CASE STUDIES

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Heat Cramps During Tennis: A Case Report

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A 17-year-old, nationally ranked, male tennis player (AH) had been experiencing heat cramps during tennis match play. His medical history and previous physical exams were unremarkable, and his in-office blood chemistry profiles were normal. On-court evaluation and an analysis of a 3-day dietary record revealed that AH’s sweat rate was extensive (2.5 L · hr⁻¹) and that his potential daily on-court sweat sodium losses (89.8 mmol · hr of play⁻¹) could readily exceed his average daily intake of sodium (87.0–174.0 mmol · day⁻¹). The combined effects of excessive and repeated fluid and sodium losses likely predisposed AH to heat cramps during play. AH was ultimately able to eliminate heat cramps during competition and training by increasing his daily dietary intake of sodium.

Key Words: sodium, sweat rate, urine specific gravity, potassium

Excessive and repeated fluid and sodium (Na⁺) losses have been frequently implicated in the development of heat-related muscle cramps (heat cramps) during prolonged exercise (2, 3, 8, 10–13, 15, 22, 23, 25). A contracted extracellular fluid compartment, as a result of an acute body water deficit, may cause changes in the ionic concentrations of the extracellular space as well as mechanical deformation of motor nerve terminals during muscle shortening (12, 15). These conditions, along with the release of chemical substances (e.g., acetylcholine, potassium ions, and metabolites) during muscle activity, may initiate hyperexcitability in selected motor nerve terminals. This could prompt what might appear to be spontaneous muscle cramps following just subtle volitional contractions of the same muscles (12, 15). Further, unless water and Na⁺ are replaced, rehydration will be incomplete and the extracellular fluid compartment will remain contracted (16).

Several other mineral deficiencies have also been implicated as potential etiologic factors in the development of muscle cramps during prolonged, vigorous exercise. These include hypocalcemia (8, 13, 15, 23), hypomagnesemia (8, 13–15, 17–19, 23, 26), and hypokalemia (3, 13, 15, 23). In 1989, professional tennis player Michael Chang created a seemingly indelible image in the minds of many tennis coaches and players as he, on international television, ate bananas in an

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62
attempt to avert cramps in his thighs during his fourth-round match at the French Open. Today, increased dietary intake of potassium (K') continues to be a popular choice of tennis players for preventing heat cramps (1).

Methods

Subject and Case History

A 17-year-old, nationally ranked, male tennis player (AH) had been experiencing heat cramps (primarily in the quadriceps, hamstring, and calf muscles) for 2 years during tennis match play, particularly during intense, long tournament matches. Although the incidence of cramps was higher in hot weather, they occurred periodically in more temperate climates and sometimes during indoor matches and other activities (e.g., high school basketball games). AH’s height was 193.0 cm and his weight was 81.4 kg. He was very fit and had a weekly schedule that included approximately 18 hr of tennis competition and practice and 3 hr of supplemental training (e.g., running, weight lifting, etc.). When he was not traveling to play in tennis tournaments, AH resided in a northern section of the 48 contiguous United States.

During the summer months of 1994, the incidence of AH’s heat cramps had increased considerably. During the same period, AH was examined by several trainers and a number of internal medicine and sports medicine physicians. His medical history and physical exams were unremarkable. There was a family history of high blood pressure, although AH’s blood pressure was normal. In addition, AH’s father had experienced several similar incidents of heat cramps during long-duration, recreational exercise (e.g., cycling). Three separate, in-office blood chemistry profiles were completed for AH. All results were similar and unremarkable, except for the usual elevated serum enzyme activity (CK, 1,294 U · L⁻¹; LDH, 278 U · L⁻¹) commonly seen in athletes engaged in training and competition (20, 24). Results of concomitant urinalyses were also normal. AH was not taking any medications at the times of his cramping experiences or subsequent medical evaluations.

