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Long-term Neurological Sequelae of Exertional Heat Stroke in Athletes - A Narrative Review

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Abstract

Background. Exertional heat stroke (EHS) is a severe, life-threatening condition defined by extreme hyperthermia and central nervous system (CNS) dysfunction. With rising global temperatures and growing participation in endurance and outdoor sports, the long-term neurological consequences of EHS are becoming increasingly relevant for both competitive and recreational athletes.

Aim. This narrative review aimed to synthesise the mechanisms, risk factors, and clinical outcomes of long-term neurological sequelae following EHS with a focus on athletic populations.

Material and methods. A comprehensive literature search was performed in PubMed, Scopus, and Web of Science from 2000 to April 2025, supplemented by selected foundational studies. Thematic narrative synthesis was conducted on original articles, case series, case reports, and reviews focusing on neurological outcomes after exertional (not classic) heat stroke in athletes and physically active individuals.

Results. EHS leads to CNS injury through direct thermal damage, blood-brain barrier disruption, and intense neuroinflammation, with selective vulnerability of cerebellar Purkinje cells. Cerebellar syndromes, including ataxia, dysarthria, and nystagmus, represent the most frequent long-term sequelae. Permanent neurological deficits occur in a significant minority of survivors, and the speed of cooling is the most critical modifiable factor influencing neurological prognosis.

Conclusions. Long-term neurological sequelae after exertional heat stroke, particularly cerebellar dysfunction, constitute a clinically important and potentially preventable burden for athletes. Current guidelines focus mainly on systemic recovery, while specific neurological return-to-sport protocols are lacking. There is an urgent need for athlete-specific prospective studies, standardised neurocognitive follow-up, and development of evidence-based neurological clearance criteria to ensure safe return to sport.

Key words: exertional heat stroke, neurological sequelae, cerebellar ataxia, athletes, return-to-sport, rapid cooling, EHS

1. Introduction

Exertional heat stroke (EHS) is a life-threatening condition characterised by extreme hyperthermia and central nervous system (CNS) dysfunction. It remains one of the leading causes of collapse and death in athletes and the military personnel [1, 2]. With rising global temperatures and increasing participation in endurance sports, both the incidence of EHS and its potential long-term consequences are gaining greater clinical importance [3, 2].

1.1 Epidemiology and Clinical Significance of EHS

EHS is one of the top causes of non-traumatic death in athletes, including healthy young individuals engaged in vigorous physical activity, particularly in endurance training, team sports, and military training, and is the most severe exertional heat illness [1, 2]. Incidence rates vary significantly depending on the setting, with well-documented cases in marathon and road races as well as large military cohorts [1, 2, 4]. Climate change and more frequent heatwaves are expected to further increase the risk of EHS in outdoor sports and occupations [3, 2, 5].

Clinically, heatstroke is defined by a core body temperature typically exceeding >40-40.5°C accompanied by CNS dysfunction. It is classically divided into two main forms: classic (non-exertional) heat stroke, which usually affects vulnerable populations during environmental heatwaves, and exertional heat stroke, which occurs during intense physical exercise, often in hot and humid conditions [1, 3].

Aspect	Classic Heat Stroke	Exertional Heat Stroke	Citations
Typical population	Elderly or individuals with comorbidities	Young, fit athletes and soldiers	[3, 1, 6]
Trigger	Passive exposure to extreme heat	Vigorous exercise (± hot/humid environment)	[3, 1, 6]
Core features	>40 °C + CNS dysfunction, risk of multi-organ injury	Same diagnostic core	[3, 1, 6]

Table 1: Comparison of classic vs exertional heat stroke definitions and populations

1.2 The Neurological Dimension: Why It Matters

CNS dysfunction (e.g., delirium, seizures, ataxia, or altered consciousness) is the cardinal diagnostic criterion of heatstroke and is present in nearly all severe cases [1, 3]. While many acute neurological manifestations improve with rapid cooling, a significant minority of survivors develop persistent deficits, including cerebellar ataxia, dysarthria, cognitive impairment, memory disturbances, and executive dysfunction [7, 8].

Cerebellar dysfunction is particularly common among those who experience long-term neurological sequelae [7]. In athletes and military personnel, such deficits - together with residual heat intolerance and psychological symptoms - can substantially impair return to sport, occupational performance, and overall quality of life [9, 2].

