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CHEMICAL BURN INDUCED OXIDATIVE STRESS AND MORPHOFUNCTIONAL CHANGES IN THE THYROID GLAND

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Abstract: This article presents an analytical review of systemic pathophysiological mechanisms that arise after chemical burns, particularly corrosive injury of the upper digestive tract, and evaluates their possible influence on the morphofunctional state of the thyroid gland. Chemical burn injury is interpreted not only as a local lesion of the mucosa, but also as a complex systemic process involving exotoxic shock, microcirculatory impairment, metabolic acidosis, hemolysis, inflammatory mediator activation, oxidative stress and secondary organ dysfunction. The thyroid gland is highly sensitive to systemic stress because thyroid hormones regulate energy metabolism, oxygen consumption, thermogenesis, protein synthesis, immune response and reparative processes. In severe burn disease, changes in thyroid hormone economy may reflect non-thyroidal illness syndrome rather than primary thyroid pathology. Therefore, the assessment of the thyroid gland after chemical burns requires an integrated analysis of hormonal, biochemical and morphological indicators. Particular attention is paid to follicular diameter, thyrocyte height, colloid condition, stromal edema, vascular congestion and signs of cellular dystrophy as morphologic criteria that may characterize post-burn endocrine adaptation. The article substantiates the relevance of studying oxidative stress as a key pathogenetic link connecting chemical injury, systemic intoxication and thyroid tissue remodeling. The proposed analytical model can serve as a theoretical basis for experimental morphological research within doctoral dissertation studies devoted to post-burn thyroid changes and their possible correction.

Keywords: chemical burn, corrosive injury, acetic acid, thyroid gland, morphofunctional changes, oxidative stress, non-thyroidal illness syndrome, thyrocyte, colloid, endotoxicosis.

Introduction. Burn injuries remain a serious public health problem in modern medicine. According to the World Health Organization, burns cause approximately 180,000 deaths annually, and the overwhelming majority of fatal cases occur in low- and middle-income countries. Non-fatal burns are also clinically important because they may result in long-term hospitalization, functional limitation, disfigurement, psychological distress, social stigmatization and economic burden for patients and their families [1].

Chemical burns occupy a specific place among burn injuries because the damaging agent continues to act on tissues until it is sufficiently diluted, neutralized by biological buffers or removed by medical intervention. Corrosive substances can damage the oral cavity, pharynx, esophagus, stomach and, in severe cases, deeper parts of the digestive tract and respiratory system. The severity of injury depends on the chemical nature of the agent, concentration, exposure time, amount of contact with tissues and the general condition of the patient. In clinical practice, chemical burns of the digestive tract are especially dangerous because local mucosal necrosis may be accompanied by systemic intoxication, hemodynamic instability, metabolic disturbances and multiple organ dysfunction [2,3].

The endocrine system plays a significant role in the organism response to severe injury. The thyroid gland is one of the central endocrine organs involved in the regulation of basal metabolism, oxygen consumption, thermogenesis, cardiovascular function, immune reactivity and tissue repair. Under critical illness, trauma and severe inflammatory stress, thyroid function tests often change even in the absence of primary thyroid disease. This phenomenon is generally described as non-thyroidal illness syndrome, also called euthyroid sick syndrome [4,5].

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The relationship between chemical burn disease and thyroid morphofunctional status remains insufficiently systematized. Existing literature extensively discusses burn epidemiology, caustic injury management, oxidative stress and endocrine responses to critical illness. However, fewer studies focus specifically on morphological changes in the thyroid follicular apparatus after chemical injury of the digestive tract. For this reason, a theoretical analysis of the pathogenetic chain linking corrosive injury, systemic intoxication, oxidative stress and thyroid tissue remodeling is scientifically relevant.

Aim of the study. The aim of this article is to analyze the systemic mechanisms that develop after chemical burns and to substantiate their possible influence on the morphofunctional state of the thyroid gland, with special emphasis on oxidative stress, inflammatory response, microcirculatory impairment and post-stress thyroid hormone imbalance.

Materials and methods. The article was prepared as a narrative analytical review. Scientific sources concerning burn epidemiology, corrosive injury of the digestive tract, oxidative stress, thyroid physiology, thyroid function in critical illness and morphological adaptation of endocrine organs were analyzed. Priority was given to international review articles, clinical toxicology sources and publications indexed in major biomedical databases.

The analysis was structured according to the following criteria: the global clinical significance of burns; the local and systemic effects of chemical injury; the role of oxidative stress and inflammatory mediators; morphologic indicators of the thyroid follicular apparatus; interpretation of thyroid hormone changes in critical illness; and the theoretical basis for studying post-burn thyroid recovery. Data were synthesized in a comparative and pathogenetic manner in order to form a coherent model suitable for further experimental morphological research.

