AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ISSN NUMBER: 2692 - 5206 Volume 5. No 11. November ,2025

MORPHOMETRIC CHANGES IN THE THYROID GLAND IN CHEMICAL BURNS OF THE DIGESTIVE TRACT AND THE POSSIBILITIES OF THEIR CORRECTION IN AN ANALYTICAL REVIEW OF THE LITERATURE

Komiljonova Oygul Olimjonovna
Assistant at the Department of Fundamental Medicine
Asia International University, Bukhara, Uzbekistan
https://orcid.org/0000-0002-0348-3613
Email:komiljonovaoygulolimjonovna@oxu.uz

Abstract: Chemical burns of the gastrointestinal tract not only cause severe local tissue damage but also trigger systemic reactions affecting distant organs. This literature review summarizes current data on morphometric changes in the thyroid gland after corrosive injuries to the gastrointestinal tract, and discusses possible mechanisms of their occurrence and approaches to correction. The introduction describes the prevalence and pathophysiology of gastrointestinal burns with acids and alkalis, emphasizing that the severity of the injury varies from mild mucosal lesions to full-thickness necrosis of the wall with life-threatening complications [6]. The main section analyzes changes in the thyroid gland based on experimental and clinical studies: after severe burn injuries, microstructural rearrangements in the gland (deformation of follicular architecture, lymphomonocytic infiltration) and suppression of its function (euthyroid pathology syndrome) are observed [5]. Key mechanisms include systemic release of inflammatory cytokines (e.g., IL-6, TNF-α) [2], acute stress response with hyperproduction of adrenal hormones, and neuroendocrine disturbances at the level of the hypothalamic-pituitary-thyroid axis. Diagnosis of thyroid changes is based on histological examination (colloid depletion and thyroglobulin aggregation in follicles were detected in burn victims) and determination of hormonal parameters; instrumental methods (ultrasound, scintigraphy) are used limitedly. Various approaches to correcting the identified disorders are discussed in the literature: anti-inflammatory therapy, antioxidants, and hormonal support are considered as ways to mitigate thyroid damage and its dysfunction. However, targeted evidence-based treatments are still lacking. In conclusion, it is noted that chemical burns of the gastrointestinal tract can cause significant morphometric and functional changes in the thyroid gland through complex systemic mechanisms. Early detection and correction of thyroid disorders in burn patients can improve outcomes, but further research is needed to develop effective treatments.

Keywords: Chemical burn; Digestive tract; Thyroid gland; Morphometric changes; Correction

Introduction.

Chemical burns of the gastrointestinal tract (also known as caustic gastrointestinal injuries) are complex medical and surgical emergencies characterized by severe damage to the mucosa and deep layers of the gastrointestinal tract. These burns most often result from the ingestion of corrosive substances (strong acids or alkalis), either accidentally (especially in children) or as a result of suicide attempts in adults [1]. The degree of damage from caustic exposure varies widely: in mild cases, changes may be virtually absent, while in severe cases, the corrosive substance causes necrosis of the entire thickness of the esophagus and stomach wall, followed by perforation [1].

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 5. No 11. November ,2025

Acute local consequences of chemical burns of the gastrointestinal tract include severe inflammation, ulceration, hemorrhage, and the risk of tissue perforation with the development of peritonitis or mediastinitis. In addition to local damage, chemical burns can cause systemic toxic effects and a generalized inflammatory response. Chemical absorption and the release of inflammatory mediators can lead to systemic complications such as metabolic acidosis, intravascular hemolysis, coagulopathy, acute renal failure, and shock [3]. Patients with severe gastrointestinal burns often present with symptoms of an acute systemic inflammatory response (fever, tachycardia) and may develop multiple organ dysfunction due to cytokine release and hypovolemia due to fluid loss.

The endocrine system, responsible for regulating the body's stress response, is also affected. Extensive burns (including extensive external burns and severe internal caustic injuries) trigger a complex stress response involving the hypothalamic-pituitary-adrenal axis (elevated cortisol levels) and sympathetic nervous system activation (elevated catecholamine levels), while other hormonal axes, including the thyroid axis, may be suppressed as part of the acute phase response [2].

