Evaluation and Treatment of Injury and Illness in the Ultramarathon Athlete

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KEYWORDS

- Ultramarathon
- Injury
- Illness
- Medical
- Musculoskeletal
- Hyponatremia

KEY POINTS

- In ultramarathoners, most musculoskeletal and skin-related issues are minor and can be treated successfully during the course of a race.
- Common medical illnesses, including hyperthermia and exercise-associated hyponatremia, require prompt assessment.
- Multistage ultramarathoners are more likely to experience hypernatremia than hyponatremia.
- Continued research should focus on preventative and optimal treatment strategies in hopes of preventing long-term complications in this unique athletic population.

INTRODUCTION

Ultramarathon races represent any foot race longer than 42 km; an estimated 70,000 runners participate yearly in running races throughout the world.¹² Most races are single-stage point-to-point continuous races occurring over a specific period (ie, 1 to 2 days). Multistaged races are point-to-point races that occur over 3 to 7 days. Ultramarathons typically occur in more extreme environments with variations in terrain...
(mountains, snow, sand dunes, river crossings, slot canyons), temperature, and humidity. Ultramarathon runners require different equipment depending on the length and environment of the race. In multistage races, runners must be prepared to carry all their gear (eg, food, water, protective clothing) throughout the race.

Our knowledge of common injuries and illnesses in ultramarathon runners continues to increase. In the 1990s, studies focused on musculoskeletal injuries, noting that most running-related injuries were caused by Achilles tendinopathy (2%–18%) and patellofemoral pain (7%–15%).

In 2011, Krabak and colleagues' prospective study of multistage ultramarathon runners suggested that 95% of injuries are minor and are caused by skin-related disorders (74.3%), musculoskeletal injuries (18.2%), and medical illnesses (7.5%) (Table 1). Other studies have focused on often asymptomatic but potentially injurious diseases, like exercise-associated hyponatremia (EAH) and acute kidney injury (AKI), reporting an EAH incidence of 8% to 50% and AKI in more than 50% of the studied athletes.

With the increase in ultramarathons come inherent challenges relating to the unique environments, training demands, nutritional preparation, and equipment. These challenges provide education and research opportunities for both physicians and athletes.

### Table 1

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Marathon, n (%)</th>
<th>Multistage Ultramarathon</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Major, n (%)a</td>
<td>Minor, n (%)a</td>
</tr>
<tr>
<td><strong>Medical illnesses</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercise-associated collapseb</td>
<td>863 (59.4)</td>
<td>35 (56.5) 43 (3.9)</td>
</tr>
<tr>
<td>Altitude sickness</td>
<td>—</td>
<td>0 11 (1.0)</td>
</tr>
<tr>
<td>Serious medical diagnosisc</td>
<td>2 (0.14)</td>
<td>1 (1.6) 1 (0.1)</td>
</tr>
<tr>
<td>Other medical diagnosisd</td>
<td>7 (0.48)</td>
<td>0 27 (2.4)</td>
</tr>
<tr>
<td><strong>Musculoskeletal injuries</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bursitis</td>
<td>—</td>
<td>1 (1.6) 11 (1.0)</td>
</tr>
<tr>
<td>Sprain</td>
<td>19 (1.3)</td>
<td>2 (3.2) 25 (2.3)</td>
</tr>
<tr>
<td>Strain</td>
<td>207 (14.3)</td>
<td>1 (1.6) 27 (2.4)</td>
</tr>
<tr>
<td>Tendonitis</td>
<td>—</td>
<td>7 (11.3) 115 (10.3)</td>
</tr>
<tr>
<td>Othere</td>
<td>4 (0.28)</td>
<td>3 (4.8) 29 (2.6)</td>
</tr>
<tr>
<td><strong>Skin disorders</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abrasion</td>
<td>27 (1.9)</td>
<td>0 43 (3.9)</td>
</tr>
<tr>
<td>Blister</td>
<td>289 (19.9)</td>
<td>10 (16.2) 642 (57.8)</td>
</tr>
<tr>
<td>Cellulitis</td>
<td>—</td>
<td>1 (1.6) 8 (0.7)</td>
</tr>
<tr>
<td>Hematoma (subungual)</td>
<td>—</td>
<td>1 (1.6) 106 (9.5)</td>
</tr>
<tr>
<td>Otherf</td>
<td>—</td>
<td>00 23 (2.1)</td>
</tr>
</tbody>
</table>

a Major, unable to continue in race; minor, able to continue in race.
b Hyperthermia, normothermia, hypothermia.
c Hyponatremia, hematuria, renal stone.
d Blurry vision, conjunctivitis, diarrhea, dyspepsia, epistaxis, hematochezia, insect bite, neuropathy, pharyngitis, upper respiratory infections.
e Fracture, metatarsalgia, contusion, costochondritis, laceration, splinter.
f Callus, nail avulsion, rash, paronychia, wart.

