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Sudden death during jungle trekking: a case of heat stroke

Kasinathan NADESAN *MRCPath, FRCPA,* ***Siew Pheng CHAN** *MBBS, FRCP* **and *Catherine Mee Ming WONG** *MRCP*

Departments of Pathology and *Medicine, Faculty of Medicine, University of Malaya, Kuala Lumpur.

Abstract:

Heat stroke, which is also known as "sun stroke," is a medical emergency, and fatalities can occur unless it is diagnosed early and treated efficiently. Heat stroke may manifest quite suddenly, giving little time to differentiate it from extreme physical exhaustion in collapsed subjects. It is also known to lead to serious disseminated intravascular coagulation. Sudden death in a young female is presented who collapsed after trekking in a hilly, jungle area in Malaysia on a warm, humid day. She had joined a weight reduction programme a few weeks earlier. She was found collapsed and in a semiconscious state in the jungle by her groupmates and was taken to hospital. On admission she was unconscious, hyperpyrexic, with rapid, thready pulse and a low blood pressure. Biochemical studies revealed metabolic acidosis, elevated liver and cardiac enzymes and impairment of renal function. Her coagulation profile was found to be impaired and she started bleeding through the mouth and nostrils. She also developed watery diarrhoea and initially a septicaemic condition, including acute enteritis was suspected. Despite active treatment, her condition deteriorated and she died eight hours after admission. Autopsy confirmed a generalised bleeding tendency, with pulmonary, oesophageal and gastrointestinal mucosal haemorrhages. Flame-shaped subendocardial shock haemorrhages were seen in the interventricular septum on the left side of the heart. The findings support a diagnosis of heat stroke. Various aspects related to heat stroke, the autopsy diagnosis and its prevention are discussed.

Key words: Heat stroke, disseminated intravascular coagulation.

INTRODUCTION

Heat stroke, which is also known as "sun stroke," usually occurs when a person is exposed to very high environmental temperature and humidity, where the heat regulating mechanism in the hypothalamus is disturbed.[1] This results in reduced or absent sweating with hyperpyrexia. This condition is a medical emergency, and fatalities can occur unless it is diagnosed early and treated efficiently. There are many factors that contribute to heat stroke besides the environmental conditions, such as the clothing worn by the person, amount of water intake during exertion, acclimatization, the nature of the physical exercise, presence of sweat rash, physical fitness, physical illness and so on.[2] It is well documented that heat stroke can lead to serious disseminated intravascular coagulation.[3] An unusual feature about this condition is that it manifests quite suddenly in most cases. There is little time to differentiate between extreme tiredness resulting from physical exhaustion and the manifestation of heat stroke, where the victim collapses.

In heat stroke the core body temperature generally reaches 39°C or above with absence of sweating. Hot and dry skin, increased depth of respiration simulating kussmaul breathing, rapid thready pulse, lowered blood pressure, and severe weakness are some of the major features.[2] Some of the victims present with watery diarrhoea and those who survive have complained of chest pain and intense thirst. Pupils are often constricted and sometimes pin-point. Lungs on auscultation may reveal generalised crepitations due to pulmonary oedema. Some of the victims present with haemoptysis or haematemesis due to disseminated intravascular coagulation (DIC).[2,3,4] Convulsions, delirium and stupor and temperatures above 42°C are poor prognostic signs. Chances of recovery are also poor with hypotension, hyperkalemia, hyperuremia, and elevated serum enzymes such as SGOT and LDH.[5,6]

In fatal cases due to heat stroke, the autopsy findings lack specificity. Perusal of the clinical notes of the victim who had managed to reach hospital and survive for a while, together with a detail history from some responsible person such as a military trainer or a colleague will be very

Address for correspondence and reprint requests: Associate Professor K. Nadesan, Department of Pathology, Faculty of Medicine, University of Malaya, 50603 Kuala Lumpur, Malaysia.

