## CASE REPORT

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# Fatal Heatstroke in a Young Woman with Previously Undiagnosed Hashimoto's Thyroiditis

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**ABSTRACT:** Heatstroke represents the most severe form of the heat-related illnesses. Potentially fatal, heatstroke most often affects the elderly, obese, or chronically ill. Thyroid disease, which may interfere with the normal regulation of body temperature, has not previously been reported in cases of heatstroke. A fatal case is reported in a young woman discovered unconscious in a sauna who was found to have preexisting Hashimoto's thyroiditis on subsequent autopsy. The diagnosis of hypothyroidism in heatstroke rests on clinical information and morphologic observations. This case underscores the importance of evaluating the thyroid in unusual cases of heatstroke.

**KEYWORDS:** forensic sciences, heatstroke, heat-related illness, sauna, Hashimoto's thyroiditis, autopsy

Death due to overexposure to heat is not an uncommon cause of death. More than 700 deaths in Chicago during a heat wave in July 1995 were attributed to the high temperature (1). Risk factors for the development of heat-related deaths include: advancing age, obesity, social isolation, debilitation, chronic illness, medications interfering with thirst recognition or sweat production, and alcohol abuse (2).

Hypothyroidism has not previously been reported in cases of heatstroke. The signs and symptoms of hypothyroidism usually occur insidiously; it may take years before being clinically recognized. Nascent hypothyroidism may be manifested by cold intolerance and lethargy; these may lead patients to seek extraordinary measures to maintain comfort. Furthermore, the physiologic adaptations to hypothyroidism may hinder appropriate response during heat stress.

A fatal case of heatstroke is reported in a young woman with previously undiagnosed Hashimoto's thyroiditis. This case illustrates the importance of evaluating the thyroid in cases of heatstroke.

### Case Report

A 31-year-old Caucasian woman presented to a northern New England emergency room in midwinter unconscious with a tympanic temperature of 40.7°C.

One year previously she sustained a closed head injury in a motor vehicle accident. In the weeks following the accident she suffered sporadic seizures that eventually abated; she no longer required anticonvulsant therapy. After three months of rehabilitation she returned to an active lifestyle that included morning visits to a health club.

On the morning of the day of her admission she lifted weights and attended a water aerobics class. As part of her routine she changed her clothing in the sauna; family members revealed she frequently ''felt cold'' and therefore sought the warmth of the sauna. She was found in the sauna approximately three hours after entering, unconscious, and fully dressed. Subsequent examination of the sauna revealed it to be in normal working condition. Cooled in the emergency room, her temperature fell to 37.6°C. Rhythmic stiffening of her upper extremities was interpreted as seizure activity, and she was treated with phenytoin. Over the course of the next three days, she progressively developed liver failure and disseminated intravascular coagulation; she died on hospital day #4. She never regained consciousness.

The patient had no known history of thyroid disease. Family history was remarkable for the mother having Hashimoto's thyroiditis and glucose intolerance.

### Autopsy Findings

A postmortem examination was performed four hours after death. The patient was a well-developed, well-nourished woman. Findings related to heatstroke were found in most organs. In the heart, occasional myocardial fibers contained contraction bands and showed early coagulative necrosis. There was no atherosclerosis. Small areas of hemorrhage were present in the subendocardium. The lungs were diffusely hemorrhagic, each weighing 370 g. Both pleural cavities contained serosanguinous effusions, averaging 1750 mL each. The weight of the liver was 1230 g; the capsule was dull and wrinkled. Massive centrilobular necrosis, a moderate degree of fatty change, and congestion were present on histologic examination (Fig. 1). The kidneys contained early tubular necrosis. The spleen weighed 260 g and was congested.

The brain weighed 1200 g. A small calcification identified on the superior lip of the sagittal suture was attributed to the previous motor vehicle accident. Ischemic neuronal changes due to the thermal insult were found primarily in the hippocampus, frontal gyrus, and cerebellar vermis. An area of glial scarring was present in the anterior and lateral aspect of the hippocampus, corresponding to her previous seizures.

