Hyponatremia in a Cold Weather Ultraendurance Race

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Abstract
We assessed the incidence and etiology of hyponatremia in the 100-mile (161 km) Iditasport ultramarathon. Subjects (8 cyclists, 8 runners) were weighed and serum sodium was measured pre- and post-race. Race diets were analyzed to determine fluid and sodium consumption. Subjects were split by post-race serum sodium concentration into hyponatremic and normonatremic groups for statistical analyses. Seven of 16 subjects (44%) were hyponatremic. The hyponatremic group exhibited a significant decrease in serum sodium concentration (137.0 to 132.9 mmol/L, and the normonatremic group experienced a significant decrease in weight (82.1 to 80.2 kg) pre- to post-race. The hypornatremic group drank more fluid per hour (0.5 versus 0.4 L/h) and consumed less sodium per hour (235 versus 298 mg/h) compared to the normonatremic group. In conclusion, hyponatremia is common in an ultraendurance race held in the extreme cold, and may be caused by excessive fluid consumption and/or inadequate sodium intake.

Keywords
hyponatremia, ultraendurance, race diet, sodium consumption, ultramarathon

Disciplines
Other Medicine and Health Sciences | Sports Sciences

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Hyponatremia in a Cold Weather Ultraendurance Race

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ABSTRACT

We assessed the incidence and etiology of hyponatremia in the 100-mile (161 km) Iditarod ultramarathon. Subjects (8 cyclists, 8 runners) were weighed and serum sodium was measured pre- and post-race. Race diets were analyzed to determine fluid and sodium consumption. Subjects were split by post-race serum sodium concentration into hyponatremic and normonatremic groups for statistical analyses. Seven of 16 subjects (44%) were hyponatremic. The hyponatremic group exhibited a significant decrease in serum sodium concentration (137.0 ± 3.4 vs. 132.9 ± 3.4 mmoL/L) and the normonatremic group experienced a significant decrease in weight (82.1 ± 5.0 vs. 80.2 ± 5.0 kg) pre- to post-race. The hyponatremic group drank more fluid per hour (8.5 ± 1.5 L/h) and consumed less sodium per hour (235 ± 80 vs. 298 ± 99 mg/h) compared to the normonatremic group. In conclusion, hyponatremia is common in an ultraendurance race held in the extreme cold, and may be caused by excessive fluid consumption and/or inadequate sodium intake.

INTRODUCTION

Hyponatremia, defined as a serum sodium level below 135 mmoL/L, is a serious consequence of endurance events lasting longer than six hours (1). Possible signs and symptoms include light-headedness, nausea, vomiting, malaise, exhaustion, altered mental status, headache, seizures, and in extreme cases, death (2).

Figure 1 shows the two leading theories that attempt to explain the hyponatremia associated with prolonged exercise (1,3,4). One theory suggests that hyponatremia is caused by an increase in total body water, resulting from excessive fluid consumption or a failure to excrete excess volume. The second theory suggests that a decrease in sodium content resulting from excessive sodium loss in sweat or inadequate sodium intake causes hyponatremia. These two theories are not mutually exclusive. Any combination of the factors displayed in Figure 1 could lead to the development of hyponatremia.

Numerous papers have been published on the occurrence of hyponatremia in triathletes (2,5-12), ultramarathoners (13-17), marathons (18-20), Grand Canyon hikers (21-22), military recruits (23-24), and subjects in laboratory studies (25-26). All of these events took place in mild to hot environments, and the studies did not include an analysis of race diets to determine both fluid and sodium consumption.

In this study, we assessed the incidence of hyponatremia in the Iditarod, a 100-mile (161 km) ultraendurance race held in Alaska each February. Additionally, race diets were analyzed to determine racers' fluid and sodium consumption.

METHODS

The study was approved by the McDaniel College Institutional Review Board. All 122 entrants in the 2000 Iditarod Human Powered Ultra-Mara-
Figure 1. Possible causes of hyponatremia. Figure modified from Montain (1).
Table 3. Race diet; values expressed as mean ± SD.