Results and discussion. Clinical and morphological essence of chemical burns

Chemical burn is a complex tissue injury caused by direct contact with a corrosive substance. Acids usually induce coagulative necrosis due to protein denaturation, whereas alkalis tend to cause liquefactive necrosis with deeper tissue penetration. In both cases, the initial lesion is followed by inflammation, vascular damage, edema, pain, microcirculatory disorders and disturbances in reparative processes. When the upper digestive tract is involved, the injury may affect mucosa, submucosa and muscular layers, thereby increasing the risk of bleeding, strictures, perforation and long-term functional impairment [2,3].

In severe chemical burns, local tissue damage is accompanied by systemic pathological reactions. Plasma loss, hypovolemia, hemolysis, metabolic acidosis, coagulation disorders and endogenous intoxication may develop. These changes can impair oxygen delivery to tissues and activate inflammatory cascades. As a result, chemical burn disease should be considered a systemic disorder rather than a purely local mucosal injury. Such systemic interpretation is essential for understanding why distant organs, including the thyroid gland, may undergo secondary morphofunctional changes.

Oxidative stress is one of the key mechanisms in the pathogenesis of burn disease. Tissue injury, hypoxia, microcirculatory failure, hemolysis and activation of inflammatory cells increase the production of reactive oxygen species. Under physiological conditions, reactive oxygen species participate in intracellular signaling; however, excessive generation of these molecules damages lipids, proteins and nucleic acids. Lipid peroxidation is especially important because it destabilizes cellular membranes and contributes to epithelial, endothelial and parenchymal cell injury [6].

Chemical burns intensify oxidative imbalance through several mutually reinforcing pathways. Necrotic mucosal tissue releases inflammatory mediators; systemic hypoxia promotes mitochondrial dysfunction; hemolysis and metabolic acidosis aggravate tissue oxygen utilization; and endotoxicosis increases the burden on detoxifying organs. In this context, oxidative stress becomes a bridge between the primary corrosive injury and secondary organ alterations.

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The thyroid gland is physiologically linked to oxidative reactions because hydrogen peroxide is required for iodination and thyroid hormone synthesis. This makes thyrocytes particularly dependent on a stable balance between oxidant production and antioxidant defense. When systemic oxidative stress exceeds antioxidant capacity, thyroid tissue may become vulnerable to membrane damage, mitochondrial dysfunction, impaired hormone synthesis and structural remodeling of follicles [6,7].

Table 1. Main pathogenetic mechanisms influencing the thyroid gland after chemical burns

Pathogenetic mechanism	Systemic consequence	Possible morphofunctional response in the thyroid gland
Corrosive mucosal injury	Necrosis, inflammation, pain and release of toxic mediators	Stress response in thyrocytes, stromal edema and vascular reaction
Hypovolemia and microcirculatory impairment	Tissue hypoxia and metabolic acidosis	Dystrophy of follicular epithelium and altered colloid rheology
Oxidative stress	Lipid peroxidation and mitochondrial dysfunction	Damage to thyrocyte membranes and impairment of hormone-synthesis processes
Inflammatory mediator activation	Cytokine release, endothelial activation and coagulation shifts	Stromal infiltration, capillary congestion and activation of reparative processes
Hypothalamic-pituitary-thyroid axis response	Adaptive shifts in T3/T4 metabolism and TSH regulation	Functional imbalance consistent with non-thyroidal illness syndrome

Post-burn thyroid response: functional and morphological criteria

The thyroid follicle is the main structural and functional unit of the gland. It consists of a follicular epithelial lining and colloid. Follicular diameter, epithelial height, colloid density, colloid vacuolization and vascular stromal condition are important indicators of thyroid functional status. In active follicles, thyrocytes may become taller and colloid resorption may increase. In hypofunctional or dystrophic states, follicles may become enlarged, epithelium may flatten and colloid may become dense or stagnant. These changes require careful interpretation because they may reflect either adaptive regulation or pathological injury.

After severe burn injury, the thyroid response is not unidirectional. In the early stress phase, activation of the sympathetic-adrenal system, the hypothalamic-pituitary-adrenal axis and inflammatory mediators may alter peripheral conversion of thyroid hormones. At the same time, systemic hypoxia, oxidative stress and microcirculatory impairment may directly affect thyroid tissue. Therefore, post-burn thyroid morphology should be assessed dynamically, taking into account the stage of burn disease and the severity of systemic intoxication.

Morphologically, the following signs are of particular scientific interest: changes in follicular diameter, alterations in thyrocyte height, changes in the nuclear-cytoplasmic ratio, colloid dilution or condensation, resorption vacuoles, stromal edema, vascular congestion, perivascular cellular reaction, dystrophic changes in thyrocytes and possible fibrotic remodeling. These indicators allow the researcher to connect histological data with biochemical and hormonal parameters.