useful to the investigating pathologist. Autopsy usually reveals the presence of dehydration with dry muscles, congested internal organs, dark, thick and viscous blood and a generalised bleeding tendency due to disseminated intravascular coagulation.[2,3,4] Pulmonary haemorrhage with blood-stained fluid in the trachea and bronchi is a common finding. Widespread ultrastructural evidence of endothelial cell injury has been reported.' In persons who survive for some time, there may be evidence of multisystem failure such as renal failure, hepatic failure and cardiovascular collapse. Microthrombi in the capillaries with microinfarcts may be demonstrated at times.[3] A significant number of victims who survive for some time also showed evidence of adult respiratory distress syndrome (ARDS).[4] ARDS is clinically manifested by pulmonary oedema and respiratory distress and there will be crepitations in the lungs. Earlier it was thought that the generalised tissue injury was due to hyperthermia itself. Later it was observed that tissue injury continued to develop in 25% of heatstroke victims even after their body temperatures had been brought down to normal.[8] Now it is believed that certain mediators including endotoxins, cytokines, activated coagulation components are responsible?

A complete autopsy including histopathological and toxicological studies and at times microbiological studies may have to be undertaken before making a firm diagnosis of heat stroke. The significance of incidental findings have to be evaluated in the overall context of the final diagnosis. There will be anxiety amongst the relatives regarding the death, because in many instances the victims are young, healthy and often fit individuals. Bleeding from mouth and nose, skin bruising, loose motion, and hyperpyrexia may mislead not only the lay people but the clinicians as well. A correct diagnosis is also essential to prevent any recurrences especially in military establishments and athletic camps.

CASE REPORT

A 30-year-old healthy single Chinese lady collapsed while jungle trekking in Malaysia on a very warm and humid evening. She was slightly overweight and had joined a trekking group in an attempt to reduce her weight. This was her fifth trekking session and she had been very enthusiastic about the weight-reduction programme. She had started taking an herbal "slimming tea" and apparently had been passing plenty of urine since then. The trekking location was a hilly, jungle area and she had been trekking up a slope for about ninety minutes before she collapsed. Shortly before she collapsed her group members had noticed that she was lagging behind them. It took a little time before they discovered her. She was found in a collapsed and semiconscious state. They also encountered difficulty and delay in getting her to hospital. It was about two hours later before she was admitted to the University Hospital Kuala Lumpur.

On admission to hospital she was semi-conscious, not responding to painful stimuli, had a rapid thready pulse of 140/min, low blood pressure of 80150 mm Hg and an axillary temperature of 40°C. Her lips and skin were dry, pupils were contracted and reacted sluggishly to light. Fundoscopy revealed blurred optic discs. Irregular areas of skin bruises and cyanosed finger nailbeds were noted. The upper and lower limbs showed multiple linear grazed abrasions and contusions, which were injuries caused when she had to be carried through the difficult jungle terrain.

She was intubated and ventilated. In spite of intravenous fluid therapy her blood pressure remained low. Biochemical studies revealed renal impairment, elevated liver and cardiac enzymes and severe metabolic acidosis. Her haemoglobin was 17.1g/L which was suggestive of dehydration. The total white blood cell and platelet counts were within normal limits. Her coagulation profile was found to be impaired and she started bleeding through the mouth and nostrils. ECG showed a sinus rhythm and CT scan of the brain was unremarkable. Her axillary temperature remained around 39 to 40°C. In spite of active resuscitation she expired about eight hours after admission to hospital and around ten hours after she had collapsed.