Ill-prepared athletes and physicians place the athlete at risk for injury and illness. By better understanding the ultramarathon athlete, the sports medicine physician can provide optimal care and it is hoped limit morbidity and mortality. In this article, the understanding of strategies for managing commonly encountered musculoskeletal injuries and medical illnesses in ultramarathon runners is reviewed.

**MUSCULOSKELETAL INJURY**

Injuries to the musculoskeletal system are common in running sports. Reported musculoskeletal injury incidence varies depending on the methodology of the study. Musculoskeletal injury rates range from 2% to 18% in continuous single-stage ultra-marathons and 19% to 22% in multistage, multiday ultramarathons. In multi-stage, multiday ultramarathons, musculoskeletal injuries accounted for 18% of the minor encounters (able to continue racing) and 22% of the major injury encounters (unable to continue racing) and are most likely to occur during stages 3 or 4 of a 7-stage race, highlighting the potential cumulative effect on the musculoskeletal system of running long distances. Although general muscle soreness affects most ultramarathoners, true musculoskeletal injuries, whether minor or major, may decrease performance and result in decreased training or medical withdrawal from a race.

Lower extremity injuries predominate, with the knee and ankle being most affected. Evaluation and treatment of these injuries may differ in an acute setting during or immediately after a race compared with the subacute or chronic setting in a standard medical clinic setting. For the purposes of this article, the focus is the acute race or outpatient setting:

**Achilles Tendinopathy/Tendonitis**

Studies suggest a prevalence of 2.0% to 18.5% and incidence of 10.8%. The mechanism of injury is usually repeated or strained plantar flexion. Runners present for evaluation complaining of posterior heel or ankle pain. On physical examination, swelling or fullness may be visible in the affected Achilles region, although this is not always present. There is tenderness to palpation or squeezing of the tendon, most commonly at the midportion of the tendon, where there is a watershed vascular region of relatively low blood supply. Alternatively, the focus of tenderness may be at the insertion of the tendon at the posterior calcaneous, indicating that an enthesopathy or bursitis may be contributing to the pain. The runners may have pain with passive, manual stretch of the Achilles tendon. They usually have pain and possibly weakness with repeated single-leg toe raises and may be unable to perform full excursion for 5 to 10 repetitions. This method of testing strength is preferable to having them plantarflex against manual resistance, because their Achilles strength is greater than any examiner’s upper extremity–provided resistance, meaning a manual test would miss all but the most severe cases. A Thompson test (passive manual calf squeeze showing plantar flexion if Achilles is intact) should be performed to rule out a significant tendon tear. Treatments in the acute setting include icing or cold compress for 15 to 20 minutes (repeated several times during the evening while not running), gentle stretching of the tendon, and providing an overnight dorsiflexion splint or bandage wrap. If elastic, stretchy tape is available, application of plantarflexion assist elastic tape can help during ambulation or continued running but should be removed at night in favor of dorsiflexion splinting. Topical antiinflammatory creams can be used at any time if the overlying skin is intact. Oral analgesics like acetaminophen can be used to help decrease pain. Current guidelines recommend that oral nonsteroidal antiinflammatory (NSAID) medications should be used only at the end of the day/race section.
when the runner can be adequately hydrated to prevent AKI associated with NSAID use. In the subacute setting, sports medicine clinical treatment strategies may include rest, physical therapy with a focus on eccentric exercises, peritendon steroid injections, intratendon autologous blood product injections, or referral to surgery for recalcitrant cases.

### Anterior Tibialis Tendinopathy/Tendonitis

The mechanism of injury is usually repeated or strained dorsiflexion (particularly on a course that has prolonged, steep hills/inclines). Runners present for evaluation complaining of anterior ankle pain. On physical examination, swelling or fullness may be visible in the affected anterior tibialis tendon (Fig. 1A), although this is not always present. Occasionally, there is associated erythema in the anterior ankle as well (see Fig. 1B), and this must be closely followed to rule out a concurrent infection/cellulitis. They are tender to palpation of the tendon. They may have pain with passive, manual stretch of the anterior tibialis tendon, which is best elicited with combined passive ankle plantar flexion and passive great toe plantarflexion. The runners usually have pain and possibly weakness with repeated resisted ankle dorsiflexion, because they should be able to support the examiner’s application of upper body weight (not simply the examiner’s arm/hand strength) against dorsiflexed ankles. Treatments in the acute setting include icing or cold compress for 15 to 20 minutes (repeated several times during the evening while not running), and gentle stretching of the tendon. Topical anti-inflammatory creams can be used at any time if the overlying skin is intact. In multistage races, anecdotal cases of using an abundance of topical cream over the tendon with a bandage or occlusive dressing overnight has worked well, but it should be used with care and attention to possible skin reaction/allergy. Oral medications (analgesics and NSAIDs) follow the same guidelines as in the section on Achilles tendonitis. In the subacute setting, sports medicine clinical treatment strategies may include rest, physical therapy, peritendon steroid injections, intratendon autologous blood product injections, or referral to surgery for recalcitrant cases.

