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Case Report

A Work-Related Thyrotoxic Periodic Paralysis in A Worker Exposed to Heat Stress: A Case Report

Shanti Puji Lestari[1*], Muchtaruddin Mansyur[1]
[1] Occupational Medicine Specialist Study Program, Department of Community Medicine, Faculty of Medicine, Universitas Indonesia
*Email: shanty_pujilestari@yahoo.com

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ABSTRACT

Thyrotoxic Periodic Paralysis (TPP) is an uncommon disorder, characterized by the triad of acute hypokalemia, muscle paralysis, and thyrotoxicosis. Attacks are typically transient, last hours to days, and may be triggered by many conditions, including exposure to heat stress in both indoor and outdoor activities. Indoor workers, especially those working around furnaces, ovens, smelters, and boilers, are at a higher heat stress risk, even higher risk if they have other individual factors such as obesity, dehydration, slower heat acclimation, consume certain medications and presence of underlying diseases.
A 48-year-old man presented a sudden onset weakness in all four limbs, associated with admission blood hypokalemia and thyrotoxicosis. No history of either alcohol or high carbohydrate intake consumption, infection, trauma, or heavy exercise. He has been working as a chef in the food procurement unit at a hospital for 28 years, 6 hours/day, six days/week. The results of the heat measurement at the cooking area where he works revealed that the heat stress index was 31,9º C Wet Bulb & Globe Temperature (WBGT).

Introduction

Thyrotoxic periodic paralysis (TPP) is a thyroid-related disorder that is manifested as recurrent episodes of hypokalemia and muscle weakness (hypokalemic periodic paralysis) lasting from hours to days. It is most commonly described among Asian men and is a well-known complication of thyrotoxicosis.[1] The absence of a family history of paralysis, male sex, presentation in the second to fourth decades of life, and the sign of thyrotoxicosis help in the diagnosis of TPP.[2] Attacks may be triggered by some conditions. These include carbohydrates—especially refined carbohydrates and after exercise. Other precipitating factors include trauma, exposure to hot weather, upper respiratory tract infections, emotional stress, menses, medications (diuretics, insulin, glucocorticosteroids, and acetazolamide), alcohol, or recreational drugs such as 3,4-Methylenedioxymethamphetamine (ecstasy).[3]

The kitchen is primarily a workplace; it is a room in which the cooking chef spends long periods. There are two types of external heat exposure sources in the workplace: weather-related and man-made heat exposure.[4] The cooking area in the mess kitchen where a large quantity of food will be cooked emits more heat on the surface and surroundings around the room. The workers involved in the cooking process will be directly affected by the heat that is released from the stove. Heat gain can be a combination of external heat from the environment and internal body heat generated from metabolic processes.[4] The metabolic process in someone with hyperthyroidism is increased, thus presenting heat intolerance as one of the clinical manifestations.

Results and Discussion

Results

A 48-year-old non-smoker, non-diabetic, non-alcoholic Asian man presented to the emergency department one evening with complaints of numbness and weakness in his lower limbs when he woke up for urination at around 2:00 am on the day of admission. It started distally symmetrically and progressed proximally and gradually involved the upper limbs in the same pattern. He described going to bed in his normal state of health. No history of either respiratory, or urinary problems, sensory loss in any limb, backache, involuntary movements, trauma, high carbohydrate intake consumption, or heavy exercise was available. A history of weakness in both lower limbs was present 2 months ago which was recovered fully without any motor sequelae and remained asymptomatic till the day of admission. The examination found that the blood pressure was slightly high, at that time was 150/90 mmHg (it was the first time he recognized a history of hypertension). He also noted at the time of presentation there was a history of more than 5 kg weight loss over three months despite increased appetite. He also complained of palpitations and excessive sweating over the last 3 months. The patient denied the use of any prescribed or non-prescribed medications. There was a history of hypertension in the family (his mother) and no history of thyroid disorders or similar illnesses in the family.

