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"Review Study On - Heat Stroke"

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ABSTRCT-

Heatstroke is a severe heat-related illness involving an elevation in body temperature, typically but not always greater than 40 C. The patient has clinical signs of central nervous system dysfunction that may include confusion, ataxia, delirium, or seizures brought on after strenuous physical exertion or exposure to hot weather. This activity illustrates the evaluation and treatment of heat stroke and reviews the role of the interprofessional team in this condition. Heat stroke is a life-threatening injury requiring neurocritical care; however, heat stroke has not been completely examined due to several possible reasons, such as no universally accepted definition or classification, and the occurrence of heat wave victims every few years. Thus, in this review, we elucidate the pathophysiology, prognostic factors related to heat stroke and also summarize the results of current studies regarding the management of heat stroke, including the use of intravascular balloon catheter system, blood purification therapy, continuous electroencephalogram monitoring, and anticoagulation therapy.

Keywords- Heatstroke, aetiology, evaluation, treatment

Introduction

Heat-related illness is a spectrum of conditions progressing from heat exhaustion and heat injury to life-threatening heat stroke. Heat stroke is a clinical constellation of symptoms that include a severe elevation in body temperature, typically, but not always, greater than 40°C. Also, there must be clinical signs of central nervous system dysfunction, including ataxia, delirium, or seizures, in the setting of exposure to hot weather or strenuous physical exertion.[1] Risk factors include environmental variables, medications, drug use, and other medical comorbidities.[2]

Aetiology

It is important to differentiate where the patient is on the heat illness continuum. The signs and symptoms of heat exhaustion may present similarly, including cramping, fatigue, dizziness, nausea, vomiting, and headache. If progression to end-organ damage occurs, it then becomes heat injury. Finally, neurologic alteration distinguishes heat stroke from heat injury. There are 2 forms of heat stroke: classic and exertional. Classic heat stroke typically affects elderly individuals with chronic medical conditions, while exertional heat stroke affects otherwise healthy people who engage in strenuous exercise in hot or humid weather.[3]

MATERIALS AND METHODS-

Epidemiology

Estimating the public health impact of extreme heat is difficult because healthcare providers are not required to report heat-related illnesses. In the United States, from 2006 to 2010, there were at least 3332 deaths attributed to heat stroke.[4] However, these numbers are believed to be largely under-reported. Mortality correlates with the degree of body temperature elevation, time to initiate cooling, and the number of organ systems affected.[5]

Pathophysiology

Normally, thermoregulation is an extremely efficient process, with a mere 1°C change in core temperature for every 25°C to 30°C change in ambient temperature.[2] In the adapted state, heat-shock proteins repair the damage caused by hyperthermia. The body's ability to dissipate heat through increased cardiac output, vasoconstriction of splanchnic circulation, and sweating maintain the effective temperature range of these proteins. However, evaporative cooling becomes ineffective if environmental humidity is above 75%. The other methods of heat loss, including radiation, conduction, and convection, do not transfer heat well when the temperature outside the body exceeds the skin's temperature.[6]

Subsequent inadequate water repletion may lead to substantial electrolyte abnormalities. Primarily, normonatremia or hypernatremia dehydration follows. If severe enough, it may lead to hemorrhage, brain edema, and permanent brain damage. Rarely, hyponatremia occurs following overcompensation with hypotonic fluid repletion, as seen in marathon runners and other exertional heat stroke populations.[2] Hyperkalemia has been associated with heat stroke, which occurs when potassium is released from muscle breakdown or acidosis, causing a shift of

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potassium from cells to plasma. Potassium is a potent vasodilator in skeletal and cardiac muscle, and severe reductions in this electrolyte lead to cardiovascular instability and reduced muscle blood flow that predisposes to rhabdomyolysis.[1] Sequelae from rhabdomyolysis can range from mild hypocalcemia to acute renal failure. Hyperkalemia and hypocalcemia together can lead to cardiac conduction abnormalities, including QT interval prolongation and ST-segment changes, and, in rare instances, may lead to fatal cardiac arrhythmias.[6] There is also a range of coagulopathies associated with heat stroke, from simple activation of the coagulation cascade and fibrinolysis to fatal hemorrhage or disseminated intravascular coagulation. Endothelial damage from heat is thought to cause downstream effects that result in platelet aggregation and microvascular thrombosis predisposing to consumptive coagulation, which paradoxically causes bleeding when platelets get used quicker than the body's ability to produce them.[2]

History and Physical

Patients who present with heat stroke typically have vital sign abnormalities, including elevated core body temperature, sinus tachycardia, tachypnea, and widened pulse pressure, and a quarter of patients are hypotensive. Other associated presenting signs/symptoms may be weakness, lethargy, nausea, vomiting, dizziness, flushing, lung crackles, oliguria, excessive bleeding, and evidence of neurologic dysfunction.[1] Classic heat stroke patients often present with hot, dry skin because of a failure of the normal sweating response, also known as anhidrosis. Meanwhile, in exertional heat stroke, anhidrosis is an uncommon finding. Instead, prolonged sweating occurs following the cessation of exercise.[6]

Evaluation

The workup of patients presenting with possible heat stroke should include frequent monitoring of vital signs and rectal temperature and laboratory studies of CBC, CMP, PT/PTT, blood gasses, serum CPK, and urine myoglobin. Based on clinical judgment, some patients may also benefit from toxicology screening, a chest radiograph, and an EKG.[3] EKG changes may show ST depression, QT prolongation, and other T-wave changes consistent with ischemia. All patients with heat stroke have tachypnea and tachycardia. The arterial C02 levels often dip below 20 mmHg, and a quarter of the patients are hypotensive.[2] Medical reconciliation is crucial when evaluating suspected classic heat stroke patients, with emphasis on the recognition of diuretics, beta-blockers, and anticholinergic medications.