### Plantar Fasciitis

Studies have suggested an incidence of 10.6%. The mechanism of injury is repeated impact/shock absorption without proper arch support (either lack of support or too much support/pressure) or with higher-impact activities, even if arch support is correct for the corresponding anatomy and running style. Runners present for evaluation complaining of pain in the sole of the foot, frequently midarch, but it can be just proximal or distal to that. On physical examination, they are usually tender to palpation at the midarch or proximally along the plantar fascia toward the origin at the calcaneous. Pain may be reproduced with passive manual dorsiflexion, particularly if combined with toe extension. The runners may also have pain with resisted toe flexion in combination with ankle plantarflexed positioning. Treatment includes ice to the area, particularly ice massage by rolling a frozen or very cold water bottle underneath the arch after the day’s running is complete. Passive manual stretch of the plantar fascia and dorsiflexion splinting at night can also help improve symptoms. The same medication guidelines given earlier for Achilles tendonitis hold true. In the subacute setting, strengthening intrinsic muscle of the foot, running gait evaluation/recommendations, and trials of varying levels of arch support/shoe style can help treat symptoms and prevent recurrence. Rest, physical therapy, corticosteroid injections, autologous blood product injections, and referral to surgery may also be used in an outpatient treatment setting.
Fig. 1. (A) Acute anterior tibialis tendonitis (arrows) in the left distal tibia in an ultramarathon runner. (B) Acute anterior tibialis tendonitis presenting similar to a cellulitis.
**Patellofemoral Syndrome**

The prevalence in ultramarathon is 7.4% to 15.6%. Incidence of knee issues is 24%, although it is not clear how many of these are patellofemoral versus other knee issues (eg, meniscus tear, osteoarthritis). This injury tends to occur more often in the chronic setting but can occur as acute pain in ultradistance races. Mechanism of injury can be underlying structural anomaly of shallow femoral groove or lateralization of the patella versus inadequate quadriceps and core muscle strength for the demand distance running. Runners present with anterior knee pain. There are usually no symptoms of frank buckling or locking, but the injury can be associated with grinding sensation or crepitus. On physical examination, there is tenderness on the patellar facets, usually more on the medial aspect. There may also be tenderness along the patellar tendon, indicating a component of patellar tendonitis/tendinopathy as well. Pain may be reproduced with single-leg or double-leg squat or resisted knee extension. Acute treatment includes ice to the area and oral medications, as described earlier, taking care not to ice if the runner plans to continue running that day. A patellar tracking knee brace or band strap may help decrease pain with ambulation and running. Athletic tape can be used to fabricate a patellar band strap if none is readily available. If practitioners are trained in using kinesio tape for patellar tracking, that may also be helpful. In the subacute setting, physical therapy with attention to quadriceps and core strengthening can be used. Rest, steroid injections to the knee, viscosupplement injections if arthritis is associated, or referral to surgery for release of the lateral retinaculum or other interventions may also be used as treatment strategies.

**Ankle Sprain**

The incidence in ultramarathons is 10.8%. The mechanism of injury is usually an inversion rolling of the ankle on uneven terrain, causing strain or tear of any of the anterolateral ligaments of the ankle. The runner presents with anterior or lateral ankle pain. There may be swelling or ecchymosis, and the athlete may or may not be able to fully bear weight. On physical examination, the athlete has tenderness to palpation over the affected ligaments. Tenderness over the bone/lateral maleolus may indicate a fracture. If able to bear weight, they may have increased pain or instability when standing on the toes with the ankles plantarflexed. An anterior drawer test may show laxity compared with the contralateral side. Initial treatment consists of rest, ice, compression, and elevation. If any instability or ecchymosis is noted, or if there is a high suspicion of fracture (based on Ottawa ankle rules), they should be removed from competition. Oral medications can be used as described in the section on Achilles tendonitis. If fracture is suspected, the patient must remain non-weight bearing until transported to a facility with radiograph services for further diagnostic evaluation. Medial-lateral ankle brace support or a rocker bottom boot should be used in cases of instability in which fracture is not suspected. A lace-up ankle brace or athletic taping of the ankle can be used for those who have no instability and are able to walk without a limp to allow them to continue if competition if desired.

**Muscle Strain**

The most common muscle strains involve the calf (incidence 13.1%) and hamstring (incidence 11.8%). Mechanism of injury is usually an eccentric contraction or quick burst movement, which stresses the muscle to the point of injury without tearing a significant number of muscle fibers (although a small tear is difficult to differentiate from a strain). Most frequently involved muscles are those that cross 2 joints, like the hamstrings (crossing both hip and knee joints) and the gastrocnemius (crossing both
knee and ankle joints) Runners present with acute pain in the affected muscle. On physical examination, they may have pain with passive stretch or active contraction of the muscle. Initial treatment consists of rest, ice (after the day’s competition is finished), compression, and elevation, as noted earlier. Oral medications can be used as described in the section on Achilles tendonitis. A runner may be able to continue in competition with a mild strain, but anything that causes limping or altered gait should be cause to consider removing the athlete from competition for risk of further injury. If a full tear of a muscle is suspected, then they should be removed from competition immediately and referred for further evaluation at a hospital or sports medicine clinic. In the subacute setting, if relative rest for 1 to 2 weeks along with oral antiinflammatory medication and the other treatments noted earlier do not resolve the issue, then, physical therapy or advanced imaging studies may be warranted.