On admission, blood pressure was 142/80 mmHg, pulse was 92/min, regular, and of good volume. Cardiovascular, pulmonary, and abdominal examinations were unremarkable. He was clinically hyperthyroid with fine resting tremor and diaphoresis of the hands, without exophthalmos, enlargement of the thyroid gland, or nodules. His upper and lower limb power was 0/5. There were no sensory abnormalities and the cranial nerves were intact. His serum potassium on admission was 2.29 mmol/L (normal 3,10 – 5,10). His thyroid-stimulating hormone was 0.01 μIU/L (normal 0.48-4.17), T4 was 143 ng/L (normal 50-113) and T3 was 2.49 ng/ml (normal 0.8-2.0). ECG showed sinus tachycardia only. This patient was diagnosed with Thyrotoxic Periodic Paralysis. After the hypokalemia condition was treated with intravenous potassium replacement, the paralysis immediately recovered.

The patient has worked as a cook in the Hospital's Food Supply Unit (UPM) for 28 years. For the last 10 years, the patient always works in the morning shift, at 07.00-13.00 (6 hours/day), 6 days/week, alternating days off with his coworkers. The patient worked specifically on the main food cooking section, not including preparing, washing, or cutting up foodstuff. During work, the patient wears uniforms provided by the hospital, colored white, and made from cotton. PPE used included masks, aprons, gloves, and hair covers. The kitchen measures 5x10m, and the main food cooking area is about ¼ part of the kitchen. There were 10 large gas stoves and 5 smaller gas stoves in the main food cooking area. Each stove is equipped with a cooker hood but due to low maintenance, its function is not optimal so when all stoves are used simultaneously, the room will be filled with smoke. There were patches with fabric in some leaking air ducts. There were 3 air conditioners, in the middle of the kitchen. The provision of drinking water for employees was located in the lounge room outside the kitchen.

We assessed heat stress exposure through measurements of the Wet Bulb Globe Temperature (WBGT) index. Measurements for WBGT were carried out using an area heat stress monitor (Model QuesTemp°36), comply with the standards set out by the American Conference of Governmental Industrial Hygienists (ACGIH), provided an average temperature of 31.9 ºC WBGT, with an average relative humidity of 79.5%. Additional information on workload, clothing worn, worker's time-activity pattern, and acclimatization were collected to make appropriate adjustments to the measured WBGT value. With a total workload of 163 kcal/hour, this patient underwent light work. Based on the worker's time-activity pattern (6 hours of accumulated work and 2 hours of rest in accumulation) included in the category 50 - 75% of work. With a working period as a cook for 28 years and no history of being absent in the past 1 month, it was considered this patient has been acclimatized. Clothing used during work is 2 layers of clothing, colored white, made from cotton, so there was no adjustment for clothing factor correction. For light workloads, with 50-75% of work and full acclimatization of the workers, a threshold limit value (TLV) of 31ºC was used.

Discussion

Kitchen workers are prone to heat stress at the workplace due to heat generated from cooking practices, especially in tropical countries.[5] In the tropics, the problem of heat exposure is an important factor that must be considered. Besides working weather, the body itself when carrying

out activities also emits heat. The body's metabolic processes that interact with heat in its environment will result in workers experiencing heat stress.[6] Heat pressure is a combination of work climate and metabolic processes. Work climate is the result of a combination of temperature, humidity, speed of movement of air, and radiation heat with the level of heat from the body of labor as a result of his work.[7]

Heat stress is the net heat load to which a worker is exposed from the combined contributions of environmental factors, metabolic heat, and clothing, all of which increase heat storage in the body.[8,9] The heat load experienced by a worker provokes a physiological response (heat strain), that increases heat loss from the body to maintain a stable body temperature. This physiological response is not always successful and, when unsuccessful, may result in heat injury and death.[8,9]