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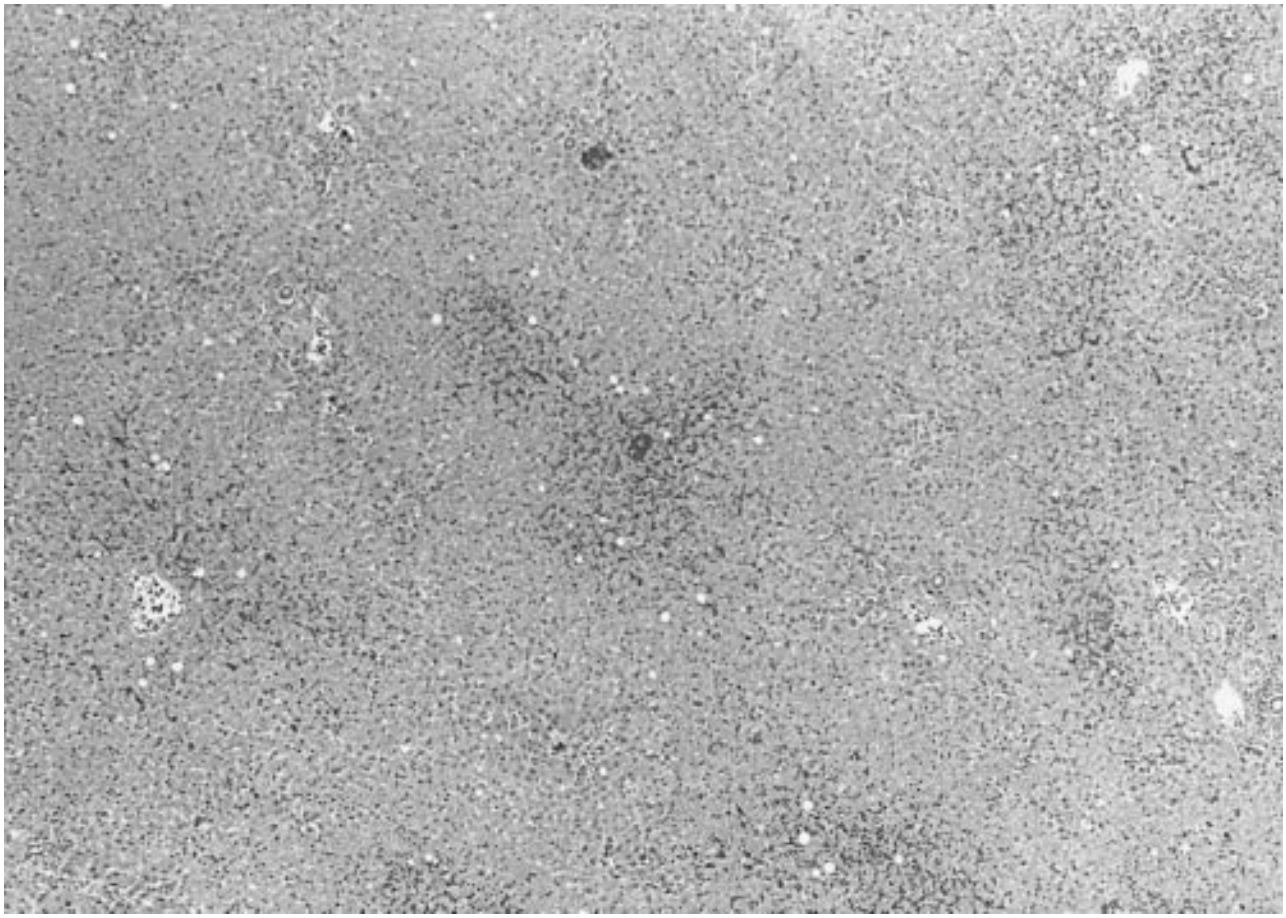

FIG. 1—*Massive centrilobular necrosis of liver.*

The thyroid, 120 g, was asymmetrical and moderately nodular. There were no masses. Microscopic examination revealed extensive lymphoplasmacytic infiltration with germinal centers, destruction of thyroid follicles, moderate to severe fibrosis, and Hürthle cell change (Fig. 2).

## Discussion

Heat-related deaths are not uncommon. During a July 1911 severe heat wave in New England, more than 1100 people died in Massachusetts with heat being listed as the cause of death; total mortality for the month exceeded the normal by 1400 (3). People at risk of heatstroke include the elderly, obese, alcoholics, and those with atherosclerosis or who take drugs that interfere with thirst recognition or sweat production, such as haloperidol, sedatives, antiparkinsonian agents, and phenothiazides.

Heatstroke is the most serious of the heat disorders and is characterized by elevated temperature, usually above 41.1°C, hot, dry skin, and central nervous system signs (4). Central nervous system signs range from lethargy to coma, and generally, higher temperatures at presentation correspond to more profound central nervous system manifestations. The greatest risk to life occurs in patients who present with coma (5).

Milder forms of heat disorders include heat cramps and heat exhaustion. Heat cramps are painful, sustained muscle contractions of heavily exercised muscles and occur primarily in the physically fit. Heat exhaustion, also known as heat prostration, is probably the most common of the heat disorders. It represents a failure of cardiovascular response to high temperature and is due to dehydration. It usually develops before prolonged exposure to heat; body temperature may be subnormal, normal, or slightly elevated. While a clear distinction from heatstroke may be nebulous, heat exhaustion is characterized by intact mental function.