**Iliotibial Band Problem**

The incidence is 15.8%. Mechanism of injury is usually an overuse, more chronic presentation in runners but can be acute overuse in ultradistance running. The prevailing theory links this entity to impingement of the distal iliobibial band (ITB) at the lateral femoral condyle during the eccentric contraction just after heel strike. Runners present with lateral knee pain at the lateral femoral condyle or just proximally or distally. They may have exacerbation of pain with compression of the band/tendon over the femoral condyle (Noble compression test) or with stretch of the ITB (Ober test). Treatment during competition consists of stretching the ITB and using topical antiinflammatory cream locally (if overlying skin is intact). Oral medications can be used, as described earlier. Icing should be used at the end of the day’s competition, as described earlier. Cross-fiber friction massage can be used in the acute setting. In the subacute setting, rest, physical therapy, deep massage with foam roller, gait analysis, flexibility training, and core strengthening can be used for treatment. Other injections (eg, steroid, autologous blood products) may be considered if conservative treatment options fail.

**Back Injuries**

The incidence is 12.4%. Mechanism of injury can be varied, from acute muscle strain, spasm or disk injury to chronic degenerative change in the lumbar spine. Runners presenting with acute axial back pain without trauma may be treated as a strain (rest, ice oral medications) or if believed to have palpable spasm, then application of heat and gentle stretching are appropriate treatments. If the presentation includes pain radiating into the thigh or lower leg, or if lower extremity numbness/tingling is associated, then, they may have an acute lumbar radiculopathy and should be removed from competition, given oral antiinflammatory medication, and referred for nonemergent evaluation at a hospital or clinic. Any presentation of back pain with notable lower extremity weakness, numbness/tingling in the groin/genitals/rectal area, or loss of control of bladder or bowel function should be treated as a spinal emergency and the patient should be transported to a hospital for immediate evaluation and treatment.

**MEDICAL**

Exercise-associated collapse (EAC) is the collapse of the conscious athlete after an exertional event who is unable to stand or walk unaided because of light-headedness, faintness, or dizziness. It is common in marathons, (59%–69% of all medical encounters at the finish line), resulting in 10.1 to 13.7 medical illnesses per 1000 runners. EAC is less common during ultramarathons, representing 6.6% of all medical encounters during a multistage ultramarathon. However, EAC
accounted for 65% of medical illnesses for nonfinishers, resulting in 118 medical illnesses per 1000 runners. Although EAC may have a variety of causes, including heat-related illness, electrolyte abnormalities, cardiovascular compromise, respiratory compromise, and seizures, it is primarily the result of transient postural hypotension caused by lower extremity blood pooling. Once critical causes are ruled out, treatment is primarily symptomatic with rest in the Trendelenburg position, total body cooling, and if dehydrated, oral hydration (intravenous fluids are generally not needed). This section focuses on common medical issues, heat-related illness, and EAH. The unique environment of the ultramarathon dictates the likelihood of other medical illnesses encountered.

HEAT-RELATED ILLNESS

Most heat-related illness is mild and responds to minimal interventions. However, more severe heat-related illness such as heat stroke can lead to significant morbidity and mortality. Identification of potential risk factors and environmental conditions are early interventions that are important to managing heat-related illnesses.

Pathophysiology

Body temperature regulation occurs through the hypothalamus, which balances heat generation verses heat loss. Significant heat generation occurs with exercise, when skeletal muscles can increase their metabolic consumption by up to 20 times, with approximately 75% to 80% of that energy converted into heat. Heat lost to the environment occurs by 4 major methods: conduction, convection, radiation, and evaporation. Evaporation is the most important method of heat loss during exercise, and significant exercise can produce 1 to 2 L per hour of sweat loss, with the evaporation of 1.7 mL of sweat consuming 1 kcal of heat. High environmental humidity lowers the water vapor pressure difference between the skin and surrounding air, which hampers the ability of sweat to evaporate and subsequent heat loss. When the body senses an increase in core temperature, the thermal center of the hypothalamus increases cardiac output, triggers dilation of surface vessels, and increases sweating. Hyperthermia occurs when the natural ability of the body to maintain its core temperature is compromised, leading to an increase in body temperature.

