**HISTORY/PHYSICAL**

**CHIEF COMPLAINT:** Found Down

**HISTORY OF PRESENT ILLNESS:** 55-year-old obese female with past medical history of diabetes and hypertension was brought in by EMS after being found unresponsive by her husband for an unknown amount of time on a late July afternoon in Bronx, NY. EMS reported that their apartment, located on the 7th floor, was scorching hot. Husband reported that there was no air conditioning in the apartment and that the last time he saw her was earlier that morning, when she complained only of a cough.

**VITALS:** T (RECTAL) 108.5°F P 139 BP 88/61 mmHg SPO2: 86% ON ROOM AIR, CBG 182mg/dL

**PHYSICAL EXAM:**
- GEN: no gross trauma GCS 7 (E1M2V4)
- HEENT: 2mm pupils, dry mucous membranes
- CHEST: B/L coarse breath sounds, left rhonchi
- HEART: Tachycardic, no murmurs
- ABD: Soft Non distended.
- EXT: +2 pulses in all extremities, no deformities
- SKIN: Dry, warm flushed skin

**LABS**

<table>
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<th>128</th>
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- AST:358
- ALT:98
- ALP: 63
- TSH 0.68
- ASA None
- APAP None
- Ammonia
- Lactic Acid 4.8
- INR: 1.3
- Troponin < 0.1
- CPK 1663
- 77mg/dL

**DIFFERENTIAL DIAGNOSIS**

- Septic Shock
- Heat Stroke
- Encephalitis
- Meningitis
- Hyperthermia
- Thyroid Storm
- Cocaine/Amphetamine
- Malignant
- Serotonin Syndrome

**BACKGROUND**

Heat Stroke (HS) is defined as rectal temperature greater than or equal to 106°F (41.1°C) along with change in mental status ranging from confusion, delirium, stupor to coma and/or convulsions. HS accounts for hundreds of deaths in United States annually, with 80% of the victims age 50 years and older. A study showed 54% increase in heat related deaths where hyperthermia was included as a contributing factor to death, between 1993 and 2003. Heat related deaths are preventable and mortality decreases with public health and healthcare workers preparedness.1-5

**DISCUSSION**

Response to heat stress is a dynamic balance between the mediators of inflammation, including endothelial cells, leukocytes, inflammatory cytokines, and endotoxins. Proinflammatory cytokines identified in HS include tumor necrosis factor (TNFα); interleukin (IL)-2, -6, -8, and -10; interferon-α and -β.6 In a study of 18 HS patients, circulating cytokine levels correlated with clinical HS severity index. Additional in vitro studies show that cooling delays the release of IL-1β, IL-6 and TNFα.

The brain is the most heat-sensitive organ in the human body.7 It has been shown that irreversible changes of neural cells start at approximately 40° C (104°F).8-14 The most important consequence of these changes are destruction of endothelial cells of the brain and leakage of serum proteins across the brain-blood barrier resulting in brain edema --- the most hazardous acute complication of pathologic brain hyperthermia.15 Additional studies show, a strong relation between heat-induced neuronal damage and edematous areas of the brain.16 Other experiments reveal that neurons can tolerate low temperatures of at least 30° C (86°F)16,17,18. Although there is limited human data, animal models have illustrated that halting early gene expression of proinflammatory genes and excitatory neurotransmitters via rapid cooling and maintaining therapeutic hypothermia (TH) [defined as core body temperature less than 35°C within 6 hours of hospitalization] plays a central role in preventing neuronal cell death. Further, TH also stabilizes the blood brain barrier and reduces CE by decreasing permeability to inflammatory cytokines and potential harmful substances such as free radicals and thrombin.8,9

**CONCLUSION**

To our knowledge, this is the first documented case of a successful outcome involving heat stroke complicated by cerebral edema that can be attributed by early aggressive rapid cooling measures. To date, evidence supports the use of hypothermia treatment in cardiac arrest patients and neonatal hypoxic-ischemic encephalopathy.9-20

However, hypothermia has not been proven to show benefit in patients with stroke and traumatic brain injury.21 Our patient was induced into a hypothermic state within 1 hour of presentation to our emergency department. As a result, future investigatory studies involving other neurological injuries (i.e. stroke, traumatic brain injury, heat stroke) should investigate a possible relationship between neurological outcome and duration of timing for inducing a hypothermic state.