The Hidden Dangers of Beaches: Cardiorespiratory Arrest Induced by Thermal Shock

HM Café[1], S Santos[2], V Pereira[3], S Chaves[4], P Faria[1], M Câmara[5], J Nóbrega[5]

ABSTRACT

Thermal shock is widely recognized by modern medicine. Its pathophysiological mechanisms are known, as are its possible consequences, but scientific reports in the literature about clinical cases with severe consequences are sparse. The authors present a case of cardiorespiratory arrest after prolonged sun exposure followed by a dive in the ocean. Other aetiological causes were ruled out, by exclusion, leading to the diagnosis of cardiorespiratory arrest caused by thermal shock. It is important to inform the public in general of the risks of negligent behaviour on the beach.

Keywords: Beach, cardiac arrest, thermal shock, water

Los Peligros Escondidos de las Playas: Parada Cardiorespiratória por Choque Térmico

HM Café[1], S Santos[2], V Pereira[3], S Chaves[4], P Faria[1], M Câmara[5], J Nóbrega[5]

RESUMEN

El choque térmico es amplamente conocido por la medicina moderna. Sus mecanismos fisiopatológicos son conocidos, así como sus posibles consequencias, pero la literatura cientifica raramente presenta casos con consecuencias graves. Los autores presentan un caso de parada cardiorespiratoria después de una exposición prolongada al sol seguida por un baño en el mar. Otras causas etiologicas fueran descartadas, conduzindo al diagnóstico por exclusión de parada cardiorespiratoria por choque térmico. Es importante informar el público en general de los riesgos de comportamiento negligente en la playa.

Palavras claves: Choque térmico, playa, água, parada cardiorespiratória

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INTRODUCTION

Rapidly diving into water after a prolonged period of sun exposure should not be done. Although this saying is based on erroneous assumptions, a review of the pathophysiological mechanisms involving the human body gives us real data suggesting that there is a considerable risk of thermal shock by contact with water, like the one that occurs with rapid immersion or submersion following a long period of sun exposure. The authors present a rare and interesting case of thermal shock with severe consequences, with the aim of opening a debate on a usually ignored subject.

CASE REPORT

A 58-year old woman, with a medical history of depression medicated with diazepam and fluoxetine, but poorly compliant with therapy (in the past two months had only infrequently used diazepam and no fluoxetine), was on the beach, accompanied by her husband, on a sunny day, with an air temperature of 25 ˚C, ultraviolet (UV) index of 11 and water temperature of 20 ˚C. After a period of sun exposure lasting several hours, she jumped, feet first, from a rock slate into a natural ocean pool. Her husband observed from inside the pool. After resurfacing, the patient swam for about 30 seconds, showing progressive difficulty in doing so, losing the ability to remain on the surface of the water and crying for help before submerging. The husband rushed toward her, and found her unconscious and floating upside down. A lifeguard intervened and she was

From: [1]Department of Cardiology, [2]Department of Anesthesiology, Hospital Central do Funchal, Funchal, Madeira, Portugal, [3]Department of Internal Medicine, Hospital Espírito Santo, Évora, Portugal, [4]Department of Internal Medicine and [5]Department of Intensive Care, Hospital Central do Funchal, Funchal, Madeira, Portugal.

Correspondence: Dr HM Café, Serviço de Cardiologia, Hospital Dr Nélio Mendonça, Av Luís de Camões, 57 - 9004-514, Funchal, Madeira, Portugal. Fax: 00351291743098; e-mail: Hmcafe@gmail.com

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taken ashore and afterwards transported to a local medical facility by the fire department, with the firemen performing basic life support during the transport. Upon arrival at the local medical facility, cardiorespiratory arrest was documented by the attending physician, who started advanced life support (ALS). After 15 minutes of ALS, the patient recovered spontaneous circulation. In the meantime, the prehospital medical emergency team arrived and took over, transporting the patient to the hospital. At hospital admission, the patient was nonresponsive, with a Glasgow Coma Scale (GCS) of 3 and blood pressure of 106/73 mmHg. She was mechanically ventilated and the electrocardiogram (ECG) showed normal sinus rhythm with 93 beats per minute. There were no signs of trauma. The patient was examined by both internist and cardiologist, and given the neurological status and ventilatory instability, she was admitted to the intensive care unit (ICU) and subjected to induced hypothermia (core temperature 32 ˚C) for 24 hours. Blood tests were performed, including complete blood count, D-dimer levels, renal and hepatic function tests, electrolytes and cardiac enzymes. All these blood tests were repeated six, 12, 24 and 48 hours after admission. All tests were normal, except for a slight increase in creatine kinase (CK) levels, attributed to cardiac compression. Serial blood gas analyses were also performed, showing good gas exchange (in a ventilated patient). Cranial computed tomography (CT) scan showed no sign of ischaemic or haemorrhagic lesion. Transthoracic echocardiogram indicated no myocardial infarction. Serial ECGs were performed, without showing signs of ischaemia, QT or PR intervals alterations or accessory pathways. Chest X-ray, on admission, was normal. Twenty-four hour Holter monitoring was performed, showing no sign of arrhythmic events. Neurologically, the patient progressed to a GCS of 6, with no further improvement. During the patient stay, she presented with myoclonic jerks of the arms, face and feet. She was evaluated by a neurologist and medicated with sodium valproate and levetiracetam with good clinical response, albeit with no complete resolution. On the 4th day after admission, an *E aerogenes* pneumonia was diagnosed and treated with meropenem according to the antibiotics sensitivity tests with good clinical and radiological resolution. At day six, the patient was given a surgical tracheotomy and was transferred on the 13th day after admission from the ICU, breathing spontaneously, but still in GCS of 6. Presently the patient maintains the same neurological state.

