ORIGINAL ARTICLE

Hyponatremia among Runners in the Boston Marathon

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ABSTRACT

BACKGROUND

Hyponatremia has emerged as an important cause of race-related death and life-threatening illness among marathon runners. We studied a cohort of marathon runners to estimate the incidence of hyponatremia and to identify the principal risk factors.

METHODS

Participants in the 2002 Boston Marathon were recruited one or two days before the race. Subjects completed a survey describing demographic information and training history. After the race, runners provided a blood sample and completed a questionnaire detailing their fluid consumption and urine output during the race. Prerace and postrace weights were recorded. Multivariate regression analyses were performed to identify risk factors associated with hyponatremia.

RESULTS

Of 766 runners enrolled, 488 runners (64 percent) provided a usable blood sample at the finish line. Thirteen percent had hyponatremia (a serum sodium concentration of 135 mmol per liter or less); 0.6 percent had critical hyponatremia (120 mmol per liter or less). On univariate analyses, hyponatremia was associated with substantial weight gain, consumption of more than 3 liters of fluids during the race, consumption of fluids every mile, a racing time of >4:00 hours, female sex, and low body-mass index. On multivariate analysis, hyponatremia was associated with weight gain (odds ratio, 4.2; 95 percent confidence interval, 2.2 to 8.2), a racing time of >4:00 hours (odds ratio for the comparison with a time of <3:30 hours, 7.4; 95 percent confidence interval, 2.9 to 23.1), and body-mass-index extremes.

CONCLUSIONS

Hyponatremia occurs in a substantial fraction of nonelite marathon runners and can be severe. Considerable weight gain while running, a long racing time, and body-mass-index extremes were associated with hyponatremia, whereas female sex, composition of fluids ingested, and use of nonsteroidal antiinflammatory drugs were not.

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As marathon running has surged in popularity during the past quarter-century,[1] reports have emerged of serious illness and death from hyponatremia,[2-8] as in the case of a 28-year-old woman who died after the 2002 Boston Marathon.[2] The incidence of hyponatremia among marathon runners is unknown, since previous studies have been small and limited to runners presenting for medical attention.[4,5,7,9-11]

Excessive fluid intake is believed to be the primary risk factor for hyponatremia, on the basis of observations of marathon runners who have collapsed[2-5,7,11,12] and studies of elite athletes.[13-17] However, other risk factors have also been suggested, including the composition of fluids consumed (e.g., plain water, rather than sports drinks that contain electrolytes), relatively low body-mass index, long racing time, lack of marathon experience, use of nonsteroidal antiinflammatory drugs (NSAIDs), and female sex.[4,5,9,18] We undertook the present study to estimate prospectively the incidence of hyponatremia among marathon runners and to identify the principal risk factors involved.

METHODS

STUDY POPULATION

Marathon runners were recruited prospectively at an exposition one or two days before the Boston Marathon, in April 2002. All registered participants 18 years of age or older were eligible, regardless of whether they registered for the marathon on the basis of a competitive qualifying time or on behalf of a charitable organization — a mechanism for which no previous marathon experience was required. Subjects were approached at random in an area adjacent to race registration and invited to participate. Written informed consent was obtained from all subjects. The study protocol was approved by the Committee on Clinical Investigation at Children's Hospital in Boston.

STUDY DESIGN

Before running the marathon, subjects completed a survey describing baseline demographic and training information, medical history, and anticipated hydration strategies for the race. At the finish line, runners provided a blood sample and completed a questionnaire detailing their fluid consumption and urine output during the race. Blood samples were centrifuged on site and frozen at −70°C until analyzed. With the use of a digital balance, the prerace and postrace weights were recorded for each runner.

OUTCOME MEASURES

The primary hypothesis of the study was that excessive consumption of hypotonic fluids is associated with hyponatremia in marathon runners. Hyponatremia was defined as a serum sodium concentration of 135 mmol per liter or less. Severe hyponatremia and critical hyponatremia were defined as serum sodium concentrations of 130 and 120 mmol per liter or less, respectively. Independent variables analyzed for association with hyponatremia included weight change during the race and self-reported fluid intake including volume, frequency, and type. Both water and a sports drink containing electrolytes were offered at each milepost, and runners were asked to estimate the proportion of their intake from each. Other predictors that we considered included sex (a dichotomous variable), body-mass index (the weight in kilograms divided by the square of the height in meters), training pace, number of previous marathons (dichotomized at a median of five), duration of the marathon in hours and minutes, use or nonuse of NSAIDs in the past week (a dichotomous variable), age, and race (a dichotomous variable [white vs. nonwhite]). Race was self-reported by the runners.