Evaluation

Evaluation of an athlete suspected of heat-related illness lies in early recognition of presenting symptoms and signs. Symptom recognition is most important, because many hyperthermic endurance athletes remain asymptomatic despite increased core temperatures. Mild forms of illness include heat rash (miliaria rubra), heat cramps, heat edema (painless swelling of limbs), or heat syncope, which is uncomfortable, but usually self-limiting. Heat exhaustion, or exhaustion in the heat, is associated with temperatures that may normal or slightly increased (37°C – 40°C), thirst, malaise, nausea, vomiting, headache, weakness, anxiety, dizziness, and an increased heart rate. Sweating may be present or absent, and skin may or may not feel warm to touch. Mental status is preserved. Heat stroke is the third leading cause of death in athletes. It is defined as a core temperature of at least 40°C, with central nervous system abnormalities, such as altered mental status, seizure, or unconsciousness. Vomiting, diarrhea, shortness of breath, increased heart rate, and multiorgan failure may occur, with mortality approaching 10%, when present with hypotension increasing to 33%. In the hyperthermic individual with an altered sensorium,
concern of heat stroke should not be dissuaded by a measurement value that may be lower than the diagnostic threshold of 40°C.²⁵

Treatment

Although there is scant evidence supporting treatments of mild to moderate heat-related illnesses, more treatments are anecdotal or extrapolated from more severe illnesses and confer minimal risk. Heat cramps, or exercise-associated muscle cramps, which are theorized to be caused by neuromuscular fatigue and altered control, are usually self-limited and rarely require hypertonic oral electrolyte solutions.³³ Heat edema is reversed by the Trendelenburg position or compression stockings. Heat syncope by definition is self-limiting. Heat exhaustion in mild forms usually resolves with removal of the athlete to a cool environment when possible, ceasing physical activity, and oral rehydration with isotonic fluids if hyperthermic, to optimize sweating and subsequent heat dispersion.³⁴ More severe heat exhaustion may require more aggressive cooling by evaporative and convective cooling: loosen clothing, spray or douse the patient with cold water to optimize the water vapor–skin interface, and maximize convection with fanning. Each athlete should be carefully assessed and fully recovered before returning to running.

The method and aggressiveness of cooling treatment depend on the type of heat-related illness. Regardless of the underlying cause, rapid reversal of the hyperthermia is critical, because the resulting morbidity is directly related to both the degree and duration of hyperthermia in heat stroke.³³–³⁹ All treatment is first directed to stabilization of the patient’s airway, breathing, and circulation before proceeding to more specific cooling therapy. Cold-water immersion therapy is the optimal and most rapid treatment for cooling heat stroke. It has been shown to provide twice the cooling rate of evaporative cooling,³⁷ with faster cooling by colder water.⁴⁰ Immersion takes advantage of the high thermal conductivity of water and resulting high thermal gradient between cold water and skin, with a resulting greater capacity for heat transfer. Cold-water immersion may be limited to a naturally occurring body of water (eg, lake, pond, or river). The person with heat stroke should have clothes and equipment removed, and their trunk and extremities should be submerged, ensuring protection against currents, aspiration, or drowning. If no cold water source is available, dousing with cold water is an acceptable alternative conductive cooling method. If immersion is unavailable, combined treatments of evaporative and convective cooling should be initiated, and the patient with heat stroke should be rapidly transported to a medical facility. In transferring care to emergency medical services, it is important to continue cooling the patient by the best available means en route to the receiving hospital.

HYDRATION AND EAH

Proper hydration management plays a crucial role in successfully completing an ultramarathon race. Studies suggest that athletes can lose up to 2 L per hour in sweat loss and body weight loss of 8% or greater without significant symptoms or consequences.²⁷,³⁴,⁴¹,⁴² Some athletes experience a variety of symptoms relating to dehydration, including fatigue, nausea, vomiting, confusion, and weakness. The clinical diagnosis of dehydration is problematic, because these symptoms can occur with other medical illnesses.⁴³ Most cases respond to oral rehydration. Intravenous hydration should be considered in athletes who are not able to tolerate oral fluid intake and have evidence of significant hypovolemia (eg, orthostatis or resting tachycardia). If available, on-site point-of-care testing, (ie, iStat, Abbott Lab, IL, USA) should be used to confirm no evidence of hyponatremia. If point-of-care testing is not available
or unfeasible (ie, remote locations with extreme temperatures outside the capabilities of the point-of-care equipment), the physician should proceed cautiously with rehydration, considering their clinical assessment of the cardiovascular/emergent needs of the athlete, and potential risk of exacerbating EAH.\textsuperscript{43–47}

**Epidemiology**

EAH is defined as a serum sodium concentration of less than 135 mmol/L that occurs during or up to 24 hours after prolonged exercise.\textsuperscript{6} Most runners who experience EAH are asymptomatic, although a smaller subset experience symptoms potentially leading to significant morbidity or even death. The incidence and prevalence of EAH vary, depending on the length of the running race. Studies of marathon runners have noted that 3\% to 28\% of runners experience some form of hyponatremia.\textsuperscript{43–45} Studies of ultramarathon runners vary from an incidence of 5\% to 50\% for continuous single-stage\textsuperscript{7,46} events to 1\% to 12\% for multistage events.\textsuperscript{47} Although most EAH is often an asymptomatic biochemical diagnosis, there have been 12 documented fatalities.\textsuperscript{45} Therefore, awareness in prevention and treatment of EAH is an important part of care for the ultramarathon athlete.

