CASE REPORT PEER REVIEWED | OPEN ACCESS

Near-syncope after swimming in cold water

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ABSTRACT

Introduction: Swimming in cold water ("polar bear" swimming) is growing in popularity and attracting individuals of many ages and athletic backgrounds. Case Report: A case of 60-year-old woman swimmer who experienced near-syncope after swimming for 20 minutes in cold water (water temperature: 14°C) without a wetsuit. The patient did not have signs of clinically-relevant hypothermia but was brought to a sauna after swimming. Pre-existing volume depletion, with subsequent orthostatic hypotension made worse by swimming in cold water, was the most likely etiology of near-syncope in this patient. Clinical improvement was noted when the patient started shivering after being removed from the sauna, and all symptoms resolved after the administration of 1 L of normal saline. Conclusion: As cold-water swimming continues to gain in popularity, emergency medicine practitioners may see more cases of swimming-related syncope and near-syncope. Depending on the presentation, the priority of treatment may be the correction of volume depletion and orthostatic hypotension, rather than active rewarming.

Keywords: Cold water, Exercise, Hypothermia, Near-syncope, Swimming, Syncope

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INTRODUCTION

Swimming in cold water is growing in popularity and attracting individuals of many ages and athletic backgrounds [1–4]. Each year several "polar bear" events draw large crowds that endure cold water without wetsuits, typically for brief periods of time [5, 6]. More consistent winter swimming is also becoming more common [4, 6, 7].

Syncope or near-syncope after cold-water swimming has been reported in the media. In a report from Iceland, 16 cases of syncope after swimming in cold water were reported in a two-year period [8, 9]. Although medical details of these cases were not provided, it was reported that an ambulance was called for six individuals, and that one case involved a cardiac event.

A recent case involving a swimmer in San Francisco allows us to examine swimming-related syncope and near-syncope in more detail. The swimmer, a 60-year-old woman who swims frequently in cold water for recreation, provided written consent for the use of her medical information.

CASE REPORT

In November 2015, the swimmer swam for approximately 20 minutes in the San Francisco Bay without a wetsuit. The water temperature was 14°C. The swimmer swam from a beach adjacent to a local swim club; club facilities have both warm showers and a sauna. During the swim the swimmer felt mildly lightheaded. After exiting the water, lightheadedness became more pronounced. With the help of others, she was able to ambulate from the beach and up a flight of stairs to the club's locker room.

In the locker room, the patient was unable to ambulate and sank to the floor. With the assistance of others, she was taken to a sauna, where she remained on the floor in a lateral decubitus position. Although she did not become unconscious, she continued to feel extremely lightheaded. She vomited twice after attempting to drink warm fluids. Paramedics were called, and the patient was transported to a local emergency department. Mild shivering began after removal from the sauna, during transport to the hospital.

In the emergency department, the patient was alert and oriented. Initial vital signs were recorded as: blood pressure 97/84 mmHg, pulse 94 bpm, O_2 saturation on room air 96%, oral temperature 36.3°C. Body mass index (BMI) was 40.1 kg/m^2. She had a history of hypertension and had taken hydrochlorothiazide 12.5 mg orally earlier the same day. Physical examination was unremarkable. Laboratory values (hemoglobin 18.1 g/dl, hematocrit 52.9%, BUN 20, creatinine 0.9, TCO_2 27), were consistent with volume depletion. Glucose was mildly elevated (143 mg/dl). Other laboratory values were within normal limits. Electrocardiography (ECG) showed normal sinus rhythm and was without acute changes. The patient reported no shortness of breath, chest pain, or other systemic symptoms, and no dysrhythmia was noted by cardiac monitor at any time. Conventional warm blankets were applied and shivering resolved gradually. She had no further emesis. After receiving 1 liter of intravenous normal saline the patient reported that lightheadedness had resolved completely. She was discharged home after 1 hour and 45 minutes. Blood pressure on discharge was 120/79. Following discharge the patient had no complications. She resumed swimming three days after the incident and continues to swim regularly.