STATISTICAL ANALYSIS

Descriptive statistics were used to estimate the incidence of hyponatremia and to characterize the demographic information supplied by the runners. Unless otherwise specified, t-tests and Fisher's exact test were used to identify univariate predictors associated with hyponatremia, at a level of statistical significance of $P \leq 0.05$. Logistic regression (SAS software, version 9.0) and generalized additive models[19] (S-Plus software, version 6.1 for Windows) were used in the multivariate analysis to identify independent predictors of hyponatremia.

RESULTS

Table 1 summarizes the baseline demographic and training characteristics of the study population. Of 766 runners enrolled, 511 (67 percent) reported to the finish-line research station. Of these, 489 provided a blood sample (constraints such as plane flights precluded 22 runners from providing a sample). One sample was considered of insufficient quantity, leaving a total of 488 subjects for analysis.

Overall, among all 766 runners enrolled, female runners were younger than male runners (mean [±SD] age, 36.1±8.8 vs. 40.4±9.7 years; $P<0.001$) and had a lower prerace weight (58.8±6.8 vs.

Table 1. Baseline Characteristics of the 2002 Boston Marathon Study Population.*

Characteristic	Male Runners (N=473)		Female Runners (N=293)	
	Reporting at Finish Line (N=336)	Not Reporting at Finish Line (N=137)	Reporting at Finish Line (N=175)	Not Reporting at Finish Line (N=118)
Age — yr	40.4±9.6	40.4±10.0	36.3±8.8	35.7±8.8
Nonwhite race — %	9	10	6	6
Prerace weight — kg	74.6±9.5	76.6±10.7	58.9±6.7	58.7±7.1
Body-mass index†	23.7±2.6	24.5±2.7	21.4±2.0	21.4±2.1
Training pace — min:sec/mi	7:53±1:02	8:04±1:09	8:40±1:01	8:41±1:02
Previous marathons — median no. (interquartile range)	5 (2–12)	4 (1–12)	4 (2–8)	3 (1–6)
Self-reported water loading — %‡	75	79	70	85
Self-reported use of NSAIDs — %§	51	54	60	61
Race duration — hr:min¶	3:37±0:42	3:46±0:40	4:02±0:36	4:02±0:32

* Plus–minus values are means ±SD. The temperature and humidity at noon, at the start of the race, were 53°F (12°C) and 96 percent, respectively; at 2 p.m. at the finish line, they were 55°F (13°C) and 83 percent.
† The body-mass index is the weight in kilograms divided by the square of the height in meters.
‡ Water loading was defined as an increase in fluid consumption above baseline specifically in preparation for running the Boston Marathon.
§ NSAIDs denotes nonsteroidal antiinflammatory drugs. Use of NSAIDs was defined as any use within the week before the Boston Marathon.
¶ Race times of runners who did not report at the finish line were obtained by means of the Boston Marathon tracking Web site.

75.2±9.9 kg, P<0.001), lower body-mass index (21.4±2.1 vs. 24.0±2.7, P<0.001), a slower training pace (8:40±1:01 vs. 7.56±1:04 minutes per mile, P<0.001), less marathon experience (median of three vs. five previous marathons, Wilcoxon P<0.001), and longer racing time (4:02±0:35 vs. 3:40±0:42 hours, P<0.001). Runners who appeared for follow-up studies at the finish line had characteristics similar to runners who did not, except that women who reported for follow-up had completed one more previous marathon than women who did not report for follow-up (P=0.008) and were less likely to report water loading than women who were not followed up (P=0.04). Men who reported for follow-up had a lower body-mass index than men who did not report for follow-up (P=0.004) and completed the race nine minutes faster than men who were not followed up (P=0.04).

