neurosurgical trauma literature regarding indications to operate. In general, indications for surgical intervention include size and volume criteria, as well as the presence of midline shift, appearance of cisterns, and other markers of mass effect [19]. Specifically, these criteria differ for the underlying etiology of TBI, with specific guidelines existing for aSDH, SAH, EDH, and IPH [4–6, 31]. Other indications for surgical intervention include rapid expansion of mass lesions within the first hours or days following admission, presenting as new or worsening neurologic deficit, and/or new or worsening radiographic findings [19]. In accordance with the Brain Trauma Foundation surgical management guidelines, an operation is indicated for all symptomatic patients with a space-occupying lesion [30]. The decision to operate involves many clinical factors, and operations may be performed immediately (e.g., for large EDH or aSDH) or only when intracranial pressures are refractory to medical and intensive care therapies (e.g., for DCE or IPH) [13].

Overall outcomes for surgical intervention are mixed. Little debate exists about evacuation of a space-occupying lesion; however, much more controversial is DCE to relieve refractory intracranial hypertension. The DECRA trial in 2011 found that, in general, trauma patients with severe diffuse traumatic brain injury and intracranial hypertension refractory to first-tier therapies, decompressive craniectomy decreased intracranial pressure, duration of mechanical ventilation, and time in the ICU when compared with standard care [13]. However, patients undergoing decompressive craniectomy were found to have significantly lower median Extended Glasgow Outcome Scale scores (i.e., worse functional outcomes) and an overall higher risk of an unfavorable outcome, despite a relatively low rate of complications [13]. The RESCUE-ICP trial in 2016 illustrated similar results; while decompressive craniectomy in patients with TBIs and refractory intracranial hypertension was associated with lower mortality rate when compared to standard therapy, higher

rates of vegetative state and severe disability were seen [25]. In SRSBI, surgical intervention depends heavily on etiology of SRSBI and ranges from invasive monitoring to hemicraniectomy and is associated with a wide range of clinical outcomes [49].

Outcomes

The spectrum for SRSBI outcomes ranges from full recovery to death [39, 48, 49]. Outcomes vary depending on age and sport, with variability seen within each defined SRSBI pathology. Due to a lack of reliable incidence, prevalence, and outcomes data, meaningful evidence-based outcomes are not able to be provided. Some SRSBI patients are only mildly symptomatic upon presentation, and the most common presenting symptoms are headache and nausea in the setting of small SDH and/or SAH [50]. Temporary and potentially permanent neurologic deficits, along with death, are commonly seen in SRSBI.

Several published case studies highlight the broad spectrum of outcomes observed in SRSBI. Mummareddy et al. presented a case of a 16-year-old boy who suffered an EDH following a head-to-ball and head-to-head collision during a high school soccer game (Fig. 11.2) [39]. Although the patient denied loss of consciousness and complained only of a minor headache, he experienced blurred vision, along with worsening headache and nausea/vomiting. Radiographic imaging upon presentation revealed a 2.6-cm right frontal EDH, for which an emergent evacuation was performed. The patient's postoperative course was unremarkable, and at both 2-week and 3-month follow-up visits, he expressed no complaints or residual effects, eventually being cleared for return to full sporting activity.

Yengo-Kahn et al. highlighted the spectrum of SRSBI in a small case series of high school football players presenting with aSDH following in-game collisions [49]. Of note, two out of three patients had suffered a known previous SRC within 4 weeks of the catastrophic event. All three reported helmet-to-helmet trauma and subsequent loss of consciousness. Two of three patients required immediate surgical decompression, while the other patient was treated conservatively with an uncomplicated hospital course. The patient undergoing conservative management began home-schooling 5 months post-injury and continued to struggle with headaches and focal neurologic deficits at 6 months. Of the patients undergoing surgical intervention, one continued to have difficulty with exertional headaches, memory, and spatial navigation. Unfortunately, the final patient remained nonverbal and quadriplegic, with nutritional requirements met through a percutaneous endoscopic gastrostomy tube (Fig. 11.3)**.** None of the patients in the case series returned to sport.

In their larger-scale case series, Boden et al. described 94 cases of SRSBI reported to the NCCSI, with the following distribution of outcomes: death (9%), permanent neurologic injury (51%), serious injury with full recovery (40%) [2]. Of note, SDH was the most common injury in the study, with 75 isolated SDH and 10 SDH with diffuse brain edema. Of note, 39% of athletes who experienced a

catastrophic brain injury were playing with residual neurologic symptoms from a prior head injury. Most players received impact to the head via either tackling or being tackled.

Return to Play

While RTP in SRC patients is well-defined with the near universal use of a graduated activity paradigm, RTP decision-making is heterogeneous in the SRSBI population, with no official guidelines [36]. Often, neurosurgeons are left to make these challenging decisions, assuming significant liability. In general, RTP depends on a multitude of factors, including but not limited to age, symptomatology, initial injury etiology, treatment, and the type of sport the athlete is returning to. Prior studies have advised against RTP within the first year in athletes following craniotomy, so as to allow for complete bony fusion of the craniotomy site [15]. However, these studies are limited in scope and may not address adequately the additional nuances involving RTP with SRSBI. Returning to collision sports following craniotomy with resolution of the hemorrhage and symptoms is particularly controversial, with experts ranging in opinion from RTP at 3 months to no RTP advised. In our anecdotal experience, level of play is also a crucial factor to consider. In professional athletes with income-related factors and careers at stake, a more aggressive approach to RTP may be considered. Conversely, a younger athlete with little aspirations to play beyond high school, not returning to sport, or choosing a low-contact sport is more appropriate.

In a recent systematic review and expert opinion, several insights were offered on RTP considerations. In the acute setting and management of SRSBI in asymptomatic patients, operative indications are similar to non-athletes, including large hemorrhage size >10 mm, midline shift >5 mm, or increasing size on serial imaging. Anecdotally, factors that we discuss and consider important when deciding RTP are the following. There should be complete resolution of hemorrhage on standard imaging. All symptoms must resolve, with no neurologic deficit, as patients with persistent, severe motor deficits secondary to SRSBI should not be participating in high-level, contact sports. Furthermore, RTP is advised against in high-contact or collision sports following SRSBI with persistent hemorrhage on imaging; however, return to low-contact sport may be indicated. These patients are recommended to switch to a low-contact sport. Finally, RTP may be considered earlier for higher levels of sport, where expert medical personnel are available and on-call.

Conclusions

SRSBI is a rare and often underappreciated subcategory of sport-related head injury that requires additional attention, given the potentially devastating neurologic sequelae. Although difficult to ascertain the true incidence of SRSBI, it is important

to remember that many sports are indicted in SRSBI, including both collision/high-impact and low-impact sports. Outcomes are heavily dependent on the initial presentation, as well as the availability and success of timely and appropriate treatment. Recognition and prompt diagnosis of SRSBI is critical. Finally, treatment and RTP guidelines are lacking for the SRSBI population; more investigation is warranted to understand and improve long-term outcomes in athletes suffering head trauma. Future investigations and research, as well as the establishment of treatment and RTP guidelines, serve as intriguing future directions.

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