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Burn-induced physiological changes in skin layers: A macroscopic study using the chicken egg chorion model

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Abstract
Burn injuries affect skin in progressive layers, ranging from superficial to full-thickness damage.
Aim: This study aimed to evaluate the visible effects of different stages of burns using the chorion layer of the chicken eggshell membrane as a biological model of the skin barrier.
Hypothesis: It was initially expected that the chorion, due to its thin nature, would show minimal visible damage at lower temperatures and rapid significant changes under high-temperature exposure.
Methods: The chorion was isolated from the inner membrane of fresh chicken eggs and subjected to three levels of thermal exposure: mild heat (50–60°C), boiling water (100°C), and direct contact with a heated metal. Samples were observed for visible damage or changes in color, texture, and structural integrity.

Keywords: Burn injury, chorion, tissue deterioration, epidermis, lesion, boils, scarring, blisters, high temperature exposure, partial thickness, multi-organ dysfunction, burn shock, structural variation, dermal damage

Introduction

Burns are a form of injury to the skin or tissue damage caused because of exposure to excessive heat, flames, hot liquids, electricity, steam, friction, ionizing radiation chemicals including strong acids and bases. Despite having the most devastating effects on the epidermis of the skin ranging from scarring, infection at the site of the wound to lesions, red boils and deterioration of the skin's natural barrier, they are still one of the top causes of death and disability in the world. Every year, nearly 180,000 people die from burn-related injuries worldwide, with millions more suffering from long-term physical, physiological, and psychological damage. Survivors of severe burn injuries face lifelong challenges including functional limitations, chronic pain, visible scarring and disfigurement accompanied by societal exclusion and discrimination.

Burn injuries can even be traced back to ancient times wherein 1600 BC the Egyptian Smith Papyrus prescribed the use of resin and honey salve for treating mild to moderate burns, claiming honey's antioxidant properties and resin's protective barrier prevented bacterial growth at the site of infection and promoted healing. In ancient India, Ayurvedic texts, especially the Sushruta Samhita and Atharva Veda, describe burns as Dagdha Vrana and classify them by severity. Treatments used ghee, honey, and herbs like sandalwood to cool, disinfect, and heal along with restoration of balance and regeneration of tissues. However, the very first prescription for the pain was by an Arab physician 845 CE who recommended running cold water at the site of the burn. History, in fact, has records of writings by Hippocrates, Celsus, and Galen contain descriptive methods of making ointments, dressings, and treatments for different types of burns.

While ancient medicine focused on external treatment, modern science and biotechnology delves even deeper, elucidating on how burn injuries disrupt the epidermal layer at the cellular level to find out the severity of the burn and treat appropriately. There are three primary stages of burns: first-degree, second-degree, and third-degree burns, wherein each is characterized by the severity of damage caused and not necessarily based on the cause of it.

The aim of this study is to investigate and analyze the disruption of the epidermal layer, extent and severity of damage to the tissues and changes occurring at the site of the burn.

Degrees of burns

First degree burns, also known as superficial burns, usually affect only the epidermis, or the outer layer of the skin, usually marked by redness, pain and mild swelling and rare signs of blisters or scalding of the skin. At the cellular level, first degree burns involve damage to the living stratified squamous epithelium that mature and move towards the surface known as keratinocytes. Furthermore, cell responses are triggered in such a situation which stimulates the release of inflammatory mediators like cytokine, chemokines and histamine that result in pain, redness and slight swelling. It can also cause temporary change in terms of melanin pigment production by melanocytes, resulting in slight darkening of the skin. Radiation from sun exposure, more commonly known as sunburn, is also an example.

Second degree burns (partial or intermediate thickness) not only affect the epidermis, but also the underlying layer of the skin called the dermis. This stage of burns is often severe, marked by painful boils or blisters and the burn site may appear red and swollen. These partial thickness burns occur when the skin encounters high voltage sources. A deep partial thickness burn involves damage to the deeper reticular dermis, resulting in the wound being uniformly red or pink or even blanch under pressure, accompanied by scarring even after healing, which is unavoidable. Over time, a thick, delicate, white tissue called fibrinous exudate develops over the wound, consisting of fibrin protein which plays a role in blood clotting.