Figure 1: Cold-water Swimming, San Francisco Bay (Photo: S. Lauritzen).

DISCUSSION

On review, we believe that pre-existing volume depletion, with subsequent orthostatic hypotension made worse by swimming in cold water, was the most likely etiology of near-syncope in this patient. Hypothermia itself can contribute to changes in consciousness [10]. However, such changes are typically seen in the more advanced stages of hypothermia. The patient's exposure to cold water was relatively brief, and her elevated BMI was protective against core hypothermia [2–4]. Although rectal or other core temperature measurements were not made, the patient's presentation suggests that hypothermia, if present, was only mild. Additionally, she had several findings consistent with volume depletion (including hemoconcentration and an elevated BUN/CR ratio), she had taken a diuretic earlier the same day, and she improved after administration of an isotonic intravenous fluid.

Fortunately, based on media reports [5, 6, 8, 9], syncope after swimming in cold water appears to be relatively infrequent. We believe that orthostatic hypotension may account for many of these cases.

Orthostatic hypotension has been described after long-distance swimming in warm water [11] and after other forms of exercise [12, 13]. The potential mechanisms for post-exercise hypotension are complex [11–13]. Post-swim hypotension may be the result of blood pooling in the legs due to inactivation of the venous muscle pump on completion of a swim [11]. Post-exercise reductions in vascular resistance mediated by the autonomic nervous system and vasodilator substances [13], or a decrease of the hydrostatic pressure of water on the body after exiting the water [14, 15] may also contribute.

Swimming in cold water, which causes more pronounced peripheral vasoconstriction, could make orthostatic hypotension even worse. Since vasoconstriction is already maximal as the result of cold stress, a further increase in peripheral vascular resistance may not be possible. Thus, removing an already volume-depleted swimmer from cold water could result in abrupt hypotension – similar to the sudden deflation of anti-shock trousers in a patient with hypovolemic shock [14, 15].

Additionally, active rewarming without volume resuscitation could cause peripheral vasodilation and further contribute to orthostatic hypotension. Media reports of Icelandic swimming [8, 9] mention that

swimmers often immerse themselves in hot water immediately after exiting cold water. This likely increases the risk of syncope. Interestingly, our patient reported that she felt better once she started to shiver, after she was removed from a sauna; both shivering and removal from heat may have contributed to an increase in vascular tone and clinical improvement.

Thus, we believe that this case highlights an important treatment distinction. Although bystanders brought the patient in this report to a sauna, she had been exposed to cold water for a relatively brief period of time and did not have signs of clinically-relevant hypothermia. While rewarming is understandably a priority in hypothermic patients, after syncope or near-syncope rapid rewarming may worsen vascular tone and delay clinical improvement.

Prior to swimming, cold-water swimmers should make efforts to avoid dehydration or volume depletion. Diuretics and antihypertensive medications should be taken with caution. Gradually exiting the water and avoiding exertion immediately after swimming may also be beneficial.

While we speculate that orthostatic hypotension is a common etiology of syncope and near-syncope after swimming in cold water, other causes must be considered. Dysrhythmias, including those related to long QT syndrome, have been associated with swimming and other forms of exercise [16–18]. Myocardial infarction, structural heart disease, vasovagal syncope, cold urticaria or anaphylaxis [19, 20], and other possibilities should also be considered.

CONCLUSION

In summary, as cold-water swimming continues to gain in popularity, emergency medicine practitioners may see more cases of swimming-related syncope and near-syncope. Careful consideration should be given to the differential diagnosis in all cases. Depending on the presentation, the priority of treatment may be the correction of volume depletion and orthostatic hypotension, rather than active rewarming.

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Author Contributions

Thomas J. Nuckton – Conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Critical revision of the article, Final approval of the version to be published

Ritik Chandra – Conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Critical revision of the article, Final approval of the version to be published

Kelley D. Heye – Conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Critical revision of the article, Final approval of the version to be published

Susan K. Lauritzen – Conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Critical revision of the article, Final approval of the version to be published

Mary Magocsy – Conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Critical revision of the article, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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