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Current Topics in Sports-related Head Injuries: A Review

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Abstract

We review the current topic in sports-related head injuries including acute subdural hematoma (ASDH), concussion, and chronic traumatic encephalopathy (CTE). Sports-related ASDH is a leading cause of death and severe morbidity in popular contact sports like American football in the USA and judo in Japan. It is thought that rotational acceleration is most likely to produce not only cerebral concussion but also ASDH due to the rupture of a parasagittal bridging vein, depending on the severity of the rotational acceleration injury. Repeated sports head injuries increase the risk for future concussion, cerebral swelling, ASDH or CTE. To avoid fatal consequences or CTE resulting from repeated concussions, an understanding of the criteria for a safe post-concussion return to play (RTP) is essential. Once diagnosed with a concussion, the athlete must not be allowed to RTP the same day and should not resume play before the concussion symptoms have completely resolved. If brain damage has been confirmed or a subdural hematoma is present, the athlete should not be allowed to participate in any contact sports. As much remains unknown regarding the pathogenesis and pathophysiology of sports-related concussion, ASDH, and CTE, basic and clinical studies are necessary to elucidate the crucial issues in sports-related head injuries.

Key words: sports head injury, acute subdural hematoma, concussion, chronic traumatic encephalopathy

Introduction

Head injuries due to a blow to the head or a fall are especially prevalent in contact and collision sports such as boxing, judo, karate, sumo, American football, rugby, ice hockey, and soccer as well as winter sports such as skiing and snowboarding. The most common sports-related head injury is concussion; it includes mild traumatic brain injury and the most serious sequela, acute subdural hematoma (ASDH). Increasing attention—both social and medical has been paid to incidents in which repeated sports head injuries resulted in fatal brain swelling,[1–4] ASDH,[5,6] abnormal results on neuropsychological tests,[7–9] and, over the long run, chronic traumatic encephalopathy (CTE).[10–14] Many cases of severe head injury sustained during judo practice have been reported among Japanese junior and senior high school students.[15–18] Since Japanese martial arts were introduced as a compulsory subject in Japanese junior high schools in 2012, this has raised public concern about a potential increase in the number of judo-related catastrophic head injury.

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International guidelines addressing sports-related concussions were published in 2013.[19–22] The International Conference on Concussion in Sports whose members include the International Olympic Committee, le Fédération Internationale de Football, and the International Rugby Board, is held to discuss issues regarding sports-related head injuries, particularly concussions. It focuses on how to suspect and assess concussions on the field and the results of the discussions are published as a consensus statement[19,20] that has become a worldwide standard for the management of concussions. Additionally, the Japan Society of Neurotraumatology has announced interim recommendations for the development of guidelines for sports-related head injuries.[16]

In this review we focus on current topics in sports-related head injuries, particularly ASDH, concussion, CTE, and their prevention, and on return to play (RTP) criteria.

Sports-related ASDH

I. Epidemiology

Severe sports-related head injuries include ASDH, acute epidural hematoma, cerebral contusion,

traumatic cerebrovascular accidents, diffuse brain swelling, diffuse axonal injury, and skull fractures. Among them, ASDH is a leading cause of death and severe morbidity in general and in American football,[5,6,23,24] judo,[15–18] boxing,[25–27] and snowboarding,[28,29] in particular. Popular sports differ among countries. One of the most popular sports in the USA is American football, more than 1.2 million high school students participated during the 2001–2002 academic year.[5] Football is associated with the highest number of direct catastrophic injuries of any sport reported in the USA and alarming trends have been noted regarding the incidence of catastrophic brain injury.[5,6,30] Over 90% of these injuries were secondary to subdural hemorrhage. According to Forbes et al.[6] the average annual incidence from 2008 to 2012 was 2.38 times than that recorded from 1998 to 2002 (Fig. 1); approximately 90% of the injuries were ASDH. More than 95% of the football players with catastrophic head injuries were 18 years or younger. Boden et al.[5] who reviewed 94 incidents of severe football head injuries reported to the National Center for Catastrophic Sports Injury Research during 13 academic years from 1989 to 2002 documented that the incidence of catastrophic head injuries was dramatically higher at the high school than the college level. There were 0.67 injuries per 100,000 high school and 0.21 injuries per 100,000 college football players.