At the finish line, the runners had a mean serum sodium concentration of 140±5 mmol per liter (range, 114 to 158). Thirteen percent (62 of 488) had hyponatremia, including 22 percent of women (37 of 166) and 8 percent of men (25 of 322). Three runners (0.6 percent) had critical hyponatremia (serum sodium concentrations, 119, 118, and 114 mmol per liter).

Table 2 summarizes univariate and multivariate predictors of hyponatremia. Univariate predictors included female sex, a body-mass index of less than 20, longer racing time, consumption of fluids every mile, consumption of more than 3 liters of fluids during the race, and an increased frequency of voiding during the race. Hyponatremia was strongly correlated with weight gain during the race (Fig. 1). There were no differences between the runners with and those without hyponatremia in age, composition of fluid consumed, or self-reports of water loading and use of NSAIDs.

In the multivariate analysis, hyponatremia was associated with weight gain, longer racing time, and a body-mass index of less than 20. In selecting covariates for inclusion in the final model, we did not include variables for fluid consumption because of colinearity with weight gain, which we considered to be a stronger and more objective measure of fluid intake. Also excluded was training pace, which was colinear with race duration. Additional adjustment for the composition of ingested fluid, number of previous marathons, and reported use of NSAIDs was not statistically significant; their inclusion did not appreciably alter the coefficients of the remaining variables in the model.

Generalized additive models were used to assess the effects of a change in weight, race duration, and

Table 2. Univariate and Multivariate Predictors of Hyponatremia.*

Variable	Univariate Predictors			Multivariate Predictors	
	Hyponatremia (N=62)	No Hyponatremia (N=426)	P Value†	Odds Ratio (95% CI)	P Value†
Demographic characteristics					
Age (yr)	38.1±9.5	39.0±9.4	0.52	—	—
Nonwhite race (%)‡	8	8	1.00	—	—
Female sex (%)	60	30	<0.001	—	—
Body-mass index	22.8±3.7	23.0±2.5	0.68	—	—
Category of body-mass index			0.01		
<20 (%)	25	8	—	2.5 (1.1–5.8)	0.03
20–25 (%)	54	73	—	1.0§	—
>25 (%)	21	19	—	1.0 (0.4–2.0)	0.90
Training and performance					
Previous marathons (no.)	3	5	0.008	—	—
Training pace (min:sec/mi)	8:52±1:11	8:02±1:01	<0.001	—	—
Race duration (hr:min)	4:12±0:47	3:42±0:42	<0.001	—	—
Category of race duration (hr:min)			<0.001		
<3:30 (%)	13	44	—	1.0§	—
3:30–4:00 (%)	35	31	—	3.6 (1.4–11.5)	0.01
>4:00 (%)	52	25	—	7.4 (2.9–23.1)	<0.001
Fluids and electrolytes					
Self-reported fluid intake					
Frequency (%)			<0.001		
Every mile	75	54	—	—	—
Every other mile	25	36	—	—	—
Every third mile or less often	0	9	—	—	—
Volume, >3 liters (%)	42	26	0.01	—	—
Composition, 100% water (%)	8	11	0.66	—	—
Self-reported water loading (%)¶	82	73	0.16	—	—
Self-reported frequency of voiding during race (%)			0.047		
None	51	63	—	—	—
Once	27	25	—	—	—
Twice	8	8	—	—	—
Three times or more	14	5	—	—	—
Postrace weight > prerace weight (%)	71	29	<0.001	4.2 (2.2–8.2)	<0.001
Self-reported use of NSAIDs (%)‖	61	53	0.34	—	—

* Hyponatremia was defined as a serum sodium concentration of 135 mmol per liter or less. Plus–minus values are means ±SD. CI denotes confidence interval, and NSAIDs nonsteroidal antiinflammatory drugs. Dashes indicate not applicable. Percentages may not sum to 100 because of rounding.

† For the univariate analysis, all continuous variables were analyzed with the use of t-tests, all categorical data were analyzed with the use of Fisher's exact test, and the number of previous marathons was analyzed with the use of the Wilcoxon rank-sum test. For the multivariate analysis, P values were determined by Wald tests, and profile-likelihood confidence intervals were determined with the use of logistic regression.

‡ Race was self-reported.

§ This group served as the reference group in the multiple logistic-regression analysis.