In classical heat stroke, respiratory alkalosis predominates, whereas exertional heat stroke may also have concomitant lactic acidosis. Electrolyte derangements are variable between the 2 etiologies, but commonly in exertional heat stroke hypocalcemia, hyperphosphatemia, and hyperkalemia, they reflect muscle breakdown that occurs. Rhabdomyolysis is more common in exertional than classical heat stroke, with a higher elevation of CPK markers reported. In classic heat stroke, AST and ALT elevations are the most common lab abnormalities reported.[2] Associated kidney injury, liver manifestations, and other end-organ damage may also occur in either presentation.[1]

Treatment / Management

Management of heat stroke includes ensuring adequate airway protection, breathing, and circulation. After ABCs, rapid cooling becomes the mainstay of treatment with ancillary management in response to other end-organ damage. Intubation for profound unconsciousness is rarely needed, as rapid cooling quickly improves the Glasgow coma scale.[6] Adequate rehydration is essential without over-correcting the sodium if derangements exist. It is mandatory to continually measure core temperature with a rectal or oesophageal probe, and cooling measures should be stopped once the temperature is 38 to 39 degrees Celsius. No definitive studies support any cooling method over another.[7] Ice bath immersion is the timeliest to reduce core body temperature, however, in older populations, it may not be realistic as cardiac monitoring may not be feasible and extreme agitation may hinder compliance.[8] Other common methods include ice pack applications to the groin or axilla and evaporative cooling using a fan with cool saline on patients' skin.

Several pharmacologic adjuncts also merit consideration in the treatment of heat stroke. Dantrolene is a skeletal muscle relaxer, shown to reduce heat production in sustained muscle contracture, and is useful for treating malignant hyperthermia. However, it has been shown that heat stroke does not affect patient outcomes.[9] A small study suggested that high-dose benzodiazepine may blunt the shivering reflex and decrease oxygen consumption, providing a theoretical benefit to patients. The problem is that heat stroke patients may be unable to compensate through mechanisms such as shivering.[10] Therefore, the universal use of benzodiazepines is not the current recommendation but could be tailored to the shivering, agitated patient. There is no role for antipyretics in the treatment of heatstroke patients and may be toxic to the liver.[1]

Differential Diagnosis

The common differentials include polypharmacy, toxic ingestions, meningitis, sepsis, neuroleptic malignant syndrome, serotonin syndrome, and malaria. A detailed medication review can exclude several of these disease processes. Except for heat cramps, neither muscle rigidity nor muscle clonus are signs of heat stroke and

can distinguish neuroleptic malignancy and serotonin syndrome. Travel history should be questioned, and exposure to malaria-endemic environments and the particular malaria species in that area should be evaluated. However, malaria, sepsis, or meningitis are not commonly present at the same elevations in core body temperature.

Prognosis

Patients presenting with heat stroke have high mortality depending on the etiology of the presentation. However, the death rate from exertional heat stroke is relatively low (3 to 5%) compared to classic heat stroke (10 to 65%).[5][4] The increased mortality rate is likely due to the higher prevalence of comorbidities and older age in the classic population. If immediate rapid cooling is successful, there has been a zero-fatality rate for young exertional heat-stroke patients.[4]

Complications

The sequelae of the insult may persist beyond the initial CNS dysfunction, involving injury to the gut, kidney, skeletal muscle, or other organ systems. Complications of heat stroke include acute respiratory distress syndrome, disseminated intravascular coagulation, acute kidney injury, hepatic injury, hypoglycaemia, rhabdomyolysis, and seizures.[5] Despite normalization of core temperature with cooling, many patients continue to display core temperature disturbances and multi-organ dysfunction. Research has shown that even reversible complications following heatstroke may take longer than 7 weeks to resolve.[11]

Deterrence and Patient Education

Prevention is the definitive treatment for heat stroke. It is essential to check on the elderly frequently, especially individuals who do not have access to air-conditioning. Wear appropriate clothing, avoid leaving children unattended in cars, and reschedule strenuous activities if there is hot, humid weather.[6] Individuals should seek shade if experiencing the signs and symptoms of heat stroke. Once the clinician has diagnosed heat stroke, rapid cooling should be initiated immediately with careful monitoring and reassessment. After treatment for exertional heat stroke, the patient must abstain from exercise for 7 days minimum. Follow-up in all cases should occur a week after presentation to screen for signs of end-organ damage.[12][6]

CONCLUSION-

Optimal treatment in heatstroke patients relies on early recognition and expedition of rapid cooling. If expecting a heatwave, a designated area with equipment for multiple patients should be available in the emergency department. The physician should understand what resources are available for cooling patients, as each facility may use evaporative, ice-bath, or other methodologies depending on its protocol. Careful coordination with nursing staff is necessary. Close monitoring of patients with a continuous rectal or oesophageal temperature probe is necessary, with careful communication to stop cooling measures at the desired 38 to 39°C. The physician should identify patients requiring cardiac monitoring, and additional consultants, including intensivists, should be contacted early in the management if other end-organ damage has occurred. The use of dantrolene has been shown in several small trials to be ineffective and is not recommended in treating heat stroke (Class 1).[9] The use of benzodiazepines in heat stroke may have merit for a patient who is agitated and shivering; however, empiric treatment is inadvisable until further studies are undertaken (Class II).[10][8] Finally, expedited rapid cooling has been shown in several RCT both directly and indirectly to be the most effective treatment to limit mortality in heatstroke.[8]

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