The Japan Sports Councils (unpublished data on their home page http://www.jpnsport.go.jp/anzen/anzen_school/bousi_kenkyu) collected data on various sports injuries that occurred in Japanese elementary, junior high, and senior high schools in the course of 14 academic years (1998–2011). Among 88 cases of catastrophic head injury, judo was the leading cause followed by rugby and boxing (Fig. 2). Elsewhere[15,18] we reported that 30 Japanese athletes with catastrophic judo head injury submitted insurance claims to the All Japan Judo Federation between 2003 and 2010. Most were for ASDH associated with avulsion of a cerebral bridging vein suffered as a consequence of a hit to the occipital head upon being thrown by various techniques. The average age of the claimants was 16.5 years. The incidence of injury revealed two peaks at different academic grade levels; one peak was in the first year of junior high school and the other in the first year of senior high school (Fig. 3). Approximately 50% of the injured students were novice players without fully developed physical power. Ukemi, a protection technique applied while being thrown, appeared insufficient.[15]

In boxers with serious head injuries, ASDH not associated with cerebral contusion was reported.[25–27] ASDH accounts for approximately 75% of moderate to severe head injuries in boxers[27]; it was also found in rugby players.[31] In winter sports, the incidence of ASDH was higher in snowboarders than skiers.[28,29] While ASDH is rare in other sports, it has been reported in soccer[32] and basketball players[33] and in women wrestlers.[34]

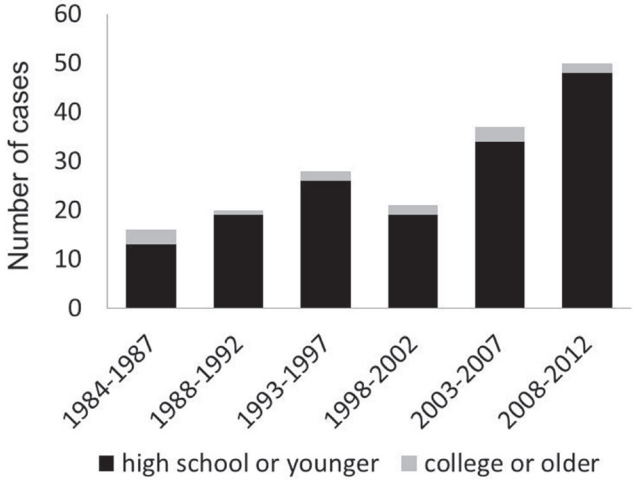

Fig. 1 Catastrophic head injuries in American football. The graph shows the annual data on catastrophic head injuries in American football players.[6,30] The incidence of catastrophic head injuries continues to rise particularly in players 18 years old or younger. The average yearly incidence from 2008 to 2012 was 2.38 times higher than in 1998 to 2002.

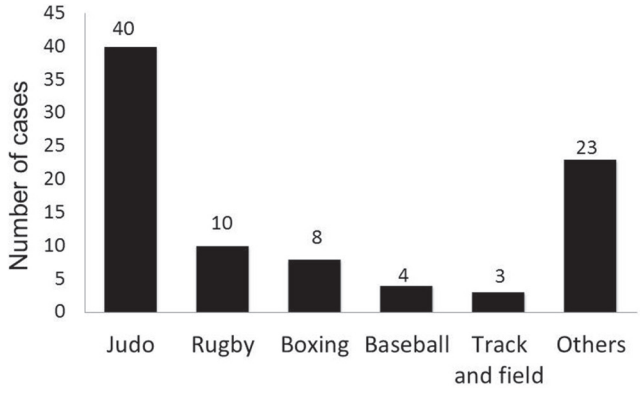

Fig. 2 Catastrophic head injuries in Japanese students reported by the Japan Sports Council. The graph shows the number of elementary-, junior high-, and senior high school students aged 7–18 years who suffered catastrophic sports-related head injuries during the period from 1998 to 2011. Judo was a leading cause of death or severe morbidity; among 88 athletes with catastrophic sports head injuries, 40 participated in judo.