AH’s resting serum [Na⁺] was normal (145 mmol · L⁻¹). Although AH’s resting serum levels of K⁺ (4.6 mmol · L⁻¹), calcium (Ca²⁺, 2.3 mmol · L⁻¹), and magnesium (Mg²⁺, 1.0 mmol · L⁻¹) were also within normal ranges, several physicians prescribed Ca²⁺, Mg²⁺, and K⁺ supplements and instructed AH to drink a carbohydrate–electrolyte drink (Gatorade®) during play. Further, athletic trainers at several tournaments instructed AH to drink a lot of water and to eat bananas. None of these “remedies” reduced the frequency or severity of heat cramps during play. AH’s personal tennis coach contacted the United States Tennis Association (USTA) Player Development Office in Key Biscayne, FL, and an on-court evaluation was arranged.

On-Court Evaluation

In August 1994, AH traveled to Key Biscayne, FL, for the determination of his prematch hydration status, on-court sweat electrolyte (Na⁺ and K⁺) and fluid losses during competition in the heat, and the amount and pattern of his fluid intake during play. In addition, a 3-day, preevaluation dietary intake was recorded and evaluated (The Food Processor® II [3.04], ESHA Research, Salem, OR).
AH arrived in Key Biscayne, via a commercial airline flight, approximately 24 hr prior to the evaluation. The next day, upon arrival at the test site, AH provided a urine sample for determination of urine specific gravity. AH was subsequently weighed nude to within ±0.1 lb (A & D Model UC-300, A & D Engineering, Inc., Milpitas, CA), and then he dressed for playing tennis (cotton t-shirt, shorts, etc.). AH promptly proceeded to the court where he began his warm-up for a singles tennis match against a competitively matched male opponent. The match was played at 1400 hr, using regular scoring and odd-game changeovers, on a hard-surface, stadium court. Six new tennis balls were used, and the players retrieved their own balls. In addition, a standard 10-min warm-up preceded the start of the match.

Ambient air temperature (31.6 °C) and relative humidity (62%) were measured with a thermohygrometer (Tri-Sense® Model 37000-00, Cole-Parmer Instrument Company, Niles, IL) at approximately 1.0 m above the ground surface (in the shade). The visually estimated cloud cover was 50%.

During the beginning part of the match, AH drank only water ad libitum. After approximately 20 min of play, the match was interrupted during a convenient changeover, for less than 2 min, so that AH’s nondominant forearm could be prepared for sweat sample collection using a previously described method (5). After the sweat collection (approximately 25 min) was completed, AH consumed Gatorade® ad libitum for the remainder of the match. Immediately after play ended, AH was again weighed nude. Fluid intake was monitored and sweat rate was calculated, as previously described (5).

Urine specific gravity was measured by a handheld refractometer (Spartan Refractometer, Model A 300 CL, Japan). Sweat [Na⁺] and [K⁺] were measured by selective, ion-specific electrodes (Beckman Synchron CX-3 Clinical System, Beckman Instruments Inc., Brea, CA).

**Results**

Match play lasted 1.8 hr. Exercise intensity (i.e., percentage of maximal heart rate reserve) during play was assumed to be similar to previous findings (6). Prematch urine specific gravity was 1.022.

AH’s sweat rate was 2.5 L·hr⁻¹. He consumed 3.3 L (1.8 L·hr⁻¹) of fluid during play, which equaled about 0.2 L (7–8 oz) during each changeover, and had a body weight deficit of 1.4% by the end of play. AH’s sweat [Na⁺] and [K⁺] were 35.9 and 5.4 mmol·L⁻¹, respectively. These values translated to estimated Na⁺ and K⁺ loss rates of 89.8 mmol (just over 2 g) and 13.5 mmol (about 0.5 g) per hour of play, respectively. AH’s 3-day dietary record indicated a total energy intake of approximately 3,000–4,000 kcal·day⁻¹, with an average Na⁺ consumption of approximately 87.0–174.0 mmol·day⁻¹ (2–4 g of Na⁺ per day). Importantly, AH indicated that this amount of daily Na⁺ intake was likely considerably higher than it had regularly been in the past.