Autopsy findings:

An autopsy examination was performed on the same day. The deceased was a well-nourished healthy looking, slightly obese young lady who weighed 71 Kg and was 155cm tall. She had multiple areas of grazed abrasions confined mostly in the lower limbs. There were also irregular areas of skin bruising scattered over the body but mainly over the front of her chest wall, inner aspects of both upper arms and around venepuncture sites. Her hydration appeared satisfactory. Blood-stained froth was seen at the nostrils and in the trachea and bronchi with patchy

areas of mucosal haemorrhage in the air passages. The lungs showed generalised blotchy subpleural haemorrhage and areas of collapse. Their cut surfaces appeared congested, oedematous and haemorrhagic. The heart was normal except for flame-shaped subendocardial shock haemorrhages seen mostly over the interventricular septum on the left side (Fig. 1). The oesophageal mucosa showed linearly placed streaky haemorrhages (Fig. 2). The stomach contained frank blood and the gastric mucosa was congested and haemorrhagic (Fig. 3). Spotty and blotchy haemorrhages were seen on the serosal surface of the entire small and large intestine. Both small and large intestines contained large amounts of blood-stained fluid and the mucosae were diffusely congested with areas of streaky haemorrhages. The brain was slightly oedematous and subarachnoid haemorrhage was seen over the right temporal area. Cut sections of both kidneys appeared pale but the cortico-medullary pattern was preserved. No other gross abnormalities were seen at autopsy.

Histology showed severe pulmonary oedema with extensive intra-alveolar haemorrhage and congestion of the blood vessels. The Martius-scarlet-blue stain did not reveal any microthrombi in the pulmonary capillaries. The lung architecture was maintained and no evidence of infection was seen. There was congestion of liver sinusoids. Kidney, heart and brain tissues appeared normal. Blood culture for microorganisms and toxicology for therapeutic drugs, drugs of abuse and common poisons gave negative results.

Based on the clinical history and the autopsy findings, the cause of death was concluded as due to heat stroke.

DISCUSSION

Heat stroke is an acute medical emergency and unless it is deteckd early and managed efficiently and effectively it may result in death or permanent organ damage.[10] Heat.stroke was originally described more than 2500 years ago. However, the association between high environmental temperature, hyperthermia and the clinical manifestation of heat stroke was not fully established until the middle of the nineteenth century.[11] Malamud et *al* in 1946 were the earliest who threw more light in the understanding of the pathophysiology of heat stroke. They established that heat stroke led to multiple-organ damage.[12] Despite the recognition of this serious condition for many years, the mortality is still remains high – varying from 10 to 80 percent.[13]

A delicate balance between production and dissipation of heat maintains thermal homeostasis.[1] The temperature of the normal human body is usually maintained at 37^{0}C (98.6^{0}F). T his value is, however, only approximate. There are physiological variations from person to person, and individual variations according to age, state of rest or physical activity, the time of the day and other factors. Normal heat regulation depends on the prevention of various physiological mechanisms responsible for the loss of heat such as sweating, adequate circulation and acclimatisation. The average human body is more vulnerable to an elevation than to a diminution of temperature. Under resting conditions, sweating is initiated when the environmental temperature reaches 30^{0}C. Under persistently unfavourable environmental conditions, the overall loss of urea, salts and other plasma elements can be considerable. Sweat loss is compensated for by decreased rate of urine formation and increased water intake. With a very high sweating rate, however, after 4-6 hours a condition of fatigue supervenes in the overworked sweat glands, sweat ceases, and the body temperature rises. The metabolic rate increases by 5-14 percent for each degree in body temperature.[14] Oxygen utilisation increases proportionately, and in contrast, the oxygen combining capacity of haemoglobin decreases. The greater stability of oxyhaemoglobin in association with alkalosis further impairs the release of oxygen to the tissues.[14] In severe hyperpyrexia the arterial oxygen saturation decreases by approximately 25 percent, and venous oxygen saturation increases in proportion.[15,16]

Systemic hyperthermia can be induced by four basic conditions: (1) an intolerable elevation of environmental temperature, (2) a rise in body temperature in conjunction 'with an endogenous disorder, such as an infection, (3) an accidental, unpredictable febrile state like drug fever, and (4) an overwhelming heat exposure to fire.[17] The human body responds to the elevation of the environmental temperature by reducing the production of heat and by increasing the dispersion of heat. Heat dispersion is achieved by peripheral vasodilatation and increased sweating. High relative humidity and lack of air movement hamper the sweating mechanism. Once sweating ceases the hyperthermia syndrome becomes manifest namely heat cramps, heat exhaustion, and heat stroke, the latter being a very serious clinical condition. The clinical

FIG. 1: Left ventricle showing extensive subendocardial shock haemorrhage, mostly confined to the interventricular septum.