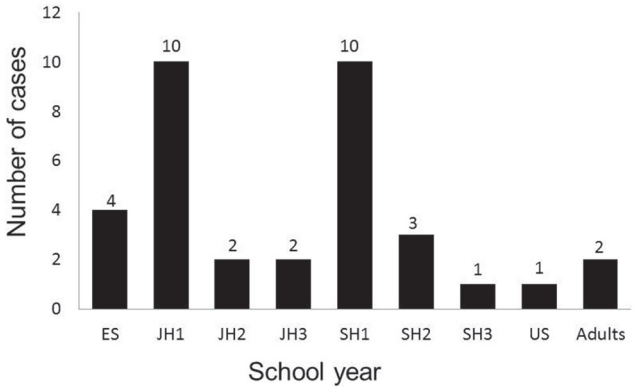

Fig. 3 Judo-related catastrophic head injuries (2003–2011). The graph shows the number of students who suffered catastrophic judo-related head injuries in different school years. There were two peaks in the incidence of head injuries; one peak is seen in the first year of junior high school and the other in the first year of senior high school. Each column represents the number of injured judo participants. ES: elementary school, grade 1 to 6, JH: junior high school, JH1 (first year) to JH3 (third year), SH: senior high school, SH1 (first year) to SH3 (third year), US: university students.

II. Pathophysiology of ASDH

Traumatic ASDH can arise from three sources, hemorrhagic contusion, rupture of a bridging vein,[35] and laceration of the cortical artery.[36] Bridging vein rupture is considered the major cause of sports-related ASDH.[6,15,31,35,37] Among catastrophic judo head injuries, most were pure subdural hematomas due to rupture of a bridging vein without major cerebral contusion.[15] The source of bleeding in boxers with ASDH was also either a bridging or a cortical vein.[26,27] Laceration of large bridging or cortical veins may exacerbate cerebral perfusion by venous congestion or infarction, resulting in brain swelling.[37] Hits directed eccentrically to the head of boxers produce rotational acceleration.[27] Angular rotation of the skull carries a higher risk of severe head injury than linear movements, theoretically by creating greater tension on bridging veins and brain tissue.[27,37] Primate experiments showed that rotational acceleration in the sagittal plane and in a forward direction tends to produce ASDH due to rupture of parasagittal bridging veins.[6,35,37–40] These veins are fragile and rupture more easily upon rotational than translational acceleration because the amplitude of relative brain-skull motion is higher in head rotation than in purely translational head motion.[35]

III. History of prior head injury

In a review of 94 catastrophic head injuries sustained between 1989 and 2002 by high school and college players of American football,[5] 35 (59.3%) of 59 athletes whose medical records were available had suffered a previous head injury; 24 (40.7%) had no known history of head injury. An analysis of 35 catastrophic head injuries suffered between 2003 and 2011 by participants in judo showed that 8 (22.9%) had a prior history of judo-related head injury with posttraumatic headache (unpublished data). In four of these eight cases, computed tomography (CT) and/or magnetic resonance imaging (MRI) detected a thin subdural hematoma when they were tested for persistent headache. They were treated conservatively and resumed their participation in judo 2 weeks to 6 months after the disappearance of the hematoma and their symptoms. Tragically, all four developed catastrophic subdural hematoma thereafter. These findings strongly suggest that a history of prior head injuries and, in particular, post-concussion headache with a thin subdural hematoma is a strong risk factor for catastrophic ASDH.

IV. Symptoms and signs

The principal initial symptoms of ASDH are loss of consciousness (60%), headache, vomiting, dizziness, and convulsive seizures.[5,15] Most patients later manifest decreased consciousness. Intervals of lucidity occurred in 83% of patients with severe judo-related head injuries although 50% deteriorated rapidly within few minutes.[15] Among 51 American football players with catastrophic head injury, 41 (80.4%) lost consciousness.[5] Other common symptoms included headache, visual disturbance, paresis, and seizures, and 8 injured players (15.7%) progressed to coma with fixed dilated pupils and decerebrate posture. Persistent headache after mild head injury in contact-sports participants appears to alert to the presence of a thin or small subdural hematoma.

