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The First Case Report of Accidental Severe Hypothermia from Tropical South India

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Abstract: Severe accidental hypothermia is a life threatening emergency associated with a high morbidity and mortality rate when untreated. Although common in the cold countries, hypothermia has never been reported in South India which has a tropical climate. We report, what we believe is the first documented case of severe accidental hypothermia (core temperature of 26.3° C) from Tamilnadu in a 40-year-old male patient. The homeless man was found on the street, drenched in the rain, intoxicated, with his clothes removed and curled up to escape the cold. He was saved by re-warming methods alone. In this case report we will review the physics of heat loss and effects of hypothermia on the human body and the various electrocardiographic changes in hypothermia with particular interest to the J wave, its genesis and its occurrence in other conditions.

Key words: J Wave · Giant Osborn Waves · Paradoxical Undressing · I_{to} Current

INTRODUCTION

On a cool rainy night in December 2013, a 42 year old male was brought to the ER by bystanders. He was a homeless man found on the roadside, drenched in the rain with most of his clothes by his side, wearing only his undergarments.

When he was brought to the ER he was semiconscious, disoriented and cold to touch. His pulse was feeble at a rate of 25/min, blood pressure measured 70/40 mmHg, respiration was slow at 6/min and blood glucose measured 187 mg/dl.

Temperature recorded with a digital rectal thermometer showed 26.3° C. There was a faint odor of alcohol in his breath. Heart sounds were normal and chest was clear on auscultation. His limbs were rigid and pupils dilated & sluggishly reactive to light. A diagnosis of accidental hypothermia facilitated by alcohol was made and he was intubated to protect the airway. He was treated with active external and internal rewarming with blankets and warm saline. His vitals and neurologic status improved gradually He was extubated after 6 hours when he became fully conscious and oriented. History and examination did not reveal any abnormality. Serum electrolytes,TSH and cardiac enzymes were within normal limits. He made a full recovery without any adverse effects and was discharged after 2 days. Serial ECGs revealed following changes.

DISCUSSION

Hypothalamus is the centre for temperature control in our body. Human beings are homoeothermic maintaining a core temperature of 36.5-37.5°C in spite of fluctuations in environmental temperature. The main response to changes in environmental temperature is behavioral modifications in the form of appropriate dressing and assumption of body positions to facilitate or diminish heat loss. The human body is better designed to face high rather than low temperature.

Death from accidental hypothermia occurs throughout the world. While typically associated with regions having severe winters, hypothermia can also occur in areas with warm climate. Tamilnadu lies in a tropical climate zone with hot sultry weather most of the year. The average low temperature during the coldest month of January is around 21°C leading to no reported cases of accidental hypothermia from this part of the world [1]. But various factors can interact to produce even profound hypothermia in tropical climate.

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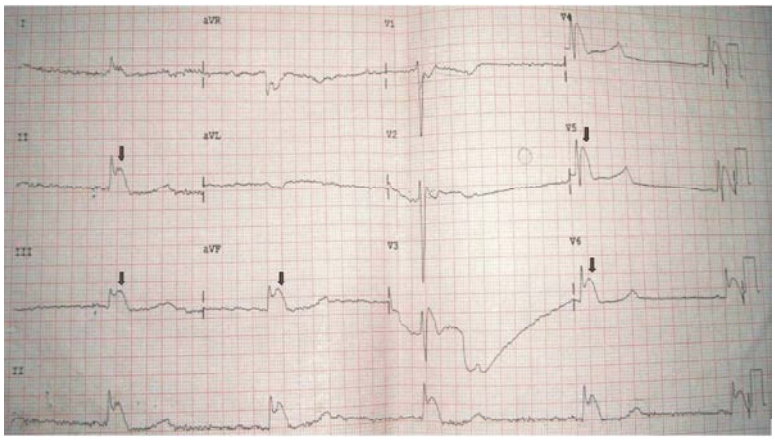

Fig. 1: ECG at presentation (26.3° C)
ECG showing severe sinus bradycardia at rate of 28/min, prolonged QRS interval (140ms) with giant Osborne waves (Arrows) more prominent in Leads II, III, aVF and V3-V6, prolonged QT_c interval (640ms)