Heatstroke is classified into exertional and classic forms. Exertional heatstroke occurs sporadically and in previously healthy individuals who are subjected to severe environmental thermal stress, typically while performing strenuous labor such as exercising. Classic heatstroke, on the other hand, occurs in older individuals in epidemic form with lesser degrees of exposure (6).

While heatstroke occurs most frequently during periods of hot climatic conditions, cases have occurred in saunas, coal mines, operating rooms, next to heaters, and in infants excessively swathed in blankets (7–11). The ambient temperature of saunas, including the case reported herein, may often exceed 100°C and makes for an intense heat load, particularly in a cool environment such as the northern U.S. in the winter. Clothing, such as those found on the patient at discovery, also adds to the risk of heatstroke in reducing the flow of convection air currents.

The basic pathophysiologic mechanism of heatstroke is the failure of the body to maintain normal body temperature. As the body temperature rises to temperatures above 41°C, there is generalized vasodilatation, increased cardiac output, and redistribution of blood away from the splanchnic organs to the skin in order to maximize

heat loss. This effectively reduces the blood volume. As the metabolic rate and oxygen demand increase, cardiac output increases. Eventually, the heart begins to fail, and patients suffer from high output failure and distributive shock.

Virtually no organ system is spared in heatstroke. The cellular changes are not only due to the heat load but also due to anoxia from shock. The effects on the brain are the most conspicuous and consist of progressive degeneration of the neurons with replacement by glia (12). The effects of heatstroke on the liver also have been studied considerably; changes in the liver progress from initial cloudy hepatocyte swelling to centrilobular necrosis at 16 hours after initial hyperthermic insult (13).

While the majority of the cellular alterations in heatstroke are due to a combination of anoxia and heat load, the features incidentally found in the thyroid in this case connote previous disease. The classic patient with Hashimoto's thyroiditis is a middle-aged woman with goitrous enlargement of the thyroid gland associated with hypothyroidism. Clinically, approximately one-half of patients with Hashimoto's thyroiditis are either hypothyroid at presentation or become so on follow-up (14,15). The diagnosis is generally made using clinical features and laboratory data; histologic confirmation is not necessary. In fact, histologic features in thyroiditis do not necessarily correlate with thyroid function (16).

The postmortem diagnosis of hypothyroidism is fraught with difficulty, as studies for thyroid hormones during this period are unreliable (17). Thyroxine values tend to fall erratically to the range of hypothyroid individuals in the postmortem period (18). Similarly, blood taken from patients shortly before death may also show low thyroid hormone levels. Bonell (19), in a study of 40 autopsy patients not dying of sudden cardiac death, showed evidence of hypothroxinemia in all patients within three days prior to death. Therefore, low thyroid hormone levels, either pre- or postmortem, in deceased patients must be interpreted with caution; they need not be indicative of hypothyroidism.

Postmortem thyroid stimulating hormone levels, on the other hand, tend to be fairly stable in the euthyroid patient for 24 hours. Theoretically, the value of TSH should predict hypothyroidism; however, its stability in the malfunctioning thyroid remains to be seen (18). Furthermore, O'Malley et al. (20) demonstrated that increases in body temperature of hypothyroid patients are accompanied by a decreased TSH level, obviating its use here. Thyroid hormone studies were not performed in this case. Nevertheless, given her clinical symptoms of cold intolerance, family history, and thyroid findings, one can speculate that hypothyroidism led to her ultimate demise.

The body's response to hypothyroidism is diametric to that in hyperthermia. In patients with hypothyroidism, thermogenesis is decreased, owing to a diminished basal metabolic rate. Essential to the hypothyroid patient is conservation of heat; and in defense, there is intense skin and subcutaneous vasoconstriction (21). This leads to some of the recognized clinical manifestations of hypothyroidism, such as cold, dry skin and cold intolerance. As a result of the intense vasoconstriction, one of the primary mechanisms of heat loss, sweating, is hampered. In conditions of high temperature,