V. Imaging studies

On CT scans, ASDH is seen as a high or mixed high and low density mass over the hemisphere or the interhemispheric fissure with marked midline shift and with or without hemispheric swelling. Focal cerebral contusion is not usually associated with subdural hemorrhage because most ASDH in sports-related head injuries are pure ASDH without cerebral contusion.[5,15,18] To rule out ASDH, athletes with persistent headache after mild head injury should be subjected to CT or MRI studies with thin slices or coronal views.[16] Small or thin ASDH can become large hematomas in the subacute or chronic stage with the possibility for a catastrophic outcome.[15]

VI. Treatment and outcome

Most patients with consciousness deterioration

require surgery including a variety of decompression craniotomies with, in most cases, subdural hematoma removal.[5,15] Among 94 American football players with ASDH, 8 (8.5%) died as a result of the injury, 46 (48.9%) suffered permanent neurologic deficits, and 36 (38.3%) recovered completely from their serious injuries with intracranial pressure monitoring.[5] Of 30 judo participants with ASDH,15 (50%) died and 7 (23.3%) remained in a persistent vegetative state; the outcome was poor in most patients even after decompression surgery.[15]

Concussion

I. Definition

Concussion is defined as transient impairment of brain function due to an external force to the body; it involves complex pathological conditions.[19–22] With or without transient loss of consciousness or amnesia, concussion is characterized by somatic symptoms such as headache, dizziness, nausea, and a spaced-out sensation and by various objective symptoms associated with temporary impairment of brain function including mental/cognitive dysfunction, emotional disturbance, and balance and sleep disturbance (see symptoms and assessment). Approximately 10% of individuals with concussion experience temporary loss of consciousness.[21]

II. Pathophysiology

Concussion is thought to be attributable to rotational or angular acceleration injury resulting from a shaking of the brain.[8,19–22,40,41] Depending on the severity of the rotational or angular acceleration, external injuries sustained in sports such as judo, boxing, and American football may lead to the rupture of bridging veins and the development of ASDH.

Concussion is considered a functional rather than a structural abnormality because it shows no anomalies by standard imaging modalities such as CT and MRI.[19–22] However, the possibility of microscopic axonal injuries cannot be ruled out. In animal models, application of an external force impairs homeostasis of the ionic or energy metabolism of neurons or nerve fibers, leading to the development of functional disturbances.[42–45] Details such as the threshold for functional impairment remain unclear. In animal models of concussion, the rapid destruction of nerve cell membranes induces a rapid extracellular release of potassium ions and a calcium-dependent release of various excitatory amino acids particularly glutamine, leading to neural excitation followed by inhibition.[45] Within 24 hours post-injury, the supply-demand balance of the energy metabolism is disrupted and changes in the brain blood flow and brain glucose metabolism result in axonal involvement or neurological dysfunction.[8,43] Such abnormalities in the biochemical metabolism lasted for about 10 days.[45,46]

Therefore, in the state of concussion, the neurological functions and the response speed of the brain are diminished. Young brains are thought to be more susceptible to concussions and repeated injuries lead to prolonged recovery times.[41,43,45–47] Slow recovery of the brain blood flow or glucose metabolism, changes in neurotransmitters, or axonal injury and repeated injuries may result in subsequent delayed neuronal death.[42–45] As the same phenomena may occur in humans, complete rest and abstinence from any sports activities are mandatory after a concussion.

