A 66-year-old man was admitted with anasarca and congestive cardiac failure. He was started on intravenous diuretics and fluid restriction and lost nearly 35 kg of fluids in 13 days. He then developed hyperthermia and delirium. An extensive workup was inconclusive. Analysis of his inpatient fluid balance chart revealed the chance association of massive fluid loss and hyperthermia. Rapid diuresis-induced hyperthermia and volume contraction were considered, and his fluid balance was adjusted. Within 2 days, his temperature normalized and he regained consciousness. Hyperthermia and its causative mechanism are discussed in this article.

Keywords: Cardiac failure, causes, diuresis, fever, hyperthermia, pump failure, rapid diuresis
**DISCUSSION**

Temperature regulation is an intricate process masterminded by the hypothalamus [Figure 2].

Internal heat is generated via metabolism and muscle contraction. The excess “waste” heat is lost to the environment through radiation ($H_{\text{rad}}$), convection ($H_{\text{conv}}$), conduction ($H_{\text{k}}$) (sensible heat) (dry heat loss [DHL]), and evaporation ($H_{\text{evap}}$) (latent heat). With exercise, increasing metabolism, or increasing environmental temperatures, the first three methods (DHL) are limited and primary heat loss is through evaporation. The environmental WBGT (an index of heat stress) also influences the rate of $H_{\text{evap}}$.[1] The WBGT (a combination of air temperature, mean radiant temperature, absolute humidity, and air movement) is a widely used index of heat stress. As the WBGT increases, the heat stress increases. Thus, a thermo-neutral heat balance equation is one where heat production equals heat loss and is represented by the following equation:[2]

$$S = (H_{\text{m}} - W_{\text{e}}) \pm H_{\text{evap}} \pm H_{\text{conv}} \pm H_{\text{rad}} \pm H_{\text{k}} \pm H_{r}$$

Where $S$ = Change in body heat content

$H_{\text{m}}$ = Internal metabolic heat

$W_{\text{e}}$ = External work performed

$(H_{\text{m}} - W_{\text{e}})$ = Heat to be dissipated to the surrounding (“Waste heat”)

$H_{\text{evap}}$ = Evaporative heat loss from the body

$H_{\text{conv}}$ = Convective heat loss

$H_{\text{rad}}$ = Radiated heat loss

$H_{\text{k}}$ = Conductive heat loss

$H_{r}$ = Heat loss through respiration.

For normal heat balance, $H_{\text{in}} - W_{\text{e}}$ (“Waste heat”) = $H_{\text{evap}} + H_{\text{conv}} + H_{\text{rad}} + H_{k} + H_{r}$. When there is an imbalance either by increase in $H_{\text{m}}$ or a decrease in $H_{\text{evap}} + H_{\text{conv}} + H_{\text{rad}} + H_{k} + H_{r}$, then the body accumulates heat.

In cool environments, our excess heat load is dissipated to the environment by radiation and convection from our skin. With increasing workloads or higher environmental temperatures, evaporative cooling is the primary heat loss mechanism. Hence, when evaporative cooling is hindered by dehydration, clothing, increasing humidity, or poor air movement, human beings become vulnerable in high temperatures.

Hyperthermias are disorders of increased body temperature. These can be broadly classified as fever or hyperthermic states. Whereas fever is induced by an elevation of the hypothalamic set point and is aspirin responsive; in the other hyperthermic states, the hypothalamus and the temperature regulatory circuitry are overwhelmed. Hyperthermia classically is defined as a core body temperature $>40^\circ\text{C}$–$40.5^\circ\text{C}$ along with central nervous system dysfunction.[3-5]

True hyperthermia is due to (1) excessive heat production as in exercise hyperthermia; (2) impaired heat dissipation as in
dehydration, environmental heat exposure, skin diseases, or anhidrosis; or (3) pathological or pharmacological factors that impair thermoregulatory mechanisms [Figure 3].

Surprisingly, CCF is associated with an increased basal metabolic rate (BMR) of approximately 18% more than normal, which contributes to cardiac cachexia. Furthermore, the BMR increases in direct proportion to the severity of the heart failure.

Furosemide has been implicated in hyperthermia in rare cases, due to either drug fever or dehydration in a neonate and as an idiosyncratic reaction in an adult. However, this magnitude of dehydration and the correlation with rising temperature have not yet been documented.

The rate of heat loss at rest in an air-conditioned room is approximately 370 kJ/h (sensible heat 210 kJ/h and latent heat 160 kJ/h). Usually, heat-related illness is uncommon with a WBGT <82°F (28°C). Although our ICU was relatively “cold” with a WBGT of approximately 78°F (25.5°C), we presume that our patient developed hyperthermia by the following mechanisms:

1. Increased heat production ($H_m$) as CCF is associated with a raised BMR.
2. Inability to increase heart rate and stroke volume by the failing heart “pump failure,” leading to internal heat accumulation
3. Intravascular volume depletion secondary to massive diuresis – reduced heat carried to periphery for dissipation
4. Diuresis-induced peripheral vasoconstriction
5. Immobility and reduced behavioral adaptive responses worsening the spiral of heat accumulation and hyperthermia.

Hence, in our patient, the heat balance equation tilted toward heat retention “by increased $H_m$ and reduced $H_{\text{evap}}$ with the other five variables remaining constant. The massive forced diuresis, underlying “pump” failure, and general debility contributed to accumulation of a “heat load” and hyperthermia. Physicians should consider an acutely impaired fluid balance as a possible cause of fever or hyperthermia in the ICU. The traditional “start low, go slow” technique of diuresis and close attention to the fluid balance are preferable even in severe cardiac failure, to avoid such a calamitous complication.

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**Conflicts of interest**

There are no conflicts of interest.

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