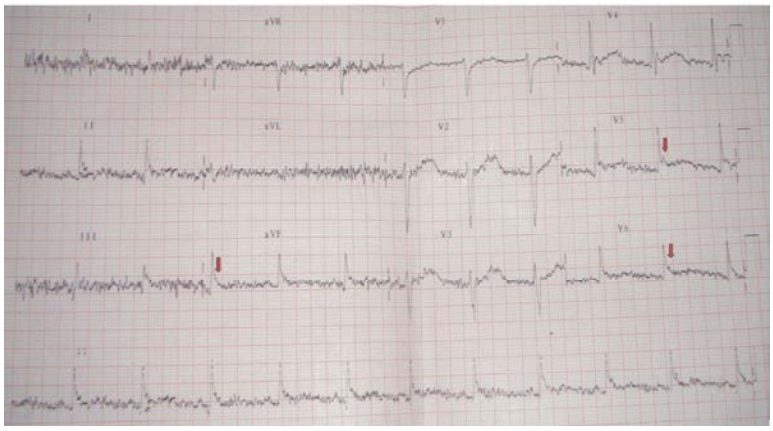

Fig. 2: ECG 3 hours after re-warming (31° C)
Note increase of heart rate to 65/minute, narrowed QRS(100ms), reduced amplitude of Osborne waves and pronounced shivering artefacts.

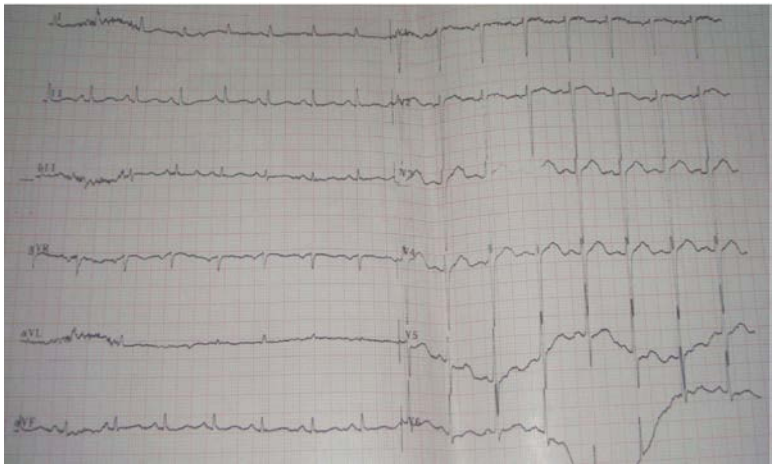

Fig. 3: ECG after resuscitation 6 hours later (36.5°C)
Note the disappearance of shivering artefacts and Osborn waves. QT_c is still prolonged (540ms) while QRS interval has normalized (90ms)

Hypothermia: Hypothermia is defined as a core temperature below 35°C. It is broadly categorised into:

- Mild hypothermia (32-35°C)
- Moderate hypothermia (28-32°C)
- Severe hypothermia (<28°C)

Causes of Hypothermia: The causes of hypothermia can be divided into 3 categories:

Accidental Hypothermia: It occurs due to extreme cold in a patient with normal thermoregulatory function and is predisposed by extremes of age, malnutrition, alcohol or exhaustion before cold exposure [2].

Endogenous Hypothermia: It is caused by

- Defects in thermoregulatory function due to brain injury / stroke / degenerative disorders.
- Metabolic derangements due to hypothyroidism / hypoglycemia.
- Excessive heat loss from skin due to erythroderma / extensive burns.

Iatrogenic Hypothermia: Induced deep hypothermia is a recognized form of neuroprotection in patients undergoing cardiac surgery. Induced mild hypothermia is used in patients resuscitated from out-of-hospital sudden cardiac arrest. It has been proven to improve neurologic outcomes after recovery [3].

Of these, accidental hypothermia is the most severe form and clinically most relevant. Majority of clinically significant hypothermia occur due to environmental insult like prolonged exposure to low temperatures without adequate clothing or during cold water submersion.

How Hypothermia Occurs In Warm Climate Areas?

In a report from Moran DS et al from the Israeli Defence (Subtropical region), 76% of hypothermia cases occurred in the winter, 10% in spring, 13% in autumn and 1% even in summer[4]. So hypothermia is surprisingly common in warmer seasons as well. To understand this, we review the physics of heat loss from our body. Heat loss from the human body occurs by the following mechanisms [5]:

Radiation: 60% of heat loss occurs through radiation in form of infrared rays emitted from the human body.