III. Epidemiology and risk factors

The actual incidence of sports-related concussion varies depending on the reporting individuals or organizations, the period covered by the investigation, and the type of sport. Nonetheless, it is thought to be on the rise. In the USA, an estimated 1.6 to 3.8 million individuals overall suffer concussions each year;[13,21,22] 5–9% occur in participants of various sports activities. The incidence of concussion is particularly high in contact sports such as American football, boxing, ice hockey, basketball, and soccer.[19–22,48,49] An epidemiologic study conducted in the USA reported concussions in 3.02 male players per 1,000 American college football games and in 0.38 male and 1.8 female players per 1,000 soccer games. It has been claimed that females are more susceptible to concussion than males.[19–22,48]

After sustaining a first concussion, the risk for a second concussion is 2 to 5.8 times greater, and with each subsequent concussion, the risk for concussion increases, the symptoms tend to become more severe,[48–50] and the recovery time is longer.[50] Although there is no evidence that children and adolescents are at greater risk for concussion, it takes younger individuals longer to recover.[46,51]

IV. Symptoms and assessment

According to the most recent international conference on concussions, an individual with one or more of the following symptoms is suspected of having a concussion[19,20]:

1. Subjective symptoms: somatic (e.g., headache), cognitive (e.g., feeling like being in a fog)
2. Physical signs (e.g., loss of consciousness, amnesia)
3. Behavioral changes (e.g., irritability)
4. Cognitive impairment (e.g., slowed reaction time)
5. Sleep disturbance (e.g., insomnia)

The most common concussion symptom is headache,

followed by dizziness and a spaced-out feeling. The incidence of loss of consciousness is only around 10%.[19–22] As cognitive impairment may manifest several hours after a head injury careful monitoring is required. Headache, dizziness, nausea, fatigue, and drowsiness are factors that increase the risk for a second concussion.[21,22,49]

To avoid overlooking any of these concussion symptoms, the International Conference on Concussion in Sports recommends a grading scale for concussion known as the Sports Concussion Assessment Tool (SCAT). Its latest version, SCAT3, was introduced at the 4th International Conference,[19–20] SCAT3 for children (aged 5 years to 12 years) was established, and the Pocket Concussion Recognition Tool was revised. This revision is noteworthy. In its introduction it stresses "visible clues of suspected concussion" because on the field, signs of concussion must be recognized immediately and the injured athlete must be removed from the game. Concussion assessments are revised regularly, new findings are incorporated, and different countries and sports organizations participate in the updating process.

V. Repeated concussion

In 80% of concussions, symptoms resolve spontaneously within 10 days.[47,51] However, in 10% to 20%, concussion symptoms persist for several weeks to months.[20,21] Children, adolescents, and individuals with prior concussion require more time to recover and the recovery time becomes longer with each subsequent concussion.[48–53] Even after symptom abatement, the physiological/biochemical impact on the damaged brain can last for up to 10 days as shown in animal models of concussion.[45,46]

An athlete with a concussion or minor head injury who returns to play before complete recovery and then sustains a second minor head injury may suffer fatal brain swelling. This is known as the second impact syndrome.[1–4] Cases of second impact syndrome have been confirmed in the types of sport where concussions and mild head injuries are common such as American football. With respect to the pathogenesis of second impact syndrome, impairment of cerebral vascular autoregulation is thought to underlie the development of acute brain swelling although the existence of this phenomenon is controversial[54] and no conclusion on the existence of this pathological condition has been reached.

As second impact syndrome is occasionally accompanied by ASDH,[4,54,55] the mechanism underlying its development may be its association with ASDH. Regardless, it is undoubtedly dangerous, and it may be fatal, for an athlete to RTP before resolution of the symptoms of the first concussion as even a minor head injury can result in a severe condition. If headache or other symptoms persist, diagnostic imaging is necessary to rule out small subdural hematoma.[16]

CTE

Repeated concussions increase the risk for future concussions and prolong the duration of concussive symptoms.[21,48–51] With repeated concussions the risk for not only functional but also structural/microscopic anatomical brain damage increases, and accumulated brain damage may lead to CTE.[10–14] Visually noticeable brain atrophy may develop in the long run and result in higher brain dysfunction, psychiatric disorders, or the motor symptoms of Parkinson's disease. Martland[56] first reported this condition in boxing as the "punch-drunk" syndrome and it may be anticipated in other contact sports such as American football, soccer, ice hockey, and martial arts. Compelling epidemiological evidence indicates that a single moderate to severe traumatic brain injury increases the risk for the development of progressive disorders and cognitive impairment leading to dementia.[14]