**Pathophysiology**

The pathologic mechanism of hyponatremia is mostly a result of fluid overload or impaired urinary water excretion resulting from inappropriate secretion of arginine vasopressin (AVP).\textsuperscript{6,45,48} The primary cause of hyponatremia in the marathon runner is overhydration from excessive hydration.\textsuperscript{42,45} Increased consumption of hypotonic solutions along the course of the race because of ease of access can lead to intravascular overload, resulting in lower sodium levels. Several studies of marathon runners have found a strong association between higher incidences of EAH and drinking volumes greater than 3 to 3.5 L during a marathon.\textsuperscript{41,42,45} Other risk factors for marathon runners include slower race pace (>4 hours), female sex, and low body weight.\textsuperscript{6,41} The other major cause of EAH is inappropriate AVP secretion. In these cases, there is a failure to suppress AVP, which results in an inability of the kidneys to excrete the water load. This situation leads to an inability to produce diluted urine and resulting serum hyponatremia. Factors contributing to inappropriate AVP secretion include the response of the body to nonosmotic stimuli (prolonged exercise, stress, hypovolemia, fatigue, pain, and sleep deprivation), excessive sweating, or inadequate sodium supplementation.\textsuperscript{5,7,41,42,49} A recent study of continuous single-stage ultramarathoners with EAH\textsuperscript{7} noted that only 23.8\% were classified as overhydrated, whereas 35.6\% were dehydrated (weight change <\textsuperscript{\textminus}3\%). The study noted a weak association between postrace sodium levels and change in body weight, with hyponatremia more common in runners with increased weight loss. In contrast, a recent study of dysnatremia in multistage ultramarathon runners\textsuperscript{47} noted that hypernatremia (52.3\%, rate of 0.15 per person-stage) was more common than hyponatremia (14.8\%, rate of 0.03 per person-stage). In this study, runners became more dehydrated over the course of the race (22.5\%–53.5\%) and less overhydrated (44.9\%–17.2\%). The findings highlight the difference in dysnatremia and hydration status in marathon runners, continuous single-stage ultramarathon runners, and multistage ultramarathon runners. Each population needs to be treated in an appropriate manner, taking into account the differences noted earlier.

**Evaluation**

Recognizing the most common clinical symptoms of EAH is essential in avoiding more significant illness. Initial symptoms may include nausea, vomiting, sensation
of bloating, puffiness, and headaches. All event support staff should be educated on EAH and instructed regarding appropriate treatment. The symptoms should not be attributed to athlete fatigue and should prompt a thorough medical evaluation. As symptoms progress, athletes experience altered mental status, including confusion, disorientation, agitation, and delirium. Severe symptoms can lead to respiratory compromise, seizure, and death. Cases of suspected EAH or athletes undergoing intravenous rehydration should ideally have serum sodium levels measured by on-site point-of-care testing (ie, iStat) if available. As noted earlier, if point-of-care testing is not available or unfeasible (ie, remote locations with extreme temperatures outside the capabilities of the point-of-care equipment), the physicians should proceed cautiously with rehydration, considering their clinical assessment of the cardiovascular/emergent needs of the athlete and potential risk of exacerbating EAH.

Treatment

Treatment of EAH depends on the clinical symptoms, as well as confirmation of serum sodium levels. In cases of EAH confirmed by blood analysis with absence of altered mental status, initial treatment should include an avoidance of hypotonic fluids. Milder forms of EAH can be treated successfully with fluid restriction, close observation, and natural diuresis. If possible, small amounts of salty food or oral hypertonic solutions (concentrated broth) are appropriate. For patients who cannot tolerate oral intake and present with neurologic compromise, isotonic fluids should be avoided, and athletes can be given 100 mL of 3% NaCl over 10 minutes × 2, with expedited transfer to a medical facility. In cases in which blood analysis confirmation is not available, caution should be exercised, because the symptoms and signs may represent dehydration and hypovolemia, as opposed to hyponatremia.50 As noted earlier, the incidence of hyponatremia in continuous single-stage ultramarathon races is 31% to 50%, so treatment with fluid restriction or hypertonic saline solution is more likely to help. However, in multistage ultramarathons, greater caution is warranted, because athletes are more likely to be hypernatremic and dehydrated over the course of the race. In these races, cavalier use of fluid restriction or hypertonic saline solution may be more harmful than helpful.

Prevention strategies for hyponatremia focus on education of the ultramarathon runner. The primary strategy should be an avoidance of overhydration by adequate fluid consumption. Fluid ingestion based on the sensation of thirst during a race seems to be a safe relatively effective method for avoiding the extremes of overhydration and dehydration. The American College of Sports Medicine position statement on fluid replacement recommends adequate fluid intake to prevent greater than 2% of body weight loss from dehydration and excessive changes in electrolyte balance based on an individualized program.52 Studies have suggested that most ultramarathon athletes should avoid weight gain and can afford a loss of 2% to 3% of their body weight without significantly affecting running performance. Athletes may consider measuring their hourly sweat rate and fluid consumption during training (ideally, similar to race environment) in preparation for a race. However, weight loss is not a reliable measure for excluding the diagnosis of EAH.6,50 Food and hydration should be planned properly during the training schedule and should not significantly change during the course of the race. In multistage ultramarathon races, runners should be alerted that medical illnesses tend to occur during the first stage of the race, so fluid management should be minimally adjusted on the first day. Although many athletes use sodium supplementation, the exact role and impact of supplementation in preventing hyponatremia are not clear. All athletes should be educated
regarding the typical symptoms of hyponatremia and need to seek medical evaluation, as appropriate.