¶ Water loading was defined as increasing fluid consumption above baseline specifically in preparation for running the Boston Marathon.

‖ Use of NSAIDs was defined as any use within the week before the Boston Marathon.

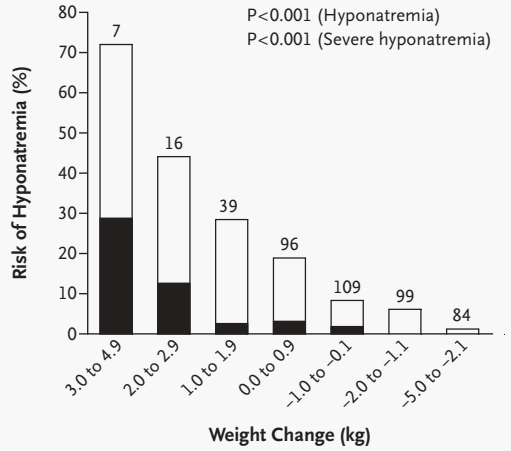

Figure 1. Risk of Hyponatremia and Severe Hyponatremia According to Weight Change among Runners in the 2002 Boston Marathon.

The total height of the bar represents the percentage of runners in a given stratum of weight change with hyponatremia (serum sodium concentration at race completion, ≤135 mmol per liter), whereas the black area of the bar represents the percentage of runners with severe hyponatremia (serum sodium concentration at race completion, ≤130 mmol per liter). The numbers above each bar denote the sample size in each weight-change category. The numbers do not sum to 488 because of missing data. On the x axis, positive numbers denote weight gain, and negative numbers weight loss. P values were determined by a test for trend.

body-mass index as continuous predictors of hyponatremia (Fig. 2). There were no significant departures from linearity for weight change (P=0.47) or race duration (P=0.40), but body-mass index had a strong nonlinear (approximately quadratic) relation with hyponatremia (P=0.002). A 1-kg increase in weight conferred an odds ratio of 2.0 (95 percent confidence interval, 1.6 to 2.6; P<0.001), and a 30-minute increase in running time conferred an odds ratio of 1.6 (95 percent confidence interval, 1.3 to 2.1; P<0.001). Additional adjustment for female sex (P=0.20) or drinking 100 percent water (P=0.89) was not statistically significant and did not appreciably alter the coefficients of the remaining variables in the model.

Given the strength of weight gain as a predictor of hyponatremia, we performed a secondary analysis to identify the determinants of weight gain. Thirty-five percent of the runners gained weight during the race (range, 0.1 to 4.1 kg). In a multivariate analysis, intake of 3 or more liters of fluid, fluid intake every mile, longer racing time, female sex, and body-mass index of less than 20 were associated with weight gain.

DISCUSSION

We observed that hyponatremia occurs in a substantial fraction of marathon runners and can be severe. The strongest single predictor of hyponatremia was considerable weight gain during the race, which correlated with excessive fluid intake. Longer racing time and body-mass-index extremes were also associated with hyponatremia, whereas the composition of fluids consumed (plain water, rather than sports drinks that contain electrolytes), female sex, and reported use of NSAIDs were not.

These results are consistent with earlier reports that suggested a link between excessive fluid consumption and hyponatremia.[3-5,7,9,13,17,18] However, earlier studies were limited by a small sample size, a retrospective study design, or a focus on elite or ultraendurance runners whose risk of the development of hyponatremia seems to be substantially lower than the risk among nonelite runners. In contrast, our study focused on a large, athletically diverse cohort of marathon runners followed prospectively to estimate the incidence of hyponatremia.

These observations suggest that hyponatremia — and particularly severe hyponatremia — may be a greater problem than previously recognized. If our sample was representative of the overall 2002 Boston Marathon field of runners, we would estimate that approximately 1900 of the nearly 15,000 finishers had some degree of hyponatremia, and that approximately 90 finishers had critical hyponatremia.