According to recent studies in which American football players underwent neuropsychological testing and functional MRI, even a minor head injury with no concussive symptoms can lead to abnormal results in neuropsychological tests. This is known as subconcussion and has drawn attention because repeated subconcussions can result in abnormal findings on functional MRI or the development of CTE.[9,13]

Detailed postmortem studies on CTE resulting from repeated mild head injuries detected this condition not only in American football players but also in individuals who experienced blast injuries in a war zone. There were striking similarities in the severity of their antemortem higher brain dysfunction and psychological and motor dysfunction, and in pathological findings that detected tau-positive neurodegenerative changes, neuronal loss, axonopathy, and brain atrophy.[11] McKee et al.[11,57] classified the pathological severity into four stages (Table 1). In Stage IV, brain atrophy, neuronal degeneration, and the accumulation of phosphorylated tau protein (p-tau) in widespread regions of the frontal lobe, temporal lobe, hippocampus, and parietal lobe are present.

RTP Criteria after Sports-related Head Injuries

I. Post-concussion RTP criteria

To avoid fatal consequences or chronic brain

damage resulting from repeated concussions, post-concussion RTP criteria must be understood. First, if a concussion is suspected on the field, the athlete must be removed immediately from the game. If the state of consciousness or the severity of neurological dysfunction is suggestive of a condition more serious than concussion, e.g., intracranial hematoma, the injured athlete must be transported immediately by ambulance to an urgent care facility with a neurosurgical service. To determine whether the athlete has sustained a concussion, the Pocket Concussion Recognition Tool or SCAT3 can be used.[19,20] When a concussion is suspected, the athlete must be evaluated by on-site medical staff with knowledge about concussions, or examined by a physician.

When concussion is diagnosed, the athlete must not be allowed to RTP on the same day[19–22] and must take sufficient physical/mental rest.[19,20,47] It is not acceptable for the athlete to RTP before complete resolution of the concussion symptoms. Children and adolescents need more time to recover.[19–22,46,50] After the symptoms have resolved, the athlete should be placed in a step-by-step RTP program that starts with light aerobic exercises and gradually increases the load (Table 2).[16,19–22] The athlete can proceed to the next step every 24 hours. The program entails 6 steps, therefore the minimum interval between the injury and the RTP is 1 week. Before ascending to the last step, the athlete must undergo a medical checkup. If symptoms appear during the course of the program, the athlete must take a break and re-start from step one.

Table 1 Pathology and clinical symptoms in patients with chronic traumatic encephalopathy

CTE stage	Number of patients	Clinical symptoms	Pathology
I	6	Headache, loss of attention	P-tau deposition in dorsolateral part of frontal lobe and around the cerebral vessels
II	14	Headache, depression, mood swings	Mild ventricular dilatation, multiple p-tau deposition in frontal and temporal lobe
III	12	Higher brain dysfunction dementia	Mild brain atrophy, p-tau deposition in cortex and subcortex, hippocampus, axonal loss
IV	15	Dementia, apathy, Parkinson syndrome, aggressiveness	Brain atrophy, neuronal and axonal degeneration in whole cortex, white matter and deep gray matter

Contents were summarized from autopsy study reported by McKee et al.[11] CTE: chronic traumatic encephalopathy, p-tau: phosphorylated tau protein.

II. Proper management of athletes with organic brain damage

In the confirmed presence of brain damage or subdural hematoma the athlete must not be permitted to return to any contact sports involving frequent blows to the head or rotation of the head (especially boxing, karate, judo, sumo, rugby, American football, soccer, ice hockey, snowboarding) even if the symptoms have cleared and the lesions are no longer visible on neuro-images.[16] In the literature on sports-related head injuries published in Japan, Europe, and the USA we found no clear guidelines on whether and when athletes with organic brain lesions such as intracranial bleeding can be allowed to RTP if their symptoms have resolved and abnormalities on neuro-images have disappeared. According to Davis et al.,[58] athletes with a small subdural hematoma that does not require surgery can be allowed to RTP as long as the athlete has become asymptomatic and there are no abnormalities on neuro-images. However,