Third-degree burns, or full-thickness burns, are the most destructive form of burn injuries involving complete

destruction of the epidermis and dermis and partial surface level harm to the subcutaneous tissues beneath the layers. In much more severe cases sometimes even extending into the muscle, blood capillaries, tendon or bone. These burns typically appear waxy white, leathery, charred, or dry, and are characteristically painless, owing to damage of the sensory nerve endings. Consequently, patients with third degree burns are at high risk for impaired thermoregulation, sepsis, immunosuppression leading to infection and potential burn shock, a life-threatening situation where adequate oxygen is not received by the body's tissues and organs. The risks also involve multi-organ dysfunction—including kidney injury, edema, Hypovolemia, tachycardia, and cardiac as well as pulmonary stress.

Materials and methods

To analyze the physiological effects of burn injuries on the skin, fresh chicken eggs were used to isolate the chorion layer from the inner eggshell membrane. After cracking the eggs open, the inner membrane was carefully detached from the shell. The chorion is the outermost thin membrane surrounding the developing embryo and it was taken to replicate the human epidermis for this experimental setup. The membranes were immediately rinsed in lukewarm distilled water to remove any remaining shell fragments and surface contaminants. Once clean, the membranes were carefully separated to expose the chorion layer and placed flat before exposing to different temperatures.

To simulate different stages of burn injury, the prepared chorion samples were subjected to controlled heat exposure. Three levels of burn were applied based on duration of heat exposure:

1. First-degree burn: mild heat exposure of 50 °C
2. Second degree burn: - moderate heat exposure at boiling point of water
3. Third degree burn: - exposure to flame or hot metal

After heat application, the samples were dipped in lukewarm water to cool and were examined for visible changes. Observations were made based on four parameters: colour changes, texture, and structural variations.

The experiment was conducted with utmost precaution while handling the heat sources and the samples were preserved before complete deterioration as in the experimental setup for third degree burns.

Results

Table 1: Experimental Conditions and Visual Indicators Used to Differentiate Burn Stages

Burn stages	Description	Heat source	Exposure time	Visible effects observed
Superficial burns	Mild heating	Hot water (50°C to 60°C)	20 seconds	Slight whitening of the membrane; minimal changes observed
Partial or intermediate thickness burns	Moderate heating	Boiling water (100 °C)	30 seconds	Visible large white patches, slight shrinkage, red or pinkish tint
Full thickness burns	Intense heat	Heated metal (direct contact)	3-4 seconds	Extreme shrinkage, brown and charred appearance on the sides, brittle texture.

The effects of thermal exposure on the chorion layer of the inner eggshell membrane varied noticeably across different burn stages, as depicted in table 1.1. Each condition produced distinct changes in color, texture, and structural integrity. In the superficial burns group, the specimen suffered slight whitening signifying the death of most of the live cells, minimal to negligible shrinkage at all and the chorion largely retained its original appearance. However, the second-degree burn group placed in boiling water at 100 °C showed shrinkage, and visible softening, along with reddish-pink tint retained by the chorion right after cooling. This phenomenon can be explained by a heat induced reaction of the glycoproteins present in the membrane, similar to the reddish swollen appearance of the epidermis in a burn wound. The skin most likely becomes more sensitive and more permeable to damage at this stage than in the previous condition. Lastly, in the third degree burn sample, pronounced shrinkage, black, charred and brittle texture was noticed, suggesting the complete structural breakdown of the membrane during burn injuries to the skin.

Conclusion

The chorion layer present in the eggshell membrane in this experiment portrayed the visible damage with increasing burn injury severity and increasing temperature. Contrary to the initial hypothesis, the chorion layer showed clear macroscopic changes across all burn stages, suggesting the chorion is more sensitive to thermal damage than expected. These changes reflect how burns affect human skin layers: superficial changes in the epidermis with mild burns, deeper dermal damage under intense heat.

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