FOOT CARE

Friction blisters are arguably the most common medical problem encountered in any endurance race. Blister rates vary by distance, ranging from 0.2% to 39% for marathons, 32% to 45% for multistage adventure events, and up to 70% of all medical visits in multiday ultramarathons. For some, a foot blister may be considered merely a training nuisance; for others, it may an unavoidable injury that can ruin a run (Fig. 2), necessitate dropping out of an event, or even progress to cellulitis or sepsis, highlighting the importance of a thorough evaluation and optimal treatment. Blisters and foot issues remain the most often encountered injury in the endurance athlete.

Evaluation

Understanding the mechanism of a friction blister injury can assist in a thorough evaluation. The main cause is the repeated action of skin rubbing against another surface. As the external contact of either sock or footwear moves across the skin, the frictional force (Ff) opposes this movement. When horizontal shear forces overcome this resistance, repeated sliding at a friction point causes exfoliation of the stratum corneum and erythema in and around this zone. This injury is experienced initially as a sensation of heat: the hot spot. Continued friction on a hot spot causes epidermal cells in the stratum spinosum to delaminate and split, leading to fluid accumulation and blister formation. The intact superficial cells of the stratum corneum and stratum granulosum form the roof of the blister. A deeper injury involving the dermal plexus presents as a blood-filled blister, and an infected blister may contain cloudy fluid or surrounding erythema. Subungual hematomas present as a collection of blood underneath the nail bed or blister underneath the cuticle. The cuticle may be fluctuant with a large swollen vesicle, and the nail is often elevated, with dark blood underneath it. Blister evaluation is centered on appreciating the stage of the friction injury, which ranges from a hot spot to a torn-open or unroofed blister, and then, customizing the treatment to minimize friction to prevent reoccurrence or progression of injury.

Hot Spot Treatment

When treating a hot spot, the key to prevent blister formation involves decreasing shear forces on the skin. There are a few studies examining efficacy of various

Fig. 2. Posterior heel blister during a multistage 250-km ultramarathon race.
products such as powders, antiperspirants, lubricants, or tapes. The concept of a friction prevention layer at a high friction spot is to have a layer over the skin such that the resulting shear occurs between the barrier and the footwear, not the footwear and the skin. The shelves of drugstores and running stores are stocked with products. Ideally, taping products should be thin, easy to apply, adhere well, and provide limited seams, which may themselves be friction points. Paper tape is anecdotally useful, and although not ideal in overly wet conditions, because of its low cost, ease of use, and silky feel, it is our first-line product to treat a hot spot to prevent blister formation.

Antiperspirants and powders have been proposed as other measures to decrease the amount of moisture at the foot-sock interface. The largest antiperspirant trial found antiperspirant to be effective in prevention of blisters, but its high incidence in skin irritation (57% vs 6% of controls) likely limits its usefulness to those suffering from hyperhidrosis. Despite widespread marketing of powder compounds, there is no published scientific evidence to suggest that these products prevent foot blisters. Lubricants ostensibly prevent blister formation by decreasing friction at the foot–contact material interface. Several studies have shown that after applying lubricating substances to skin, there is an initial decrease of the coefficient of friction, but that within an hour, it returns to baseline with a subsequent increase in friction 35% higher than baseline over the next 4 to 6 hours. The studies suggest that with prolonged exercise, the use of lubricants might contribute to blister formation, so if used, they need to be reapplied frequently.

Treating a blister as soon as possible improves outcome, reduces pain, and minimizes complications from either subsequent tissue damage or infection. In the early stages of blister formation, the presence of a sensation of warmth from the hot spot is a warning sign. Prompt attention and rapid treatment can stop the abrasive process to prevent progressive blister formation. Proper blister care is not complicated, yet, it may be time intensive, depending on the extent of damage to the feet. Individuals should become familiar with techniques before heading outside and facing a blister predicament. Our medical experiences with ultraendurance races have shown that implementation of mandatory personal foot care kits for competitors and the expectation of self-care take a huge burden off the medical team.

**Blister Treatment**

Any tape used for blister treatment should be applied as smoothly as possible. The tape ideally acts as a second layer of skin, so rubbing acts on the tape, not on the underlying skin. Any folds or wrinkles in the tape should be avoided, because they may lead to high pressure and friction areas. Cutting the tape corners to round them and avoiding dog-ears helps in avoiding further blisters. All tape should be cut long enough to extend well beyond the border of the blister and any blister pads underneath the tape. Constriction through overlapping of tape and circumferential wrapping of the feet should be avoided, because it may lead to venous congestion and subsequent swelling.