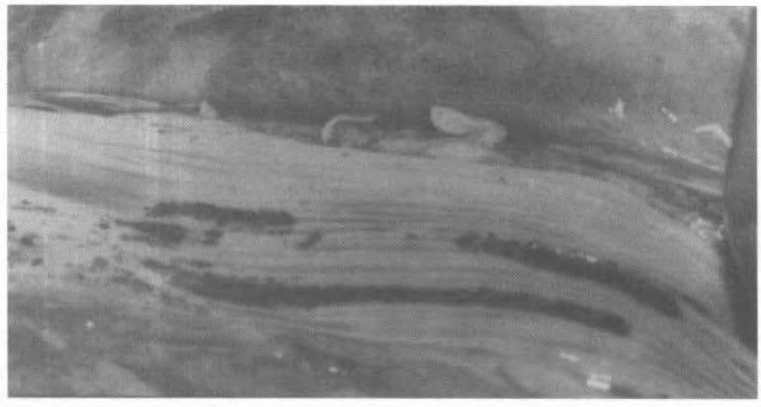

FIG. 2: Oesophageal mucosa showing linearly placed streaky haomorrhage.

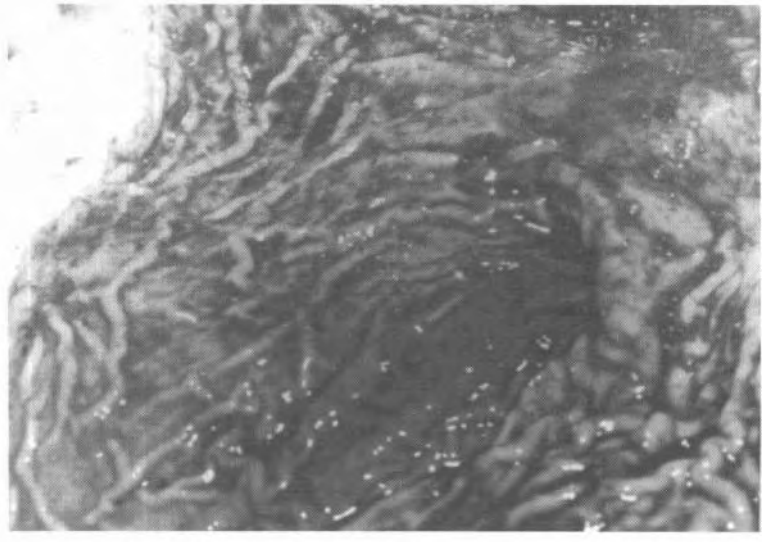

FIG. 3: Stomach containing frank blood. The gastric mucosa is congested and haemorrhagic.

manifestation of heat cramps and heat exhaustion are prominent enough to warn the individual to take precautionary measures. But heat stroke often manifests itself suddenly, and unless it is diagnosed immediately and treated effectively, may result in a fatal outcome.

Heat stroke may occur in young military recruits who are made to perform rigorous physical exercise during their early training period before getting acclimatised, particularly in tropical countries. In such situations the victims are often healthy and fit. Their physical fitness further contribute to their vulnerability, because in spite of harsh environmental conditions and severe exhaustion, they try to push themselves beyond their limits and may suddenly manifest by heat stroke, the later also called as "exertional heat stroke". Even multiple deaths during a single event have been reported amongst military recruits.[2] The old and frail, chronic alcoholism, chronic cardio-vascular disease, obesity and the use of drugs such as anti-cholinergic agents and diuretics too contribute to heat stroke, particularly in less harsh environmental conditions.[10] In temperate climates too several deaths due to heat stroke have been reported during summer months particularly during heat waves with associated high ambient temperature and humidity.[18] Heat stroke occurring purely due to high ambient temperature and humidity without actual physical exertion is called as "classic heat stroke".