1.3 Gap in the Literature and Aim of This Review

Numerous reviews have addressed the pathophysiology, acute management, risk factors, and multi-organ consequences of EHS, often combining classic and exertional forms or heterogeneous patient populations [3, 1, 2]. Long-term neurological outcomes are typically mentioned only briefly and are based largely on case reports, small case series, or mixed populations, with limited athlete-specific data and insufficient integration of underlying mechanisms.

To date**, no dedicated review has focused specifically on long-term neurological sequelae of exertional heat stroke in athletic populations**, synthesising mechanisms of CNS injury, sport-specific risk factors, clinical trajectories, and implications for return-to-sport.

Moreover, while much of the long-term data derives from military cohorts, differences in exposure and follow-up limit direct extrapolation to athletic populations.

This narrative review therefore aims to:

- synthesise the current evidence on mechanisms of CNS injury in EHS,
- describe the types, incidence, and long-term neurological and neuropsychological outcomes after EHS, with particular emphasis on athletes and military personnel,
- outline known and potential risk factors for persistent CNS sequelae, and

- highlight clinical implications and research priorities to improve prevention, follow-up, and safe return-to-sport in athletes.

2. Methods

This narrative review aimed to synthesise the current evidence on long-term neurological sequelae of exertional heat stroke (EHS) in athletes.

2.1 Search strategy

A comprehensive literature search was performed in PubMed, Scopus, and Web of Science. The search covered publications from January 2000 to April 2025, with selected foundational studies published before 2000 included when relevant. Keywords and MeSH terms were combined using Boolean operators (AND/OR): “exertional heat stroke”, “heat stroke”, “neurological sequelae”, “long-term outcomes”, “cerebellar ataxia”, “cognitive impairment”, “athletes”, and “sports”. No initial language restrictions were applied, although only articles available in English or Polish were included in the final review.

2.2 Inclusion and exclusion criteria

Articles were included if they reported neurological outcomes following exertional (not classic) heat stroke in competitive or recreational athletes or physically active populations. Eligible publication types included original research articles, case series, case reports, and previous narrative or systematic reviews. Articles were excluded if they focused exclusively on classic (non-exertional) heat stroke, presented only animal data without clear human translation, or were editorials, commentaries, or conference abstracts without full-text availability.

2.3 Data extraction and synthesis

Titles and abstracts were screened, followed by full-text assessment of potentially relevant articles. Data extraction and synthesis were conducted manually by the author using a thematic narrative approach. Studies were organised according to the following main themes: pathophysiology of central nervous system injury in EHS, types and incidence of long-term neurological sequelae, risk factors, neuroimaging and biomarkers, and clinical course with implications for return-to-sport. No formal quality assessment of individual studies or meta-analysis was performed, consistent with the narrative review design.

2.3.2. AI.

AI was utilized for two specific purposes in this research. Text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. AI were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

3. Results

3.1 Pathophysiology of CNS Injury in EHS

Exertional heat stroke (EHS) causes central nervous system (CNS) injury through multiple overlapping mechanisms, including direct hyperthermia-induced neuronal damage, disruption of the blood-brain barrier, and a pronounced neuroinflammatory response. Key mediators involved in this cascade include high-mobility group box 1 (HMGB1), the NLRP3 inflammasome, interleukin-1β (IL-1β), and IL-18 [1, 3, 10, 11]. These processes lead to cytotoxic edema, particularly affecting vulnerable brain regions such as the cerebellum, hippocampus, thalamus, and basal ganglia [1, 10]. A characteristic histopathological finding is the selective degeneration of Purkinje cells in the cerebellum, which is strongly associated with long-term neurological deficits [12, 13].

3.1.1 Blood-Brain Barrier Disruption in Exertional Heat Stroke

One of the critical mechanisms underlying central nervous system (CNS) injury in exertional heat stroke is the disruption of the blood-brain barrier (BBB). Under severe hyperthermia, tight junctions between endothelial cells are compromised, leading to increased permeability of the BBB [14]. This process is mediated by the activation of matrix metalloproteinases, particularly MMP-9, which degrade components of the basement membrane and tight junction proteins such as occludin and claudin-5.

The opening of the BBB allows extravasation of plasma proteins and inflammatory mediators into the brain parenchyma, exacerbating cytotoxic edema. This edema further increases intracranial pressure and contributes to secondary neuronal injury, particularly in vulnerable regions such as the cerebellum and hippocampus [15]. In the context of EHS, this vicious cycle of hyperthermia-induced BBB breakdown, neuroinflammation, and cytotoxic swelling is considered a central pathway leading to long-term neurological sequelae, including cerebellar ataxia and cognitive impairment [16].