<table>
<thead>
<tr>
<th></th>
<th>Hyponatraemic</th>
<th>Non-hyponatraemic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 7)</td>
<td>(n = 9)</td>
</tr>
<tr>
<td>Total kcal</td>
<td>5,481 ± 2,499</td>
<td>7,334 ± 5,322</td>
</tr>
<tr>
<td>Kcal/l</td>
<td>212 ± 64</td>
<td>252 ± 166</td>
</tr>
<tr>
<td>Percent carbohydrate (%)</td>
<td>60.0 ± 16.1</td>
<td>68.0 ± 14.4</td>
</tr>
<tr>
<td>Percent fat (%)</td>
<td>21.7 ± 16.4</td>
<td>22.4 ± 11.6</td>
</tr>
<tr>
<td>Percent protein (%)</td>
<td>9.1 ± 2.5</td>
<td>9.6 ± 4.1</td>
</tr>
<tr>
<td>Total Na+ (mg)</td>
<td>6,216 ± 3,299</td>
<td>8,558 ± 4,730</td>
</tr>
<tr>
<td>Na+/kg</td>
<td>235 ± 92</td>
<td>298 ± 144</td>
</tr>
<tr>
<td>Total fluid (L)</td>
<td>11.9 ± 5.7</td>
<td>12.2 ± 4.0</td>
</tr>
<tr>
<td>Fluid loss (L)</td>
<td>0.5 ± 0.2</td>
<td>0.4 ± 0.2</td>
</tr>
</tbody>
</table>

a higher percentage than has been reported for athletes participating in triathlons or ultramarathons held in mild to hot environments. Speedy (2) and Hiller (6) reported that hyponatraemia occurs in 18-27% of race participants, and others (7,8,16) have found that 9-36% of athletes who seek medical care are hyponatraemic. It is interesting to note that the hyponatraemic athletes were significantly lighter than the normonatraemic athletes, and that the two female subjects in the sociologically defined hyponatraemic group. These results confirm the findings of others (1,2,29) that women and smaller individuals are more at risk for developing hyponatraemia because they need to consume less fluid than larger individuals to dilute serum sodium to hyponatraemic levels. All of the hyponatraemic and normonatraemic athletes in the present study were hydrated at least one of the signs and symptoms of hyponatraemia. This is not surprising since the signs and symptoms of the condition (light-headedness, nausea, vomiting, malaise, exhaustion, altered mental status, headache, and seizures) (2) are very nonspecific.

Figure 1 shows the possible causes of hyponatraemia, including an increase in total body water, resulting from excessive fluid consumption or a failure to excrete excess volume, and a decrease in sodium content, resulting from excessive sodium loss in sweat or inadequate sodium intake. The majority of reports (2,5,8,10,12,15,18,22-24) indicate that excessive fluid consumption is the most likely cause of hyponatraemia, although others have suggested that a failure to excrete excess volume (7, 17,30) or excessive sodium loss in sweat (6,19,21) may be contributing factors.

We speculate that the hyponatraemia seen in lidisport athletes is caused by fluid overload and/or inadequate sodium intake. The normonatraemic group demonstrated a statistically significant weight loss (-1.9 kg) during the race, compared to the hyponatraemic group that experienced a statistically insignificant weight loss (-0.8 kg). Others (9,11, 31) have reported that nonfluid weight loss during very prolonged exercise may account for as much as 2 kg of weight loss during the event. Sources of nonfluid weight loss include loss of fat, glycogen, and water stored with glycogen. The corollary of this observation is that athletes who drink sufficiently to maintain their weight during an ultramarathon event may in reality be overhydrated by 2 liters. This suggests that the hyponatraemic lidisport athletes may have had a fluid excess of 1.2 L, and that the normonatraemic athletes were essentially euhydrated.

Both the hyponatraemic and normonatraemic groups exhibited a decrease in hematocrit and an increase in plasma volume following the race, although these changes were more pronounced in the hyponatraemic group. These findings agree with the reports of others (5,9,10,23,25), and may be indicative of hypervolaemia (9,10).