Table 2. Recommended indicators for assessing the morphofunctional state of the thyroid gland

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Assessment area	Morphological indicator	Functional indicator	Scientific interpretation
Follicular apparatus	Follicular diameter and epithelial height	Serum T3 and T4 levels	Reflects follicular activity and hormone-synthesis status
Colloid condition	Colloid density, vacuolization and resorption margins	Thyroglobulin and hormone reserve	Indicates the balance between secretion and resorption
Stroma and vessels	Edema, capillary congestion and perivascular infiltration	Inflammatory markers	Reflects microcirculation and immune response intensity
Cellular injury	Dystrophy, apoptosis and nuclear changes	Oxidative stress biomarkers	Shows thyrocyte resistance to stress and reparative potential
Reparative remodeling	Fibrosis, stromal restructuring and follicular architecture	TSH and peripheral conversion indices	Indicates long-term recovery or persistent imbalance

Non-thyroidal illness syndrome and post-stress thyroid imbalance

In critical illness, changes in thyroid function tests frequently represent a systemic adaptive response rather than primary thyroid pathology. Non-thyroidal illness syndrome is commonly characterized by decreased peripheral T3 concentration, changes in T4 metabolism and normal or reduced TSH levels. These changes are associated with altered deiodinase activity, cytokine effects, hypothalamic-pituitary regulation and reduced peripheral conversion of thyroxine to triiodothyronine [4,5].

This principle is important for interpreting thyroid status after chemical burns. A reduction in T3 or a shift in TSH should not automatically be diagnosed as primary hypothyroidism or hyperthyroidism. In the context of severe injury, such findings may reflect metabolic adaptation aimed at reducing energy expenditure and limiting tissue damage. However, persistent or excessive hormonal imbalance may also contribute to delayed recovery, impaired thermogenesis, reduced protein synthesis and weakened reparative processes. Therefore, both adaptive and maladaptive aspects of thyroid response must be considered.

For experimental morphological studies, the combination of hormonal tests with histological analysis is especially valuable. Hormonal indicators provide information about systemic endocrine regulation, whereas histological indicators show the structural condition of the follicular apparatus. This integrated approach makes it possible to identify whether post-burn changes are transient, compensatory, dystrophic or reparative.

Treatment and morphofunctional recovery

The main clinical objectives after chemical burns are limitation of primary tissue injury, stabilization of breathing and circulation, correction of metabolic disturbances, prevention of infectious complications and support of reparative processes. Management of corrosive injury must be performed only by trained medical professionals because the risk of airway compromise, perforation, bleeding and systemic shock may be high [2,3].

From the standpoint of thyroid protection, the most promising research directions include reduction of systemic oxidative stress, preservation of microcirculation, correction of metabolic acidosis and support of antioxidant defense. Selenium, zinc, vitamins C and E, polyphenols and other antioxidant strategies have been discussed in the literature on thyroid diseases and

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oxidative stress, although their use after chemical burn injury requires experimental verification and should not be interpreted as a ready clinical protocol [6,7].

Phytotherapeutic agents such as Potentilla alba rhizome preparations have been studied in Eastern European practice in relation to thyroid disorders. Some publications discuss possible effects on thyroid volume and hormone parameters [8]. Nevertheless, active compounds, standardization, dosage safety and efficacy in post-burn thyroid dysfunction remain insufficiently established. Therefore, such interventions should be regarded as a research hypothesis rather than a confirmed method of treatment.

Scientific and practical significance. Studying morphofunctional changes in the thyroid gland after chemical burns is important for several reasons. First, it expands the understanding of burn disease as a systemic pathological process involving endocrine regulation. Second, it allows researchers to connect burn-induced oxidative stress with structural changes in an endocrine organ that is highly dependent on redox balance. Third, integrated evaluation of hormonal, biochemical and morphological markers may improve interpretation of experimental findings and help distinguish adaptive thyroid response from tissue injury.

The proposed pathogenetic model may be used as a theoretical framework for doctoral dissertation research. It can guide the formulation of experimental groups, selection of histological criteria, interpretation of thyroid hormone changes and evaluation of potential corrective approaches. The model also emphasizes the need for a multidisciplinary view of chemical burn disease, combining clinical toxicology, morphology, endocrinology, biochemistry and pathological physiology.

Conclusion

1. Chemical burns should be regarded as systemic pathological processes that combine local mucosal injury with exotoxic shock, microcirculatory impairment, metabolic acidosis, oxidative stress and inflammatory response.

2. The thyroid gland is sensitive to systemic post-burn stress because its hormones regulate energy metabolism, oxygen consumption, thermogenesis, immune response and tissue repair.

3. Post-burn thyroid hormone changes should be interpreted in the context of non-thyroidal illness syndrome and adaptive metabolic regulation rather than automatically classified as primary thyroid disease.

4. Oxidative stress and lipid peroxidation represent key pathogenetic links that may contribute to thyrocyte injury, colloid alteration and remodeling of the follicular apparatus.

5. Morphofunctional assessment of the thyroid gland after chemical burns should combine histological, biochemical, hormonal and clinical indicators in order to obtain a scientifically grounded interpretation of post-burn endocrine adaptation and recovery.

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