3.2 Types and Incidence of Long-term Neurological Sequelae

Cerebellar syndromes, manifesting as ataxia, dysarthria, and nystagmus, represent the most common long-term neurological sequelae after EHS. In a review of case reports and case series of environmental heat stroke, cerebellar dysfunction accounted for approximately 71% of persistent neurological deficits among survivors who developed long-term complications [7]. Other frequently reported sequelae include cognitive impairment (particularly anterograde amnesia), epileptic seizures, and frontal-subcortical dysfunction [8, 17, 18].

Magnetic resonance imaging (MRI) commonly demonstrates cerebellar atrophy within 90 days post-event, with changes that may persist or progress in some cases [7, 17, 12]. Overall, permanent neurological deficits have been documented in approximately 23-34% of survivors with known outcomes, although these figures derive largely from mixed or environmental heat stroke cohorts. Many affected individuals are young and previously healthy athletes or military personnel [7, 9].

3.3 Risk Factors for Developing Neurological Sequelae

The development of long-term neurological sequelae is strongly associated with the severity and duration of the initial CNS insult. Key risk factors include a low Glasgow Coma Scale score on admission, delayed cooling, recurrent hyperthermia during hospitalisation, co-existing rhabdomyolysis, and disseminated intravascular coagulation (DIC) [9, 17, 19].

The most important modifiable factor is the time to effective cooling. Achieving a rapid reduction in core temperature (e.g., cooling rate >0.15°C/min) is consistently linked to better neurological outcomes [1, 20]. Additional factors such as higher disease severity scores (SOFA or APACHE II) and possible genetic predispositions (e.g., RYR1 mutations) have also been suggested. In athletic contexts, psychological factors including high motivation and pressure to

perform may indirectly increase risk by encouraging individuals to push beyond safe physiological limits [21].

3.4 Neuroimaging and Biomarkers

Neuroimaging findings in EHS-related CNS injury are often delayed. Early MRI may appear normal despite evolving damage, while cerebellar atrophy and lesions in the hippocampus or basal ganglia typically become evident weeks to months after the event [7, 17, 12]. Serum biomarkers such as S100β and neuron-specific enolase (NSE) show potential for assessing acute CNS injury severity, although their ability to predict long-term outcomes remains limited due to timing variability [24]. In selected cases, single-photon emission computed tomography (SPECT) may provide additional prognostic information when structural MRI is inconclusive [23].

Neuroimaging plays a crucial but time-dependent role in evaluating CNS injury after EHS. In the first 48 hours, brain MRI is often normal despite significant clinical encephalopathy, reflecting the delayed evolution of structural damage [24].

After 7-14 days, characteristic changes typically become visible. The most common findings include symmetrical hyperintense lesions on T2-weighted and FLAIR sequences in the cerebellar vermis, dentate nuclei, hippocampi, thalami, and basal ganglia. Cerebellar atrophy, particularly involving the vermis, is a frequent late finding and correlates with persistent ataxia [25, 24]. In severe cases, diffuse cerebral edema or selective neuronal necrosis may be observed. These imaging patterns highlight the selective vulnerability of certain brain regions to hyperthermic injury and are valuable for prognostic assessment, although early normal MRI does not exclude the possibility of long-term neurological deficits.

3.5 Clinical Course and Prognosis

The clinical course after EHS-related neurological injury is highly variable. Some patients show partial or substantial recovery, while others experience permanent deficits that significantly affect quality of life, return-to-sport, and occupational functioning [9, 18, 26]. Cerebellar ataxia tends to be the most persistent sequela, whereas cognitive recovery is often incomplete. Rapid cooling, particularly using cold water immersion, is associated with a lower incidence of permanent deficits [1, 20]. Nevertheless, many survivors report residual symptoms such as fatigue, heat intolerance, and mood disturbances months to years after the incident [9].

3.6 Differences between Athletic and Military Populations

Although exertional heat stroke (EHS) affects both athletes and military personnel, important differences exist in exposure, constraints, and follow-up that influence long-term neurological outcomes.

In military settings, EHS often occurs during prolonged high-intensity activity under command, with heavy uniforms, body armor, and load carriage limiting heat loss and self-pacing. Recruits frequently have incomplete acclimatization, and external performance pressure is a recognised risk factor [2, 27]. These cohorts provide the most robust long-term data due to systematic surveillance.

In athletes, EHS typically arises during competitions or intense training. While athletes usually have greater autonomy and better acclimatization, strong cultural pressure to perform and return to sport quickly often leads to ignoring early symptoms [9, 28].