Race dietary analysis revealed that the hyponatraemic group drank most of their fluid (0.5 L/h versus 0.4 L/h) and consumed less sodium per hour (235 mg/kg/h versus 298 mg/kg/h) than the normonatraemic group, although the differences were not statistically significant. The ACSM recommends that athletes drink 0.6-1.2 liters of fluid per hour during exercise, with the addition of 0.5-0.7 g of sodium per liter for exercise lasting more than one hour (32). Using these guidelines and an average finish time of 27.6 h, the athletes in this study should have consumed 16.6-33.1 L of fluid and 6.3-23.2 g of sodium. Both the hyponatraemic and the normonatraemic groups consumed less fluid than the ACSM recommendations. However, the ACSM guidelines were established based on research from much shorter events than ultramarathon competitions, and they are aimed at preventing heat injuries during events held in a hot environment. The guidelines may be inappropriate high for any ultramarathon competition (33,34), and most certainly are too high for an ultramarathon event held in Alaska in February. The hyponatraemic athletes (6.2 g sodium) consumed less than the recommended amount of sodium, but the normonatraemic athletes (8.6 g sodium) were within the ACSM guidelines, suggesting that inadequate sodium intake may have been a contributing factor in the development of hyponatraemia.

It seems unlikely that the other possible causes of hyponatraemia shown in Figure 1 could account for the hyponatraemia seen in lidisport athletes. A failure to excrete excess fluid could be caused by excessive ADH, but it is known that ADH secretion is decreased in the cold (33). Hypotherically, hyponatraemia can be caused by an excessive loss of sodium in sweat. There is little support for this theory in the literature for events occurring in hot environments (1), so it seems unreasonable to suggest that this is the cause of hyponatraemia in an event held in the extreme cold, where athletes are instructed at a mandatory pre-race meeting to avoid sweating in an attempt to prevent hypothermia and frostbite.

In conclusion, hyponatraemia occurred in 44% of the athletes competing in an ultramarathon event in the extreme cold. We speculate that the hyponatraemia was caused by excessive fluid consumption and/or inadequate sodium intake.

ACKNOWLEDGEMENT

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Author's Note: These data were collected at the 2000 Idiisport Human Powered Ultras-Marathon. In 2001, the name of the race was changed to the Sustina100.

REFERENCES


(continued on pg 62)
NEURILEMMOMA: AN UNUSUAL BENIGN TUMOR OF THE CERVIX

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Edward A. Barker, MD

CASE PRESENTATION

A 47-year-old para 2 woman came for a routine examination and Pap smear in November of 2000. She was still having regular but somewhat heavier menses, and had recently begun to experience some peri-menopausal symptoms. She had no intermenstrual bleeding, no postcoital bleeding and neither dysmenorrhea nor dyspareunia. She had no vaginal discharge and no gastro-intestinal complaints. Her only other gynecological complaint was an occasional feeling of heaviness in the pelvis with some urinary frequency and mild urgency. Her past history and family history was unremarkable.

She had never had an abnormal Pap smear and the previous normal smear was 3 years ago. She had a normal mammogram 3 years earlier. She was taking a low dose oral contraceptive pill.

The general physical examination was entirely negative. There were no skin lesions, no lymph nodes and no evidence of Von Recklinghouse's disease.

On gynecologic exam the external genitalia were normal and so was the vagina. The cervix had a most unusual appearance, with a \(2^\frac{1}{2}\) cm diameter round, sessile tumor on the posterior lip. This tumor was soft, with a patchy yellow red appearance and seemingly vascular. It did not involve the endocervical canal.

Atocolposcopy, the entire squamo-columnar junction could be seen. The anterior lip of the cervix had an extension but was otherwise normal. The tumor itself was ulcerated and did not bleed on contact. It did not have any aceto - white areas and other than being vascular did not show any abnormal vessels (figure 1-a and 1-b).

On bimanual examination the cervix was not tender. The tumor was soft without any induration of the surrounding areas. The uterus itself was normal in size, location and consistency. The adenexae felt normal and there was neither induration in the parametria, nor nodularity in the posterior fornix. A pap smear was taken and reported as normal.