The specific environmental factors causing heat stress are high air temperature, minimal movement of air, high humidity, and radiant heat.[10] The results of measurements made in the kitchen of this patient's workplace indicated that the temperature of the work environment exceeded recommended exposure levels, with high air humidity. Haruyama et al.[11], studied the thermal strain in different types of kitchens. WBGT was assessed in ten kitchens of electric stove type and gas stove kitchens. The mean WBGT in gas kitchens was 29.6 C and in the electric kitchens was 25.7 C. The WBGT was significantly higher in the gas kitchens.
Physical work contributes to the total heat stress of a job because metabolic heat increases in proportion to work intensity. The amount, thermal characteristics, and type of clothing worn are also important because they alter the rate of heat exchange between the skin and the air.[10] If the body does not release heat, the body temperature will increase by 1ºC every hour. Body thermoregulation regulates the release of heat in the following ways: a) Convection (also sometimes radiation & conduction) heat, especially from the skin surface. b) Vasodilation (dilation) of blood vessels in the skin, increasing the release of heat through the skin. c) Increased evaporation of sweat through the skin. d) The blowing of hot air from the lungs. e) Heat dissipation through feces and urine.[12] Evaporation is the most important of the body's efforts to reduce body temperature during physical activity. The body's ability to be able to adapt to the hot temperature of its environment is very dependent on several factors that influence the speed of sweat evaporation, for example, the temperature and humidity of the surrounding air which can cause the evaporation of sweat to be interrupted. As a result, most sweat does not evaporate but drips.[12] High humidity in the patient's workplace inhibited the process of evaporation of sweat so it was not effective in reducing body heat.

Recent papers reported that personal factors such as heat acclimation, being older than 60, the experience of a previous heat-related illness, use of certain medications, presence of certain concurrent diseases, severe obesity, and dehydration also increase the risk of heat-related illnesses.[13-15] Heat intolerance is one of the clinical manifestations of hyperthyroid disease[16], resulting in interference with the thermoregulation mechanism. In someone with hyperthyroidism, there is also an increase in metabolic rate caused by the release of thyroxine and catecholamines (epinephrine and norepinephrine). A high metabolic rate will produce greater heat.[12]

TPP is a serious condition and is a potentially fatal complication of hyperthyroidism as a result of the movement of large amounts of potassium from extracellular to intracellular space.[17] The pathogenesis of TPP is still unclear. Hypokalemia occurs as a result of massive potassium transfer from extracellular to intracellular compartments, especially muscle cells. This is believed to be the result of an increase in the activity of the sodium potassium-adenosine triphosphatase (Na/K-ATPase) pump (figure 1). Various data indicate an increase in the number and activity of Na/K-ATPase pumps in TPP patients. The increase in number and activity was significantly different from thyrotoxicosis patients without TPP. If the thyrotoxicosis has been successfully controlled, Na/K-ATPase activity will return to levels similar to normal people. Thyroid hormones can increase Na/K-ATPase activity in skeletal, liver, and kidney muscles, causing potassium influx into the intracellular space.[17]

In addition, thyroid hormone can also affect Na/K-ATPase through catecholamine stimulation. Because in thyrotoxicosis, there is an increase in β adrenergic response.[17] Several studies have shown that there is a physiological response that occurs in labor due to the hot work climate.[7,18,19] This is indicated by the presence of sympathetic nerve stimulation in the form of increased body temperature, pulse, systolic blood pressure, and diastolic blood pressure. Most of the sympathetic postganglionic fibers are adrenergic, innervating the skin sweat glands, and blood vessels of the

skeletal muscles and the brain. The activation of these sympathetic fibers stimulates the secretion of sweat glands and the dilatation of blood vessels.[20]

The condition of hyperthyroidism in this patient affects the body's adaptation process to exposure to heat stress in the work environment, thereby increasing heat storage in the body. This can cause an increase in Na/K-ATPase activity through a mechanism of increasing adrenergic response, resulting in a shift in large amounts of potassium from extracellular to intracellular space, which in turn can trigger TPP. Recovery of the paralysis in this patient occurred immediately after the hypokalemia was being corrected, this may indicate the alterations in potassium homeostasis which can be the result of prolonged heat stress exposure. Hyperthyroid disease is an individual factor in this patient, there were no other factors outside of work that can be related to the incidence of TPP in this patient. This strengthens the justification that TPP in this patient is related to heat stress exposure at the workplace.

Conclusion

Exposure to heat stress is associated with an increased risk of TPP in light physical activity workers with thyrotoxicosis. Individual risk factors, environmental factors, and metabolic heat are the main determinants of heat-related illnesses. The effects of heat stress exposure can be fatal for workers. All workplaces must implement an acclimatization plan for workers who are exposed to heat, conduct medical surveillance and control the risk of heat stress exposure.

Declaration

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