Substantial weight gain appeared to be the most important predictor of hyponatremia and correlated with increased fluid intake. Our finding of greater frequency of voiding among runners with hyponatremia suggests that most runners gain weight as a result of excessive fluid consumption, although inappropriate fluid retention may also have a role. Most reported cases of serious illness have involved runners in the United States. Our findings indicate that the problem of excessive hydration is not an isolated occurrence but may be part of a tendency among many U.S. marathon runners, especially those in the nonelite category, in which most of the growth in running has occurred.[20]

We could find no association between the composition of fluids consumed and hyponatremia. This finding probably reflects the relative hypotonicity of most commercial sports drinks, which have a typical sodium concentration of 18 mmol per liter,

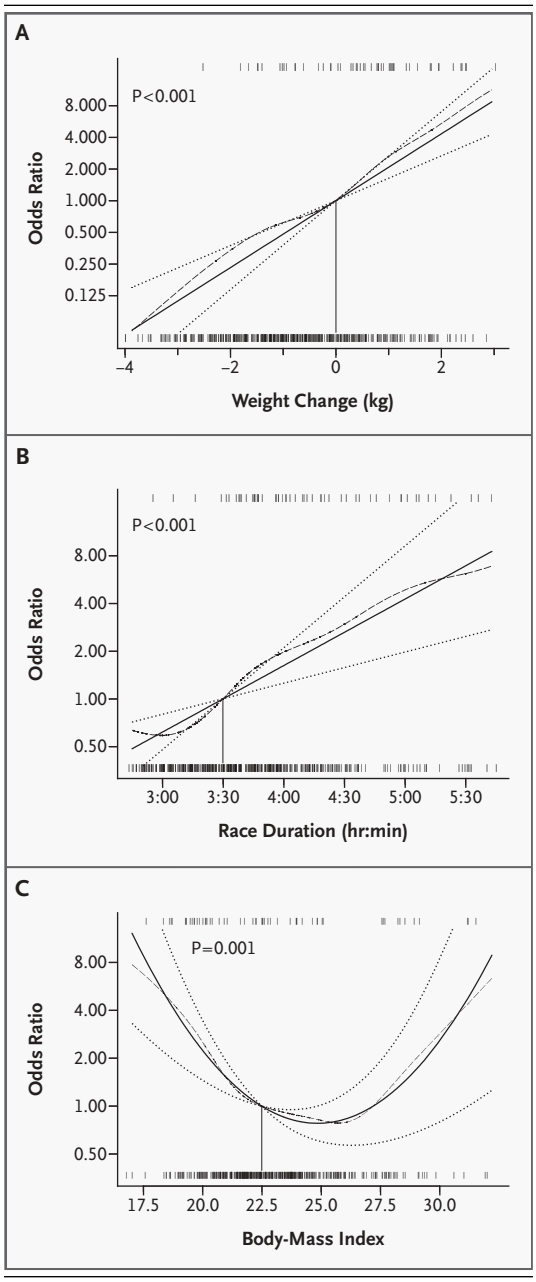

Figure 2. Adjusted Odds Ratios for Weight Change (Panel A), Race Duration (Panel B), and Body-Mass Index (Panel C) as Predictors of Hyponatremia among Runners in the 2002 Boston Marathon.

Results from a logistic-regression model showing the linear relationships of weight gain and race duration with hyponatremia, and the quadratic relationship of body-mass index with hyponatremia, were overlaid on the plot of the generalized additive model, demonstrating that the simpler parametric model adequately described the covariate effects. Dashed lines represent the fit of the generalized additive model. Solid lines represent the parametric logistic-regression fit (quadratic for body-mass index and linear for race duration and weight change). Dotted lines represent pointwise 95 percent confidence limits for the parametric fits. P values denote the overall effect of the covariate in predicting hyponatremia in the parametric logistic-regression fit. Tick marks above the odds-ratio curve represent runners with hyponatremia (defined as a serum sodium concentration of 135 mmol or less), whereas tick marks below the odds-ratio curve represent runners without hyponatremia. All models were constrained to cross at an odds ratio of unity.

less than one fifth the concentration of normal saline. Although it is difficult to rule out some effect of the type of fluid consumed on the risk of hyponatremia, our findings suggest that the contribution of the type of fluid is small as compared with the volume of fluid ingested.