**Discussion**

AH’s sweat rate and concomitant rate of sweat Na⁺ loss were extensive. These factors, in combination with a relatively conservative daily dietary intake of Na⁺, likely predisposed AH to developing heat cramps after several matches or days of competition or training. However, a precise monitoring of AH’s body water balance,
Heat Cramps

Na⁺ turnover (including total urine-electrolyte losses), and serum [Na⁺] changes, over several days of competition, would have provided a more complete, and perhaps more indicative, profile of AH’s fluid-electrolyte status during tournament play.

As a representative indication of his typical hydration status prior to playing a match, AH’s prematch urine specific gravity was high (i.e., it did not reflect a well-hydrated condition, particularly since fluid loss during play was expected to be extensive). A value closer to or below 1.015 would have been more desirable. However, AH’s fluid consumption rate during play appeared adequate and would likely keep him from incurring additional large body water deficits during most matches.

AH’s sweat electrolyte concentrations were clinically normal; however, his sweat [Na⁺] was somewhat higher than that previously measured (22.4 mmol·L⁻¹) in acclimatized male tennis players (5). Even though AH did not live year-round in a warm climate, he should have been acclimatized to the heat, given that he was evaluated during the third week of August and had been regularly playing and training in warm to hot conditions for several months. AH indicated that he was on a low-sodium diet as a precautionary measure, in response to his family history of high blood pressure. One can readily see how AH’s sweat Na⁺ loss during a long match could easily exceed his daily dietary intake of Na⁺. As a result, a progressive Na⁺ deficit could develop, which might be evidenced by a daily progressive decrease in morning preplay serum [Na⁺], as AH plays a lot of high-intensity tennis on successive days in the heat (5). With an extracellular fluid compartment that would, as a result of a Na⁺ deficit, remain contracted (16), AH would likely be predisposed to heat cramps even prior to starting his next match. Notably, AH’s postmatch concentration of serum Na⁺ would probably be normal or slightly elevated, owing to hypotonic sweat secretion, renal Na⁺ conservation, and a certain degree of osmotically driven defense of plasma volume. Therefore, a during-play or postplay serum [Na⁺] measurement may not be a sensitive indicator of ionic or other changes in the extracellular space of the muscles and, thus, may not be a valid predictor of an individual’s concomitant susceptibility to heat cramps. Further, it is not surprising that AH’s serum [Na⁺] was normal during his previous office examinations, since AH had sufficient opportunity to reestablish his fluid and Na⁺ balance prior to each blood sampling. However, as Lombardo stated (3), cramping patients whose parents have high blood pressure often do not have enough salt in their diets.

Recommendations

I recommended that AH increase his daily dietary intake of Na⁺ to at least 261–348 mmol (6,000–8,000 mg) when he competes or trains in the heat. This amount is somewhat higher than the average daily Na⁺ intake, in the United States, of 217.5 mmol (5,000 mg) (9). I suggested that AH begin his matches better hydrated, by increasing appropriate fluid intake throughout the day, especially during and following practice and prematch warm-ups, and by continuing to avoid consumption of fluids containing caffeine or alcohol. I also recommended that AH monitor his body weight changes, as a consequence of practice or play, so that he would be able to adequately replace lost fluid and better maintain a stable body water balance from day to day. And further, I recommended that AH increase his energy intake to 5,000–6,000 kcal·day⁻¹, with appropriate pre-
and postmatch carbohydrate intake (7, 21), during periods of moderate- to high-volume competition or training.

Importantly, the recommended increase in Na\(^+\) intake served two purposes: (a) It helped to replace a significant portion of the exchangeable Na\(^+\) pool that was lost via sweat, and (b) it played an important role in restoring body water content after play (16).

Although I recommended that AH continue to drink Gatorade\(^\circledR\) during play (about 8 oz, or 8 swallows, per changeover), I also suggested that he carry a small packet containing about 0.5 tsp of salt (NaCl) in his equipment bag that he brings to the court. If AH sensed that heat cramps were about to develop (as indicated by the subtle twitches that he usually felt in his legs about 30 min prior to full-blown heat cramps), then he was to add the NaCl to approximately 0.5 L of water and drink all of it over the current and subsequent changeover periods (see Follow-Up Reports and Further Recommendations, below). The [Na\(^+\)] of this mixture is 102.6 mmol L\(^{-1}\) (about 5 times the [Na\(^+\)] of Gatorade\(^\circledR\)).