Long-term outcome data are disproportionately derived from military and mixed cohorts (neurological sequelae reported in 6-24% of cases), whereas athletic data consist mainly of case reports and small series [1]. Military findings may therefore overestimate risk in civilian athletes due to heavier equipment and stricter constraints [9]. Conversely, athletic studies likely underestimate true incidence because of shorter follow-up and lack of routine neuropsychological testing [29].

3.7 Differential Diagnosis on the Race Course

In the acute setting, particularly during endurance events such as marathons, exertional heat stroke must be rapidly differentiated from other life-threatening conditions that may present with collapse and altered mental status.

The most important differential diagnoses include exercise-associated hyponatremia (EAH), severe hypoglycemia, sudden cardiac arrest (SCA), and acute ischemic stroke. EAH can mimic EHS with confusion, seizures, and coma, but is typically associated with weight gain and low serum sodium [30]. Hypoglycemia usually presents with sweating, tremor, and rapid improvement after glucose administration. SCA requires immediate CPR and defibrillation, while ischemic stroke may show focal neurological signs.

Compensatory neurohumoral responses to severe heat stress, including sympathetic activation and dehydration, can provoke dangerous blood pressure fluctuations and acute cardiovascular events, complicating field-level assessment [5].

Condition	**Core Temperature**	**Key Clinical Features**	**Diagnostic Clues**	**Initial Management**
Exertional Heat Stroke (EHS)	>40.5°C	Hot, dry or sweaty skin, CNS dysfunction	Rectal temperature >40.5°C	Immediate cold water immersion
Exercise-Associated Hyponatremia	Normal / low	Headache, vomiting, seizures, confusion	Weight gain, low serum Na+	Fluid restriction / hypertonic saline
Severe Hypoglycemia	Normal	Sweating, tremor, confusion	Rapid response to glucose	Intravenous glucose
Sudden Cardiac Arrest	Normal / low	Unresponsive, no pulse	Abnormal ECG, collapse without warning	CPR + defibrillation
Acute Ischemic Stroke	Normal	Focal neurological deficits	Asymmetrical signs, FAST positive	Neurological evaluation + imaging

Table 2. Differential diagnosis of collapse with altered consciousness during endurance events [30]

4. Discussion

4.1 Synthesis: Why Athletes Are a Distinct Population

Athletes constitute a unique population in the context of exertional heat stroke (EHS). They are typically young, highly fit, and possess substantial physiological reserves, which can paradoxically mask early signs of neurological dysfunction and delay recognition and treatment

[1, 2]. In addition, cultural and professional pressures to perform, combined with the strong motivation to return to sport quickly, may lead athletes to ignore prodromal symptoms or resume training prematurely, thereby increasing the risk of severe and persistent injury [9, 28].

Unlike classic heat stroke, which predominantly affects older individuals with comorbidities during passive heat exposure, EHS in athletes occurs in otherwise healthy young people during intense physical exertion. This distinction is important because many cases of long-term neurological sequelae develop in individuals who were previously considered low-risk [7, 9].

4.2 The Central Role of Rapid Cooling in Prevention of Sequelae

Rapid cooling is the most critical modifiable factor in determining neurological outcome after EHS. The principle of "cool first, transport second" is now widely endorsed, with cold water immersion recognised as the gold standard cooling method [1, 20, 31]. Evidence consistently shows that delays in achieving effective cooling are strongly associated with increased severity of CNS injury and higher rates of long-term sequelae, including cerebellar ataxia and cognitive impairment [19, 32, 17). Even short delays can result in irreversible neuronal damage, particularly to Purkinje cells in the cerebellum. Despite clear recommendations, implementation of immediate on-site cooling remains inconsistent, especially in non-professional or remote sporting events [9, 33].

4.3 Absence of Neurological Return-to-Sport Protocols

Current clinical guidelines, including those from the American College of Sports Medicine (ACSM), primarily emphasise systemic recovery (normalisation of core temperature, laboratory parameters, and organ function) rather than targeted neurological assessment before return-to-sport [31, 33]. There is currently no validated, sport-specific protocol for neurological clearance following EHS, in contrast to the well-established graduated return-to-play protocols developed for sport-related concussion.

This represents a significant clinical gap. Subtle neurocognitive deficits or cerebellar dysfunction may persist even after systemic recovery and could compromise both athletic performance and safety upon return to competition [9, 34]. The successful evolution of concussion management protocols demonstrates that structured, evidence-based neurological return-to-sport pathways are both feasible and effective. A similar approach is urgently needed for athletes recovering from EHS.