Conduction: It refers to heat transferred from the body to the surrounding air. Air is a poor conductor of heat and hence it contributes only minimally to heat loss.

Convection: It is more effective than conduction because the warmed air around the body is continuously replaced by colder air due to flowing currents. Conduction and convection together contribute to about 15% of heat loss.

Evaporation: The heat lost from evaporation of sweat contributes to about 20% of heat loss.

Respiration: It also contributes to a minor portion of heat loss.

Instead of air, when the body is surrounded by water, heat loss is greater due to the higher thermal capacity of water. A body submerged in cold water loses about 50% of heat by conduction. When flowing water current is involved there is even greater heat loss by convection. This accounts for the efficacy of a cold shower in rapidly cooling a patient with hyperthermia [6].

Rain drops that reach the ground actually begin as ice crystals formed in sub-freezing temperatures in higher layers of the atmosphere. As precipitation passes through the various layers of air, ice melts and falls as rain. The exact temperature of rainwater is determined by that of air the droplets fall through but it is always lower than ambient temperature.

Wind chill is the temperature perceived by the body when wind speed is combined with ambient temperature. When the speed of wind increases, heat is lost more rapidly from body surface causing core temperature to drop. For example, at 40°F ambient temperature, a wind blowing at a velocity of 40mph produces a temperature of 27°F which can lead to frost bite within 30 minutes.

So when the following four factors are present it can lead to cold stress even in warm climate-cold temperature, cold wind, cold water and humidity. Hence diving in cold water without a proper suit or high wind velocities on a cold rainy day can lower body temperature profoundly even in warm climates, particularly in predisposed individuals.

Effects Of Hypothermia: During cold stress, the initial response of the body is to generate more heat by involuntary shivering. Hypothermia occurs when heat production is overcome by excessive cold, especially

when energy stores are depleted. Initially, consciousness, breathing and circulation are intact but gradually become impaired as the body cools. Below 32°C pulse slows, shivering ceases and muscles become rigid and sensorium worsens. Below 28°C some individuals engage in *paradoxical undressing* like our patient. Hypothermia also causes diuresis and hypovolemia, respiratory and metabolic acidosis, hyperglycemia and coagulopathy [7].

Temperature Measurement in Hypothermia: Among the vital measurements, the one that is often overlooked in the ER is temperature. The usual range of readings on clinical mercury thermometers is 35 to 43°C which are better designed to measure fever and hyperthermia than hypothermia. Hence in patients with hypothermia special thermometers are required for accurate temperature measurement. Temperature recorded using a pulmonary artery catheter is considered the gold standard for core body temperature but it requires an invasive procedure. The more commonly used method for measuring core temperature is rectal thermometer. But the rectum is an isolated compartment and rapid temperature fluctuations do not reach it quickly and hence its temperature lags behind. The measurement is also influenced by impacted stool. Studies show that rectal temperature may differ to up to 11°C from core temperature during induced hypothermia [8]. Esophageal temperature is easy to measure during therapeutic hypothermia and it is reported to have better correlation than rectal temperature. Temperature measured from the urinary bladder using a Foley catheter with an integrated temperature sensor is also reliable.

ECG Changes in Hypothermia: Hypothermia can produce a multitude of changes in the ECG. Sinus tachycardia occurs initially followed by appearance of shivering artefects. It is followed by sinus bradycardia and progressive lengthening of the PR and QT interval and QRS duration. J waves appear below 32°C and progressively increase in amplitude. Atrial ectopics and arrhythmias are common below 32°C. Ventricular fibrillation and asystole can occur below 28°C.

J wave (Osborn wave): A positive deflection (>1mm) at the QRS-ST junction in the ECG with a dome or hump configuration in at least two consecutive beats is called J wave. It was first observed in a patient with hypercalcemia by Krauss and later in hypothermia by Tomazewski. But it is widely known as "Osborn wave" in honor of his extensive work on cardiac effects of hypothermia in anesthetized dogs. It is also known as "camel-hump sign", "late delta wave", "K wave" and "current of injury"(9). It is found in 80% of patients with temperature < 30°C. It is prominent in leads in facing the left ventricle and in the inferior leads. It disappears with rewarming but can persist for 12-24 hrs after restoration of body temperature to normal. It is also reported in other conditions like Brugada syndrome, early repolarisation variants, hypercalcemia [10], brain injury [11], cerebrovascular accident [12], idiopathic ventricular fibrillation, vasospastic angina [13] and after resuscitation of cardiopulmonary arrest [14].