Table 2 Graduated return to play (RTP) Protocol (modification of the literature[19–22])

Rehabilitation at age	Functional exercise at each stage of rehabilitation	Objective(s) of each stage
1. No activity	Symptom-limited physical and cognitive rest	Recovery
2. Light aerobic exercise	Walking, swimming, or stationary cycling, keeping intensity 70% of maximum permitted heart rate; no resistance training	Increase heart rate
3. Sport specific exercise	Skating drills in ice hockey, running drills in soccer; No head-impact nor head-rotation activities	Add movement
4. Noncontact training drills	Progression to more complex training drills, e.g., passing drills in football and ice hockey; may start progressive resistance training	Exercise, coordination, and cognitive
5. Full-contact practice	After medical clearance, participation in normal training activities	Restore confidence and assessment of functional skills by coaching staff
6. RTP	Normal game play	

we encountered adolescent judo participants who were considered cured from thin ASDH and returned to play; they later suffered fatal ASDH.[15,16] As most judo-related accidents happen in young inexperienced participants, even if a small subdural hematoma has been absorbed and neuro-imaging returns normal findings, in principle, children and adolescents must not be allowed to RTP.

There are no clear guidelines on how to proceed after an athlete has sustained a small brain hemorrhage, brain contusion, or presents with an organic intracranial lesion such as arachnoid cyst, or has just undergone a craniotomy procedure. For now, such cases should be dealt with on an individual basis and the best approach should be chosen after information-sharing and discussions among medical professionals, the athlete, and his/her parents, coaches, and instructors.

III. Prevention of sports-related head injuries and future challenges

Helmets or head gears are required in sports such as American football, rugby, amateur boxing, and ice hockey. Although this type of head protection can prevent direct damage to the head and brain contusion, there is no evidence that it helps to reduce the incidence of concussion and subconcussion or of rotational acceleration injury including ASDH.[19–22] Similarly, mouth guards can help to prevent injuries to the face and mouth cavity but they have not been proven effective for the prevention of concussion.[19–22]

Even if it is impossible to prevent every single incident of concussion or ASDH, studies are necessary to identify effective preventive measures and information on the risks of different sports must be relayed to sports organizations, educators, and the public.

Much remains unknown on the pathogenesis and pathophysiology of concussion, ASDH, and CTE. As the mechanisms underlying their development and contributing factors vary depending on the sport, the particularities of each sport must be understood. Clinically, it is important to explore how sports-related head injuries develop and how they can be prevented. Biomechanical studies using artificial models (e.g., dummies[59]) and studies on animal concussion models will help to address this issue.[60,61]

Registry and epidemiological studies on trauma cases and prospective studies on specific groups of athletes can be expected to yield significant findings. Increased attention has focused on the detection of subclinical functional abnormalities (the presence of subconcussion) by neuropsychological testing and functional MRI studies performed after concussion or mild brain injury.[9] Any of these studies may help to determine whether it is advisable for an injured athlete to resume former sports activities and detailed clinicopathological studies have advanced our understanding of CTE resulting from repeated injuries.[11]

Individuals such as instructors, athletes, and parents who are involved in competitive sports must be cognizant of scientific evidence and the proper clinical management of sports-related head injuries. Since the incidence of concussion is high in physical education classes in schools and in daily sports practice in after-school athletics clubs, on-site instructors and coaches must be able to identify athletes with suspected concussion and to properly manage the situation at the time of injury. Therefore, they should undergo instructor training. Information on the risk for and management of head injuries in athletes participating in different sports should be widely disseminated in educational institutions and by public relations campaigns of sports organizations. Efforts must be made to educate not only personnel and parents directly involved in the care of athletes but also the general public.

Acknowledgments

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Conflicts of Interest Disclosure

The authors have made declaration of COI every year to the Japan Neurosurgical Society. The authors have no personal, financial, or institutional relationships with other people or organizations that could inappropriately influence in this work.

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