Given the similarity of potential persistent deficits (cerebellar, cognitive, balance) to those seen after sport-related concussion, this review proposes a Graduated Neurological Return-to-Sport Protocol after EHS, adapted from the Berlin Consensus Statement on Concussion [35].

During physical exertion in extreme heat, the profound conflict between thermoregulatory skin blood flow and working muscle requirements creates a severe cardiovascular bottleneck, compounding myocardial strain [16].

Proposed 5-Step Graduated Neurological Return-to-Sport Protocol after EHS:

1. Complete Rest and Symptom Resolution - Full physical and cognitive rest until complete resolution of neurological symptoms (headache, dizziness, ataxia, cognitive fog) for at least 48 hours.
2. Light Aerobic Exercise - Low-intensity aerobic activity (e.g., walking, light jogging) in a cool environment combined with basic balance testing (BESS test). No resistance training.
3. Sport-Specific Training without Heat Stress - Gradual introduction of sport-specific movements, moderate intensity training in thermoneutral conditions, plus neuropsychological testing (e.g., ImPACT or SCAT5).
4. Controlled Heat Exposure - Training in progressively warmer conditions with monitoring of core temperature and neurological symptoms. Focus on heat tolerance and balance under fatigue.
5. Full Medical Clearance and Return to Competition - Comprehensive neurological evaluation (including MRI if indicated), neuropsychological clearance, and final approval by a sports medicine physician.

The design of post-EHS recovery and clearance frameworks must account for the high heterogeneity in athletic thermal responses. As highlighted by Burda et al. (2026) in their systematic review of passive heat exposure in athletes, thermal interventions yield highly selective and non-uniform effects on recovery and neuromuscular performance. Therefore, indiscriminate return to training without structured, graduated protocols risks inducing systemic or neurological overload rather than beneficial adaptation [36]. Each step should last a minimum of 24-48 hours with close monitoring. Any return of neurological symptoms requires regression to the previous step.

4.4 Limitations of Current Evidence

The existing literature on long-term neurological sequelae after EHS has important limitations. Much of the data derives from military cohorts or intensive care unit populations rather than civilian athletes, which may limit direct generalisability [7, 19]. Follow-up periods are often short and heterogeneous, and mild or subclinical neurological deficits are likely underreported due to the absence of routine neuropsychological testing. Most studies are retrospective case series or small cohorts, with few prospective registries available. Additionally, there remains a lack of consensus regarding standardised diagnostic criteria for post-EHS brain injury, making cross-study comparisons challenging [9, 32]. A major limitation is the predominance of military and ICU data, which may not fully reflect the risk profile and recovery patterns of civilian athletes due to differences in equipment, pacing autonomy, and post-event monitoring.

4.5 Future Research Directions

Future studies should prioritise the establishment of prospective, athlete-specific registries with standardised long-term neurological follow-up, including detailed neuropsychological assessment. The development and validation of sport-specific return-to-sport protocols following EHS are urgently required. Emerging mechanistic insights also support the exploration of adjunctive neuroprotective strategies (e.g., modulation of heat shock proteins or anti-inflammatory agents). Finally, genetic screening for variants such as RYR1 mutations may help identify athletes at heightened risk and inform personalised prevention strategies [10, 37]. Future research should prioritise prospective studies focused specifically on competitive and recreational athletes, incorporating standardised neuropsychological and balance testing.

5. Conclusion

Long-term neurological sequelae following exertional heat stroke (EHS) represent a significant clinical concern for athletes. Although many patients recover fully, a substantial minority develop persistent deficits, with cerebellar dysfunction being the most common and often most disabling manifestation. Rapid cooling remains the single most important modifiable factor in preventing permanent central nervous system injury.

This narrative review highlights that current evidence, while growing, is limited by heterogeneity, reliance on military and mixed cohorts particularly given the differences between athletic and military contexts, and the lack of standardised neurological follow-up in athletic populations. Despite systemic recovery, subtle neurocognitive and cerebellar impairments can persist and negatively affect return-to-sport, performance, and quality of life.

There is an urgent need for athlete-specific prospective studies, routine neuropsychological assessment after EHS, and the development of structured, neurologically informed return-to-sport protocols. Until such protocols are established, clinicians should adopt a cautious approach to clearance, prioritising comprehensive neurological evaluation beyond simple normalisation of core temperature and laboratory values. Implementing these measures will be essential to safeguard the long-term brain health of athletes in an era of increasing environmental heat stress.

Disclosure

Supplementary Materials:

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