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PATHOMORPHOLOGICAL CHANGES CAUSED BY EXPOSURE TO EXTREME TEMPERATURES: A FORENSIC AND HISTOLOGICAL ANALYSIS

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Abstract

Background: Exposure to extreme temperatures remains a significant cause of injury and death worldwide.

Objective: To analyze morphological and histological changes associated with thermal burns and frostbite in forensic practice.

Methods: Histological examination of skin and internal organs obtained from fatal cases of exposure to high and low temperatures was performed.

Results: High-temperature exposure resulted in coagulative necrosis, vascular congestion, and systemic organ damage. Low-temperature exposure caused vascular spasm, ice crystal formation, ischemia, and characteristic gastric hemorrhages (Wischnewsky spots).

Conclusion: Distinct histopathological patterns allow reliable differentiation between hyperthermia- and hypothermia-related deaths.

Keywords: Thermal injury, frostbite, hyperthermia, hypothermia, forensic pathology.

Introduction

Extreme temperature exposure is a well-recognized cause of both accidental and occupational mortality. According to the International Classification of Diseases (ICD-10), thermal injuries and frostbite are classified under codes T20–T35. Despite standardized classification, morphological differentiation remains crucial in forensic diagnostics. Classification of Thermal and Chemical Burns According to ICD-10 In the ICD-10, thermal and chemical burns are classified as follows: T20–T32. Thermal and chemical burns T20–T25. Thermal and chemical burns of external body surfaces, specified by anatomical location T26–T28. Thermal and chemical burns of the eye and internal organs T29–T32. Thermal and chemical burns of multiple and unspecified body regions

Included:

Thermal burns
Burns caused by electrical heating devices
Electrical burns
Flame burns
Burns from hot air and hot gases
Burns from hot objects

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Lightning injuries
Radiation burns
Chemical burns
Scalding injuries
Excluded:
Erythema (L59)
Radiation-induced changes of skin and subcutaneous tissue (L55–L59)
Sunburn (L55)
Classification of Frostbite According to ICD-10
T33. Superficial frostbite
Included:
Frostbite with partial loss of skin layers
Excluded:
Superficial frostbite involving multiple body regions (T35.0)
T34. Frostbite with tissue necrosis
Excluded:
Frostbite with tissue necrosis involving multiple body regions (T35.1)
T35. Frostbite involving multiple body regions and unspecified frostbite
Excluded:
Hypothermia and other effects of exposure to low temperatures (T68–T69)
Effects of High Temperature
Predisposing factors for overheating include individual physiological characteristics, diseases of the respiratory system, cardiovascular system, excretory organs, and age. Overheating may manifest as heat stroke or sunstroke. Death occurs due to primary respiratory arrest at a body temperature of 42.5–43.5°C, resulting from severe central nervous system dysfunction caused by circulatory disorders and hypoxemia.
No specific macroscopic or microscopic changes are observed. Diagnosis is based on marked cerebral edema and multiple scattered petechial hemorrhages in the brain.
Burn Injury
A burn is a pathological process resulting from exposure of the skin and mucous membranes to high temperatures (flame, hot steam and gases, heated objects, boiling water).
Local changes range from mild injury to necrosis. Burns are classified into four degrees:
First degree: erythema and skin edema
Second degree: blister formation
Third degree: necrosis
Fourth degree: charring
Skin samples from burn sites are collected for histological examination.
Microscopic Findings in Burns
First-Degree Burns
Marked congestion of capillaries and arteries of the skin, loosening of the subpapillary layer, and serous edema of the dermis.
Second-Degree Burns

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Formation of blisters located within the epidermis or between the epidermis and dermis. In some areas, the basal layer is disrupted. Blisters are filled with homogeneous or finely granular fluid containing epithelial cells, fibrin, and erythrocytes. The fibrous structure of the dermis is preserved; collagen fibers are homogenized with a slight basophilic tint. Blood vessels are dilated and congested. In the first hours after injury, reactive processes develop: increasing vascular congestion, leukocyte infiltration of blister contents, and progressive edema. By days 2–3, pronounced demarcation inflammation appears.

Third-Degree Burns

Characterized by necrobiotic processes. The epidermis appears as a thin, structureless, wavy brown band. The skin undergoes coagulative necrosis with preservation of epidermal cellular outlines. Dermal collagen fibers are markedly thickened, homogenized, and basophilic, showing metachromasia. Fibrocyte and fibroblast nuclei are intensely stained. Elastic fibers are straightened, thinned, and fragmented. Vascular congestion, hemorrhages, and thrombosis are present.

Fourth-Degree Burns

Charring of the epidermis and upper dermal layers with necrosis of underlying tissues. Microscopically, the charred dermis appears as uneven homogeneous black bands, sometimes elevated above the dermis. The skin is compacted and compressed, forming a homogeneous layer with remnants of fibrous structure. Nuclei of connective tissue cells are visible in deeper layers. Capillaries appear as cellular strands. Underlying muscle tissue is yellow-pink with preserved structure, and vessels are collapsed.

In adjacent areas, necrobiotic and dystrophic epidermal changes, impaired blood rheology, hemorrhages, and thrombosis are observed.