Hyponatremia developed in more female than male runners, but this difference was not statistically significant after adjustment for body-mass index, racing time, and weight change. Female runners remain a readily identifiable risk group, and our observations suggest that this may be because of body size and longer racing time, rather than sex per se. However, the influence of sex on weight change during exercise[9] merits further study. It is not clear why both high and low body-mass indexes are associated with hyponatremia. Low body-mass index may be associated with hyponatremia because smaller runners may drink larger volumes of fluids in proportion to their size than larger runners. Conversely, in proportion to their size, larger runners may lose less free water than smaller runners through evaporation (by means of sweat), as a result of a lower ratio of surface area to volume.

The data from the present study suggest that hyponatremia associated with the running of marathons — and more broadly, with high-endurance exercise[14,15,21-23] — may be a preventable condition. One relatively simple strategy to reduce the risk would be for runners to weigh themselves before and after training runs to gauge the effectiveness of their overall hydration strategy and adjust their fluid intake accordingly. This could be particularly useful during long training runs in which the distance and duration most closely approximate those of an actual marathon. Because runners vary considerably in size and in rates of perspiration, general recommendations regarding specific volumes of fluids and frequencies of intake are probably unsafe and have been superseded by recommendations favoring thirst or individual perspiration rates as a primary guide.[20,24] Sporadically checking their weight could be a relatively easy way for runners to deter-

mine whether their current hydration strategy puts them at undue risk for the development of hyponatremia.

The present study must be interpreted within the context of certain limitations. First, follow-up in our study population was 67 percent, which may have skewed results if differential follow-up occurred. However, female and slower runners, who appear to be at higher risk for hyponatremia, seemed to be less likely to follow up with the researchers at the finish line, suggesting that our observations underestimated the overall incidence of hyponatremia. Second, baseline measurements of serum sodium concentration were not obtained before the marathon, raising the possibility that electrolyte derangements present at the end of the marathon were present before the start of the race. However, we could find no data to suggest that baseline concentrations of serum sodium in athletes would be different from those in nonathletes.[9] Finally, we relied on runners' self-reports of fluid intake during the marathon, which may be an imprecise estimate of intake. However, runners' self-reports of fluid intake correlated well with weight change, which is a more objective and widely accepted clinical measure of fluid balance.

In summary, we observed that a substantial portion of runners have abnormally low serum sodium concentrations after completing a marathon. Excessive consumption of fluids, as evidenced by substantial weight gain while running, is the single most important factor associated with hyponatremia. Efforts to monitor and regulate fluid intake may lead to a reduction in the frequency and severity of this condition, which, in rare cases, can be fatal.

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APPENDIX

In addition to the authors, the following participated in the study: *Children's Hospital, Boston — Department of Medicine House Staff, 2001 to 2002:* I. Chen, E. Copeland, J. DeJong, E. Fleegler, J. Han, K. Levine, T. Lin, Y. Requena-Kassarijian, P. Sarin, L. Trasande, F. Bourgeois, D. Brahan, J. Cohen, M. Cotter, E. Flynn, S. Huang, C. Lumeng, H. McLauchlan, A. McQueen, E. Milliken, D. O'Connor, E. Wein, D. Weir, S. Wingerter, S. Agarwal, B. Feldman, B. McCabe, C. Moore, P. Lio, M. Pao, J. Raphael, M. Shah, R. Tenney, C. Ullrich, P. Weinstock; *Nursing Staff:* A. Bussiere, C. Clauson, E. King, C. O'Sullivan, J. Tucker, R. Willis, J. Powers; *Department of Laboratory Medicine:* J. Barrow-Castillo, G. Bradwin, I. Clark; *Cardiology Clinical Research Unit:* K. Alexander, L. Buckley, A. Donati, D. Donati, D. Duva, A. Geggel, L. Kyn; *General Clinical Research Center and Clinical Research Program:* K. Jordan, C. Valim; *Faculty and Staff:* S. Brooks, S. Craig, B. Fitzgerald, G. Fleisher, M. Landzberg, J. Lock, F. Lovejoy, L. Micheli, E. Neufeld, N. Rifai, A. Stracciolini; *Harvard Combined Medicine Pediatrics Program* — A. Bhatt, C. Camacho, J. Welch, M. Solomon; *Harvard Affiliated Emergency Medicine Program* — E. Binstadt; *Brigham and Women's Hospital, Boston* — J. Seifter.

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