CASE REPORT

Sudden collapse of a young female cross country runner

A S Kashyap, K P Anand, S Kashyap

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The case is reported of a young previously healthy female cross country runner who collapsed on completion of a cross country run. The cause of the collapse was non-cardiogenic pulmonary oedema as a manifestation of hyponatraemic encephalopathy. The concurrent occurrence of non-cardiogenic pulmonary oedema and encephalopathy due to hyponatraemia is unusual.

A 33 year old previously fit female cross country runner collapsed after completing a cross country run of 35 km. She was brought to the emergency room with recurrent vomiting, nausea, and watery frothy sputum. There was no history of illness or alcohol or drug abuse. Her only drugs were ibuprofen tablets taken irregularly for mild muscular pain over the preceding few months. Her menstrual cycles were normal. Clinical examination revealed a healthy young woman, who responded feebly to painful stimuli. There were no localising or lateralising neurological signs, papilloedema, or signs of meningeal irritation. Blood pressure was 110/70 mm Hg, temperature 37.0°C, pulse rate 82 per minute, and respiratory rate 32 per minute. Chest examination revealed fine extensive crepitations in infrascapular, interscapular, and axillary areas. Cardiovascular examination was normal. Other systems were normal. Laboratory results were: normal packed cell volume and blood counts; oxygen saturation 64% (normal 97% in arterial blood); bicarbonate 18 mmol/l (normal range 21–28); blood urea nitrogen 1.87 mmol/l (3.6–7.1); creatinine 58.0 µmol/l (<133); glucose 6.0 mmol/l (4.2–6.1 fasting); normal creatine kinase MB bands and troponin T; serum sodium 119 mmol/l (136–145), potassium 4.2 mmol/l (3.5–5.0), and chloride 94 mmol/l (98–106); normal calcium and phosphate concentrations. Chest radiograph revealed pulmonary oedema with normal cardiac size. Electrocardiogram and two dimensional echocardiogram were normal. Pulmonary capillary wedge pressure was subnormal. The patient was intubated and ventilated. There was poor response to ventilation and there was no change in her sensorium. In view of persistent hyponatraemia and features of encephalopathy, an endocrinology consultation was sought. A diagnosis of hyponatraemic encephalopathy clinically manifesting as non-cardiogenic pulmonary oedema was made. A computed tomography brain scan revealed cerebral oedema with effacement of the sulci and chinking of the ventricles. The patient was treated with hypertonic saline (514 mmol/l); plasma sodium increased by 11 mmol/l in 10 hours to 130 mmol/l. It gradually returned to normal over the next 18 hours on a normal saline infusion. There was clinical and radiological resolution of the pulmonary and cerebral oedema within 18 hours. The patient was conscious and oriented. Oxygen saturation was normal, and she was gradually weaned off the ventilator and discharged. She had no problems at the six month follow. Brain magnetic resonance imaging performed six months later showed normal results.

DISCUSSION

Hyponatraemia is a well documented entity in a substantial fraction of long distance runners and can be severe with critical manifestations^{1,2} This case highlights that non-cardiogenic pulmonary oedema can be a manifestation of hyponatraemic encephalopathy in healthy long distance runners. Low pulmonary capillary wedge pressure and normal cardiac size on chest radiograph and echocardiogram supports this diagnosis. Administration of hypertonic saline led to resolution of the cerebral oedema and pulmonary oedema, indicating a link between cerebral oedema and non-cardiogenic pulmonary oedema. Development of pulmonary oedema in long distance runners is well documented,^{3–5} but presentation with both cerebral oedema and non-cardiogenic pulmonary oedema is a relatively recently described entity.⁶ Premenopausal female long distance runners are more likely to develop hyponatraemia, as well as symptoms of hyponatraemia, than male runners.^{7,8}

Strenuous exercise leads to diversion of the blood supply from the gastrointestinal tract to skeletal muscle, and any water swallowed is sequestered in the bowels.⁹ Sweating leads to loss of water and sodium resulting in volume contraction and stimulation of secretion of arginine vasopressin hormone.^{8–10} The latter increases retention of swallowed water. On cessation of exercise, the blood flow to the gut increases because of redistribution, leading to sudden reabsorption of water into the vascular compartment, which in turn leads to hyponatraemia.^{9,11,12} The acute hyponatraemia leads to raised intracranial pressure, with hypoxia and non-cardiogenic pulmonary oedema.¹³ Furthermore long distance runners are known to use non-steroidal anti-inflammatory drugs, which impairs water excretion, contributing to hyponatraemia.¹⁴

What is already known on this topic

- Hyponatraemia, which can have critical manifestations, is well documented in long distance runners particularly premenopausal female long distance runners
- Hyponatraemic encephalopathy manifesting as cerebral oedema and non-cardiogenic pulmonary oedema have been reported in long distance runners

What this study adds

- A case of non-cardiogenic pulmonary oedema as a manifestation of hyponatraemic encephalopathy in a healthy long distance runner is reported and discussed

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Authors' affiliations

A S Kashyap, S Kashyap, Command Hospital (Southern Command), Pune 411 040, India

K P Anand, Command Hospital (Eastern Command), Kolkata 700 027, India

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Correspondence to: Dr Kashyap, Command Hospital (Southern Command), 1-C, Wanowrie Road, Pune 411 040, India; kashyapajits@yahoo.com

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