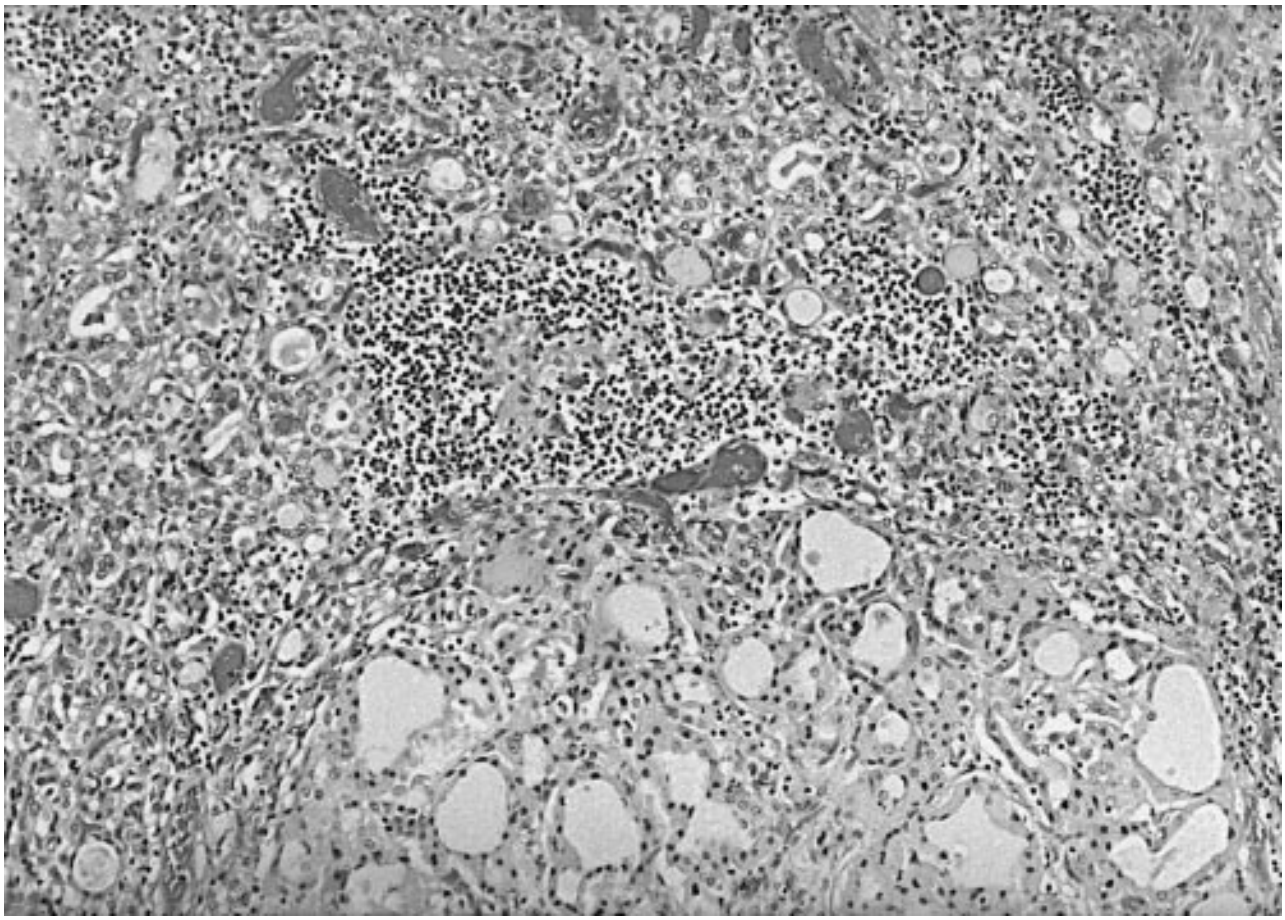

FIG. 2—*Extensive lymphoplasmacytic infiltrate of thyroid with fibrosis, follicular destruction, and Hürthle cell change.*

therefore, rather than dissipate heat via sweating, hypothyroid patients may continue to conserve heat, or, at least, have an attenuated heat loss. Furthermore, decreased adrenergic activity in hypothyroidism leads to decreased cardiac output, again interfering with the ability to effectively lose heat.

To the best of the author's knowledge, the physiologic adaptations of hypothyroid individuals have not been studied during episodes of intense heat stress. It is likely that there is some attempt to lose heat, through vasodilatation, sweating, and increased heart rate; however, it is probably diminished. Further studies may show the capacity to lose heat to be related to the degree of hypothyroidism.

Finally, it appears unlikely that seizures precipitated heatstroke in this case; they were more likely the manifestation. Central nervous system signs consummate the definition of heatstroke. Despite having a remote history of seizure disorder, she no longer required medication and was free of seizures for at least nine months. While an inciting seizure cannot be entirely ruled out, it appears more likely that the recognized seizure activity in the emergency room was simply the exhibition of heatstroke.

Previous reports of heatstroke have identified old age, debilitating illness, alcoholism, and certain medications as risk factors for death. Hypothyroidism and its resultant physiologic adaptations, however, have not been implicated. The status of the thyroid should be assessed in cases of heatstroke, as it may play a role in the development of heatstroke in hypothyroid individuals. The diagnosis must be made utilizing clinical history and gross and microscopic findings; thyroid hormone levels may not be reliable. This case adds to the list of natural disease risk factors for heatstroke.

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