Before taping, the skin should be clean of dirt and grit and as dry as possible, which enhances natural adhesion of tape. Using an adhesive substance, such as benzoine (liquid, squares, or spray), should be considered, to ensure security of the applied dressing. As a rule, blister tape should not be removed unless it is peeling off or there is increasing discomfort at the tape site. Ideally, soaking the bandages before removal loosens adhesion and minimize chances of ripping the roof off of intact blisters.

The pain from a blister is caused by pressure on an incompressible fluid between skin layers. As abrasion and pressure build, there is further pain and separation of skin layers and increasing potential for rupturing the blister, which leaves exposed
raw and sensitive skin. The best protection for a blister is its own roof, so efforts should be taken to maintain this natural skin protection. If the skin is not intact, it should be removed before dressing (Fig. 3A). Small friction blisters that are not causing significant discomfort can be left intact. If the blister is punctured with a needle and drained, it often refills within a few hours. If a large hole is made that allows continuous fluid drainage, there is the risk of losing integrity of the blister, and having the blister’s roof tear off, leaving a large damaged area. Our recommendation is to use a safety pin or similar-sized needle to create an optimum-sized hole.

Prepare the blister skin and safety pin with an alcohol pad. Puncture the blister with the prepared pin at a distal point, allowing natural foot pressure to continually squeeze out fluid. If more drainage is required, use several small holes rather than 1 large hole, limiting risk of deroofing the blister. Gently blot out the expressed fluid, cover the flattened blister with paper tape, which is cut to overlap the edge of the blister. This important step protects the roof of the blister when the overlying tape is removed. If the roof of the blister is ripped open, trim off the devitalized skin, apply a hydrocolloid layer of a Spenco 2nd Skin pad over the exposed base, and finish as described earlier (see Fig. 3B). Cover paper tape with a benzoin-type adhesive and allow it to become tacky (see Fig. 3C). As a final layer, apply shaped adhesive tape (ie, Elastikon) over the paper-taped blister (see Fig. 3D). Blister that recur under intact tape can be drained with a prepared safety pin through the tape.

Subungual hematomas are relieved by trephination of the nail bed, a necessary step, because the collection of fluid leads to pressure, which makes this condition painful. It is common to acquire a subungual hematoma on downhill hikes or runs

Fig. 3. (A) Open blister at the metatarsal region. (B) Placement of 2nd Skin over the blister after removal of the open cover. (C) Placement of paper tape over the blister. (D) Placement of external bandage.
when the toes are repetitively jammed into the toe box. Subungual hematomas are easily treated. An 18-gauge hypodermic needle is held perpendicular to the proximal nail bed over the area of greatest fullness (Fig. 4A). With gentle downward pressure, rotate the needle between the thumb and first finger; as it is twirled back and forth, it easily drills into and through the nail, releasing the hematoma. The release of blood under pressure through the 18-gauge hole may be dramatic, causing it to squirt, so appropriate universal precautions should be used. The 18-gauge needle should be recapped and may need to be reused for the same patient, because these blisters have a tendency to recur. Treating a cuticle blister is a simple treatment, as described earlier (see Fig. 4B). As with all toe blisters, only paper tape should be used to wrap the injured digit, because any other tape with texture has the potential to rub on neighboring toes and cause adjacent intertriginous blisters.

There is no one correct way to care for feet. For every technique and product mentioned, there are several different options. We recommend avoiding draining of blood-filled blisters, because these are a potential route for bacteria to enter the wound and bloodstream, which can lead to cellulitis or sepsis. Blood blisters should be left intact, unless they are large, fluctuant, extremely painful, or at risk for spontaneous rupture. To drain, use a providone-iodine preparation, clean gloves, and instruments with the techniques described earlier. Likewise, blisters deep to a callus should not be drained. These blisters are painful to access, yield little blister fluid, and quickly refill after drainage. A blister with murky hazy fluid or pus may be infected. The blister should be opened (deroofed), irrigated with providone-iodine, and then, antiseptic or antibiotic ointment should be applied to the cavity before being covered with the open blister technique. If the individual begins to show signs of worsening surrounding erythema, streaking, or systemic symptoms (eg, chills, fevers, nausea, or generalized weakness), definitive care, including oral or intravenous antibiotics, should be used to avoid sepsis. The goals of blister treatment are to optimize comfort for continued activity, minimize progression of disease, assist with epidermal recovery, and prevent further blister enlargement when resting and staying off the feet is not an option.

SUMMARY

Physicians and athletes participating in ultramarathons need to be prepared for a variety of injuries and illnesses that may occur during a race. Most musculoskeletal and skin-related issues are minor and can be treated successfully during the course of a race. Common medical illnesses, including environmental illnesses and EAH, require prompt assessment and appropriate treatment in hopes of avoiding significant
morbidity and mortality. Continued research should focus on preventative and optimal treatment strategies in hopes of preventing long-term complications in this unique athletic population.

REFERENCES