In this case, the victim was slightly obese but otherwise in good health. Her skin was healthy without any skin rash. She wore a pair of white running shorts, white loosely fitting T-shirt made of thin material, brassiere and panty. According to information she had been passing a lot of urine after started taking "herbal slimming tea". This was her fifth trekking session and it was very strenuous. Others in the group had noticed that after ninety minutes of trekking along the hilly, jungle area, she was staggering and lagging behind and after a short while they found her in a collapsed state. There had been an undue delay in getting her to the hospital because of the difficult terrain. It was evident that owing to her over-enthusiasm in reducing her weight, she had resorted to various methods such as using diuretics ("herbal tea") and extensive physical exercise in the form of jungle trekking. She had got herself involved fully in the strenuous trekking programme without getting acclimatised. On that particularly day, unfortunately the weather too was very warm, humid and windless. However, she was suitably dressed for the occasion. It is reported that thick, dark, tight fitting clothes made of polyester materials worn by military recruits have contributed to heat stroke with many fatalities in a single episode, involving a route march along a tarred road on a very warm and humid day.[2] There was no sweat rash seen on the victim, which can further reduce sweating and hence contribute to the development of heat stroke. In tropical climates in particular, the authorities involved in military and athletic training are advised to treat sweat rash effectively, especially in the early stage of the training.[2] No details were available about her fluid intake. The "slimming tea" that she was taking with the resultant diuresis may have contributed to a negative fluid balance. She may have even reduced the fluid intake in the form of sweetened drinks, fearing additional calories. Adequate fluid have to be taken particularly in the initial stage of starting rigorous exercise, especially in tropical climates with high ambient temperature and humidity. Once the person is acclimatised then there is less risk of developing heat stroke. One concedes the fact that military and athletic training is exacting and designed to toughen individuals. However, it is essential that the programme is modified in its early stages and the rules made more flexible until the new recruits are acclimatised. As mentioned earlier physical fitness too sometimes contributes to this tragedy and therefore the young military recruits and athletes are more vulnerable. A spirit of competitiveness associated with youth, service discipline and the need to implicitly comply with orders of the instructors may force the trainees to continue with the exercise despite physical exhaustion. In this case of course her over-enthusiasm about weight reduction had pushed her beyond endurance. There were no underlying diseases or conditions that were detected in her which could have contributed to the tragic outcome.

Preventive aspects

Making a diagnosis of heat stroke is essential in order to prevent recurrences. Not only does it have legal implications, but it is also a matter of public interest and concern. The relatives will be anxious to know how death had occurred in an apparently healthy and fit individual. A visit to the scene, studying all the relevant documents such as the clinical notes, interviewing the persons involved such as trainers, colleagues and the survived victims form an important part of the investigation. All the contributory factors have

to be identified. This is achieved not only by a thorough study of the scene and other related factors but by performing a detail autopsy including all the possible investigations that may seem necessary.[19]

Training camps situated particularly in the dry areas should record the day temperature and humidity. On hot and humid days a more lenient schedule should be adopted. White or light coloured, loose fitting clothing made of thin cotton materials should be worn. Sweat rash when detected should be promptly treated. The quota of water on humid days should be increased and this becomes relevant in military training where the amount of water is restricted. Instructors should be more vigilant and not force any one who appears exhausted, especially in the early stage of the training programme. Instructors should be given special training to identify the prodromal symptoms of heat stroke, and be in a position to render first aid when required. A special heat stroke treatment room and clinic should be available in military and athletic camps on similar lines as was being done at Mina Al Ahmadi harbour in Kuwait to treat tanker crew.[20]

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