(Reference 4 provides a more complete review of related factors and recommendations for managing fluid–electrolyte balance while playing tennis in a hot environment.)

**Follow-Up Reports and Further Recommendations**

AH incorporated the above recommendations into his daily routine for managing water and Na\(^+\) balance, with particular emphasis and effort during tournaments in hot environments. In the subsequent 9 months, AH experienced no heat cramps (or subtle twitches) during competition or training. Notably, during this time, he was able to play long matches in tournaments such as the Orange Bowl, the Fiesta Bowl, and the Indoor Nationals without an incident.

In May 1995, AH traveled to southern Florida to train and compete during the summer months. AH was not acclimatized to the heat (about 32.2 °C) and humidity that he encountered. During a practice match on the first day of training (AH’s second day in Florida), AH began to feel subtle twitches in his legs that were characteristic of the onset of heat cramps. Anticipating that he had about 30 min or less before full-blown heat cramps developed, AH emptied his “emergency” packet of NaCl (0.5 tsp) into a 16-oz plastic bottle of water, mixed the solution well, and promptly consumed the entire 16 oz. AH was able to continue playing and training, without heat cramps or twitches, for several more hours.

Three weeks later, AH was playing in a tournament in Florida under very hot (>35 °C) and humid conditions. On the first day of the tournament, AH completed a 2.5-hr match in the morning. In the middle of his second match, during the afternoon of the same day, AH succumbed to heat cramps and defaulted from the tournament.

Subsequently, I recommended to AH that he, during tournaments in hot and humid conditions, drink a concentrated salt solution (20–24 oz of water with 0.5 tsp of NaCl) in the evening and again after breakfast. Past experience showed that AH tolerated such a drink quite well, and it was an easy way to add an additional 102.6 mmol (2,358 mg) of Na\(^+\) (beyond the initial recommendation of 6–8 g), as well as more fluid, to AH’s daily diet. AH chose to add the 0.5 tsp of NaCl to 8–10 oz of V\(_8\)\(^\circledR\) vegetable juice (which inherently contains 620 mg of Na\(^+\) per 8 oz) instead of water. AH consumed one of these vegetable juice mixtures each
morning and night, while he continued his training and competition in south Florida. During the subsequent 6 weeks, AH experienced no further incidents of heat cramps. Notably, this period included many days when AH was training and competing for 6 hr in hot (>35 °C) and high-humidity conditions.

Unfortunately, no data were collected during the events and periods described in this section. Consequently, a precise (or even estimated) indication of AH's daily Na⁺ status or turnover, during those times, could not be obtained.

Conclusions

Exercise-related muscle cramps can be often attributed to fatigue, a lack of conditioning, or mineral deficiencies (e.g., Mg²⁺ or Ca²⁺) (3, 8, 13–15, 17–19, 23, 26). And if an athlete has a mineral deficiency, appropriate mineral supplements can help to reduce the incidence of associated muscle cramps (14, 15, 23). Yet sometimes, trainers and physicians may have to ultimately attempt to clinically distinguish heat cramps from other muscle cramps through more specific follow-up evaluations of their afflicted athletes. The characteristics, distinguishing features, and potential etiologies of heat cramps have been well outlined by Hubbard and Armstrong (11).

These findings support the viewpoint that heat cramps can be related to large fluid losses and a concomitant Na⁺ deficit. Further, these findings suggest that heat cramps, for some tennis players, can be readily prevented by an appropriate increase in daily water and Na⁺ intake and that heat cramps may be averted during play by drinking a concentrated salt solution. Importantly, such a drink should be experimented with during noncompetition situations to familiarize the player with its taste and to determine a tolerable [Na⁺] range. In addition, some players may have to be very diligent about water and Na⁺ replacement during competition and training in very hot and humid conditions, particularly when playing multiple, long matches on successive days. Last, these findings and suggestions are consistent with recent empirical data gathered from working with several other tennis players, professional and amateur, who have similarly resolved their on-court heat cramp problems by increasing their daily Na⁺ consumption (Bergeron, unpublished observations).

References


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