Genesis of J wave: In 1953, Osborn postulated acidosis induced by hypothermia as the cause of J wave. Later various other factors like delayed ventricular depolarization, early ventricular repolarisation and anoxia were considered as causes. In 1988, Litovsky and Antzelevitch proposed a difference in the distribution of 4-Aminopyridine sensitive transient outward potassium (I_{to}) current between the ventricular epicardium and endocardium as the physiological basis for J wave [15]. During ventricular repolarisation, hypothermia increases the epicardial potassium current relative to the current in endocardium. When the effect of hypothermia on action potential was studied, an increase in the amplitude and width of the notch during early repolarisation phase was recorded from the epicardium but not from the endocardium. This transmural voltage gradient is reflected on ECG as the J wave. Hypothermia also decreases conduction velocity and unmasks the latent J wave by moving it out of the QRS complex.

Clinical Significance of the J Wave: Osborn considered the occurrence of J wave during hypothermia a bad prognostic sign as it often heralded ventricular fibrillation. Other investigators have either confirmed or refuted this association leading to a controversy. The risk of arrhythmia in the presence of J wave depends on the underling condition and must be considered individually. The J waves observed in hypercalcemia and neurological disorders are not usually associated with arrhythmias. However, the J waves observed in other situations have been shown to be linked to ventricular fibrillation.

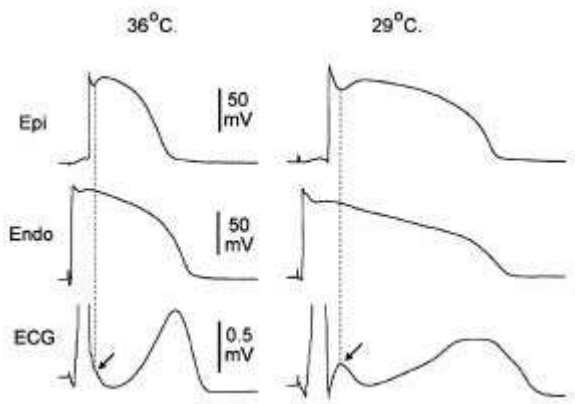

Fig. 4: Effect of temperature on action potential and ECG (adapted from: Cellular basis of electrographic J wave. Circulation, 1996; 93(2): 372-379) [15]

In addition to hypothermia, there are three clinically important J wave associations:

- Brugada syndrome is a genetic disorder due to loss of function mutations in sodium channels and is characterized by an apparent right bundle branch bock pattern with ST-segment elevation in V1-3 in absence of ischemia, electrolyte abnormalities or structural heart disease. Type 1 pattern with a J wave, coved ST-segment and terminal T wave inversion has the highest risk of sudden cardiac death(SCD). The magnitude of J point elevation has a direct correlation to the symptoms and SCD [16].
- Inferolateral early repolarisation pattern is characterized by an r′-wave or a slurred R-wave in at least two of the inferior or lateral leads or both. Several population based case-control studies have revealed that patients with idiopathic ventricular fibrillation (VF) have higher incidence of early repolarisation. Also, greater amplitude of J wave is associated with higher incidence of VF. It is also found that J point or ST-segment elevation itself is not a risk factor for mortality in absence of apparent J wave or slurring of terminal QRS [17].
- Athletes have a higher prevalence of asymptomatic J point elevations and J waves. But they are generally considered as benign variants and are independent from athlete heart syndrome diagnosed by echocardiography [18].

Although the mechanism of J wave production in hypothermia is different from Brugada syndrome and early repolarisation, Osborn waves may also have some arrhythmogenic potential leading to VF and SCD.

CONCLUSION

Even though Tamilnadu has a hot and sultry climate most of the year, environmental factors can interact to produce life threatening hypothermia under favorable conditions. In our inebriated patient, cold ambient temperature, high wind velocity and rapid convectional heat loss from rain water produced a never before reported profound hypothermia in South India. Early identification and appropriate management lead to his rapid recovery without any adverse consequences.

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