中文題目：運動熱中暑重症的治療-個案報告與文獻回顧

英文題目：Management of Severe Exertional Heat Stroke – A Case Report and Literature Review

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Introduction:

Hyperthermia is not equal to fever. Hyperthermia is elevation of core body temperature above the normal range due to failure of thermoregulation; and fever is induced by cytokine activation during inflammation. The most important causes of severe hyperthermia (greater than 40ºC or 104ºF) caused by failure of thermoregulation, included of heat stroke, neuroleptic malignant syndrome, and malignant hyperthermia. Each of these conditions may be associated with severe systemic complications and death. As core temperature rises, the preoptic nucleus of the anterior hypothalamus stimulates efferent fibers of the autonomic nervous system to produce sweating and cutaneous vasodilation. There are 2 type of heat stroke: Classic (nonexertional) heat stroke which is cause by impair thermoregulation or prevent removal from a hot environment; and exertional heat stroke, which is usually found in young healthy individuals who engage in heavy exercise during periods of high ambient temperature and humidity. We here would like to present a case with exertional heat stroke who suffered from status epilepticus, severe metabolic acidosis with rhabdomyolysis, coagulopathy due to DIC, and renal failure.

Case report:

A 26-year-old man with history of psychosis had been lived in local psychiatric care center for a long period. He was just discharged in recent 1 week with regular medical control. According to his family's statement, he had a part-time job about house building and go to work for the first day. However, the weather was hot and humid and he worked from 7am to 15pm without taking a rest. He was found more and more sleepy and then fainting. He was brought to our emergency department by ambulance ED by EMT. Initial GCS was E1V1 M2 and pupil size showed L/R:4/4 with positive light reflex. The body temperature was 43 degree C with general convulsion, hypotention, anbidrosis, irritable, and drowsy consciousness. Emergent brain CT revealed no ICH but brain swelling and arterial blood gas analysis revealed mixed respiratory alkalosis and and metaboic acidosis:PH 7.476, PCO_2 14.50, PO_2 111.20, HCO_3- 10.50, O_2 saturated 98.50%, blood analysis showed CPK: 452, PH 7.476, initially, deep urine color and oliguria was found after fluid hydration, rhabdomyolysis was impressed. CXR showed no active finding and EKG showed sinus tachycardia, blood routine showed no leukocytosis. After a lot of cooling method such as ice bags, electric fan, and ice water irrigation via NG tube and Foley

catheter, he was admitted to ICU.

After being admitted to ICU, intravenous cold fluid was given with 6-10L/hour and ice pack use for hyperthermia. Status epilepticus was noted and then oral intubation with BZD and Dilantin was prescribed, Mannitol was also given for increased intracranial pressure. Active bleeding from IV root and epitaxis was noted and then fresh frozen plasma plus cryo precipitate were given for protein supply and coagulopathy correction. Piperacillin/Tazobactam was given for hospital acquired pneumonia. Acute renal failure with anuria and severe metobolic acidosis was noted, emergent hemodialysis was arranged. Glucose water was given for hypoglycemia. Methylprednisolone 125mg was given for pancytopenia with septic shock. Followed serum CPK level was over 60000. After above treatment, general condition improved and consciousness became clear. After extubation, he was transferred to general ward with renal failure and regular hemodialysis.

Discussion:

Evaporation is the principal mechanism of heat loss in a hot environment, but this becomes ineffective above a relative humidity of 75%. The other major methods of heat dissipation — Radiation, Conduction, and Convection - cannot efficiently transfer heat when environmental temperature exceeds skin temperature. Hyperthermia will increase in oxygen consumption and metabolic rate, resulting in hyperpnea and tachycardia. If the body temperature is above 42ºC, oxidative phosphorylation becomes uncoupled, and a variety of enzymes cease to function, as a result, these patients are at risk of multiorgan system failure.

Physical examination may reveal cutaneous vasodilation, tachypnea, rales due to noncardiogenic pulmonary edema, excessive bleeding due to DIC. Evidence of neurologic dysfunction such as altered mentation or seizures may also be found. Lab data may show coagulopathy, elevated CK level, acute renal failure, respiratory alkalosis and metabolic acidosis, acute hepatic necrosis, and leukocytosis. About the prognosis, one series of 58 patients with heat stroke found an acute mortality rate of 21% and multiple organ dysfunctions were common upon admission, besides, infection were documented in over 50% of patients. Above presentations of heat stroke can almost be found in our patient and fortunately, he survived.

Conclusion:

For optimal outcomes, it is best to treat immediately with on-site whole body cooling if cardiorespiratory status is stable and then to transfer the runner for additional evaluation and care, because the main outcome predictor in exertional heatstroke is duration and degree of hyperthermia initially. Maintaining adequate hydration, avoiding heat exposure, wearing loose and light clothing,

and monitoring the exertion level should be notified to heavy workers. Rapid cooling treatment, fluid resuscitation, adequate sedation and brain image arrangement, coagulopathy correction and blood transfusion, and intubation with ventilator support should all be considered immediately in severe heat stroke, which may prevent death or permanent neurological injury.