Changes in Internal Organs in Fatal Burns

Meninges: vascular congestion, loosening, and edema

Brain: uneven blood filling, capillary and arteriolar spasm followed by dystonia and congestion; sludge and stasis; perivascular edema; petechial (diapedetic) hemorrhages; neuronal swelling with vacuolization

Lungs: bronchial lumen lined by scalloped epithelium; collapsed and locally dilated alveoli; congested interalveolar capillaries; soot particles and desquamated epithelium in small bronchi

Heart: granular dystrophy of cardiomyocytes; congested microvasculature

Liver: glycogen depletion; marked eosinophilia of hepatocytes; vascular congestion

Kidneys: dystrophy and necrobiosis of convoluted tubule epithelium; homogeneous or finely granular pale pink material in Bowman's capsule

Effects of Low Temperature

Death from Cooling (Hypothermia)

Death from low temperature may occur not only at subzero temperatures but also at above-zero temperatures under conditions of high humidity, strong wind, and aggravating factors such as physical exhaustion and alcohol intoxication. The duration of exposure leading to death ranges from 2 to 4 hours.

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During autopsy, the presence and severity of frostbite are assessed.

Degrees of Frostbite

First Degree:

Dilated papillary dermal capillaries filled with homogeneous pink material; dermal collagen fiber edema and homogenization.

Second Degree:

Blister formation within or beneath the epidermis. Epidermal necrosis appears as a homogeneous pink band. Blisters contain pale pink homogeneous material with isolated erythrocytes and leukocytes. Marked edema of dermis and subcutaneous tissue; swollen, partially homogenized collagen fibers.

Third Degree:

Hemorrhagic blisters; destruction of the Malpighian layer; necrosis of dermis and subcutaneous fat.

Fourth Degree:

Hemorrhagic blisters with mummification or liquefaction necrosis of the base; extension to bone tissue.

Phases of Frostbite Development

Pre-cold phase: decreased skin sensitivity, secondary edema due to endothelial damage (–2.7°C to –10°C)

Cold–thaw phase: formation of intracellular and extracellular ice crystals (–6.1°C to –15°C); nervous tissue highly vulnerable

Vascular stasis phase: continued ice crystal formation, plasma loss, vasospasm, and coagulation stasis

Late ischemia phase: ischemia, thrombosis, autonomic dysfunction, and protein denaturation

Macroscopic Signs of Death from Hypothermia

Blood and fibrin clots in the left heart chambers and aorta

Light red coloration of lung tissue

Petechial hemorrhages in renal pelvis mucosa

Mottled spleen (Wischnewsky sign)

Multiple gastric mucosal hemorrhages — pathognomonic for hypothermia

Wischnewsky Spots: Histomorphological Stages

Arterial and arteriolar spasm

Venous paresis and congestion with stasis

Subepithelial venous rupture and microhemorrhages

Necrosis with cone-shaped hemorrhage formation

Formation of hydrochloric acid hematin (typical Wischnewsky spot)

Subsequent stages include regenerative processes and focal sclerosis.

Signs of Body Freezing

Ice crystal formation in heart cavities and brain ventricles

Separation of cranial sutures

Fragility of extremities

Absence of postmortem decomposition

Histologically, epidermal swelling, epidermal–dermal separation, basophilic amorphous material in clefts, and needle-like cavities in internal organs due to frozen tissue fluid are observed. Thawing is characterized by rapid imbibition of tissues with hemolyzed blood.

Histological Description in Frostbite

Stomach: brown hemorrhages in mucosa, necrotic muscular layer, leukocytic infiltration, severe congestion with hemolyzed blood

Lungs: dilated alveoli, ruptured septa, hemosiderin deposition, bronchial spasm, mucus-filled goblet cells

Liver: uneven central vein congestion, preserved trabecular structure, elongated chain-like cavities along hepatic plates

Histological Description in Burns

Skin: epidermal detachment, swollen stratum corneum, melanin in basal cells, homogenized collagen fibers with clefts, black foreign particles on surface

Lungs: alveolar dilation and rupture, atelectasis, bronchial spasm, necrotic epithelium, macrophages with carbon particles, leukocytic infiltration, vascular congestion

Materials and Methods

Histological samples of skin, brain, lungs, heart, liver, kidneys, and stomach were examined using routine hematoxylin and eosin staining. Cases included fatal exposure to high temperatures (burns, hyperthermia) and low temperatures (frostbite, hypothermia).

Results

High-Temperature Exposure Skin:Epidermal detachment, blister formation, coagulative necrosis, collagen homogenization

Central nervous system: Cerebral edema, petechial hemorrhages

Lungs:Bronchial spasm, soot particles, alveolar collapse

Heart: Granular degeneration of cardiomyocytes

Kidneys: Tubular epithelial necrosis

Burn severity correlated with histological depth:

Grade I: vascular congestion, edema

Grade II: intraepidermal blisters

Grade III: coagulative necrosis

Grade IV: carbonization of tissues

Low-Temperature Exposure

Skin: Ice crystal–induced separation of epidermis and dermis

Vessels: Vasospasm, stasis, thrombosis

Stomach:Pathognomonic Wischnewsky spots due to hemorrhagic necrosis

Internal organs: Hemolysis, ischemic changes, microcavities from frozen tissue fluid

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Discussion

Thermal injuries induce primarily necrotic and inflammatory responses, while hypothermia leads to vascular dysregulation, ischemia, and characteristic hemorrhagic patterns. Recognition of these differences is essential for accurate forensic diagnosis.

Conclusion

Histopathological examination provides reliable criteria for differentiating deaths caused by extreme temperatures. The findings support standardized forensic evaluation in accordance with ICD classification.

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