DISCUSSION

Thermal shock has various pathophysiological consequences, and we shall focus on some points. The response to cold water immersion, maintaining the head above water, presents three distinct phases (1). In the first phase, contact with water causes an increased respiratory drive, followed by an increase of up to 400% in pulmonary ventilation (hyperventilation) which may, *per se,* cause small muscular spasms, tetany and drowning. Simultaneously, there is a decrease of 30 to 50% in pulmonary compliance, due to the fact that water is denser than air and, as such, causes a decrease in negative transthoracic pressure (2). These two facts together create a hypoxic effect. From the cardiovascular point of view, this is a powerful sympathetic stimulus, leading to generalized vasoconstriction and tachycardia (3). In these situations, the enhanced activity of the sympathetic nervous system is described as having a deleterious effect similar to that of obstructive sleep apnoea, a condition associated with nefarious cardiac events (4).

Because of this sympathetic stimulation, premature heartbeats are frequent, having been demonstrated even in healthy individuals and in waters at 25 ˚C (5).

We also have to mention the "diving reflex", which occurs when the neck and face are in contact with cold water, causing substantial stimulation of the trigeminal and vagal nerves (parasympathetic system), producing bradycardia, apnoea and hypotension. In a study by Datta and Tipton (6), healthy subjects dressed only with swim shorts were immersed in cold water and instructed to hold their breath. In this study, 60% of the subjects had arrhythmias, particularly supraventricular and junctional. Tipton *et al* (7) performed a study with healthy individuals submerged in water at five and 10 ˚C who, after a period of apnoea for the longest time possible, started breathing through a tube. In 29 of the 36 submersions (80.5%), there was an incidence of 91.6% of arrhythmic events, namely auricular premature beats, junctional complexes, supraventricular tachycardia runs and even ventricular premature beats. These arrhythmias occurred mainly in a period of 10 seconds after the apnoea had stopped.

This first phase of thermal shock is short and, according to Tipton and Brooks, may cause death in three to five minutes (1). Phase two, namely the inability to swim because of rapid cooling of muscles and nerves, occurs between five and 30 minutes, and phase three, which is hypothermia, occurs after 30 minutes (1). Given the characteristics of this clinical case, the authors chose not to explore these other phases.

In the present clinical case, the short period in which the situation developed is striking, as is the absence of trauma or external intervention, as the situation was witnessed by the husband. Cardiorespiratory arrest was documented by a medical doctor upon arrival at a medical facility.

Our study tried to exclude other potential events that could have caused cardiorespiratory arrest. Cranial CT scan ruled out a cerebral vascular event, blood tests ruled out electrolyte imbalances, major renal or hepatic failures, and anaemia. Blood gases, D-dimers and echocardiogram ruled out a massive pulmonary embolism, chest X-ray showed no signs of pneumothorax, and again echocardiogram, ECG, Holter and cardiac enzymes showed no sign of myocardial infarction, mechanical complication of the heart or substrate for an arrhythmic event. Also, as mentioned before, there were no signs of trauma.

Therefore, the diagnosis of cardiorespiratory arrest caused by thermal shock was considered to be the most likely. As Sherlock Holmes used to say, "Once you eliminate the impossible, whatever remains, no matter how improbable, must

be the truth." We must point out that, although the described mechanisms and quoted data relate to thermal shock with cold water, the most important factor is the difference between external body temperature and water temperature. A normothermic body immersed in water at 10 ˚C suffers the same thermal shock as a body exposed several hours to the sun and then rapidly immersed (or submerged) in water at 20 ˚C. Also, and as was already stated, even at 25 ˚C, a healthy individual can have arrhythmic events, so the important fact here is the thermal shock itself, not the absolute temperatures. The fact that arrhythmic events may occur even in healthy individuals in phase one of thermal shock may explain why our Holter study showed no alterations.

Finally, it is important to mention that this subject is not totally ignored by the scientific community. In fact, Datta and Tipton (4) stated that, although rare, there are data supporting cardiorespiratory arrest by immersion in water when it reaches the nostrils, although documented cases are not known.

CONCLUSION

Although this is an exclusion diagnosis, the authors consider that the particular aspects of this case and the data regarding thermal shock mechanisms justify the conclusion. Notwithstanding that the pathophysiological mechanisms quoted in this paper are well described, scientific literature is immensely sparse regarding clinical cases of cardiorespiratory arrest by thermal shock. It is of the utmost importance to inform not only the medical community but also the public in general of the risks of negligent behaviour on the beach, and instruct them to enter the water slowly, allowing a progressive adaptation.

AUTHORS' NOTE

The authors declare that this manuscript has been approved by all co-authors, has no financial support, is not associated to any university study and there is no conflict of interest.

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