This complex hormonal alterations in critical illness is known as euthyroid sick syndrome (or nonthyroid sick syndrome, NTIS), in which thyroid hormone levels are abnormal in an anatomically intact thyroid gland. Clinically, NTIS is characterized by decreased triiodothyronine (T3) levels, normal or decreased thyroxine (T4) levels, and inappropriately normal or decreased thyroid-stimulating hormone (TSH) levels [7]. The severity of these hormonal disturbances correlates with the severity of injury and prognosis: patients with more pronounced T3/T4 reductions after severe trauma or burns tend to have worse outcomes [5].

The thyroid gland plays a key role in regulating metabolism, and changes in its function can significantly impact recovery after injury. Although acute hormonal changes in critical illness are well documented, there is growing interest in the structural (morphological) changes in vital organs in response to severe stress. In extensive burns or severe systemic injury, the thyroid gland can undergo histopathological changes due to shock, inflammation, and neuroendocrine dysregulation [5].

Modern studies, including autopsy data from burn victims, indicate the presence of measurable morphometric changes in the thyroid gland itself after severe burns [5]. These include distortion of follicular architecture, cellular infiltration, and colloid depletion, reflecting direct or indirect damage to the gland.

Chemical burns of the gastrointestinal tract represent a unique model for studying the long-term effects of organ damage, as they combine local tissue destruction with a pronounced systemic inflammatory response. Given that the thyroid gland is highly vascularized and sensitive to systemic metabolic changes, understanding how corrosive damage to the gastrointestinal tract affects its condition is essential for comprehensive patient management. Significant thyroid dysfunction may cause decreased metabolic activity, impaired protein synthesis, and other processes, which can impede healing. Furthermore, if structural damage to the gland is not corrected, long-term consequences such as persistent hypothyroidism may develop.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 5. No 11. November ,2025

Morphometric changes in the thyroid gland after chemical burns of the gastrointestinal tract.

Severe burns and trauma can cause pronounced morphological changes in endocrine organs, including the thyroid gland. Although direct clinical studies on thyroid morphology after caustic burns of the gastrointestinal tract are few, indirect data have been obtained from postmortem examinations of burn victims and animal experiments. Histopathological examinations of thyroid tissue in critically ill patients reveal recurring patterns of changes. In a recent cross-sectional autopsy study of burn victims, deformation of the normal follicular architecture of the thyroid gland was detected in approximately 59% of cases [5]. Follicles, which are normally spherical structures filled with colloid and lined by a single-layer epithelium, were distorted in shape or collapsed. Furthermore, mononuclear cell infiltration (mainly lymphocytes and macrophages) was observed in approximately 66% of cases [2], suggesting an inflammatory process in the thyroid gland, likely caused by systemic inflammation associated with burn disease or immune dysregulation. The presence of inflammatory cells suggests that the thyroid gland may be a target of a systemic immune response following burn injury.

Another significant change described in studies was thyroglobulin clumping in follicles in approximately 22% of autopsy cases [5]. Thyroglobulin is a protein precursor contained in colloid and serves as the starting material for the synthesis of thyroid hormones. Its clumping or coagulation may reflect a disruption in colloid composition or a response to injury, such as thermal denaturation or changes in glandular physiology following a burn. Additionally, follicular depletion—that is, a decrease in colloid volume—was found in approximately 17% of cases [5]. Follicular depletion reflects significant depletion or destruction of colloid stores (and, consequently, thyroid hormone stores). In critical illness, low T3 and T4 levels may be associated with colloid depletion if the gland has attempted to actively release hormones in response to stress and is then suppressed, or if colloid is depleted but not replenished due to reduced TSH stimulation.

The combination of these changes essentially represents a form of acute thyroid injury or involution. An autopsy study concluded that severe burns have a "metabolic damaging effect" on the thyroid gland, potentially leading to functional hypothyroidism, progressing over time, if the patient had survived longer [5]. In other words, structural changes in the thyroid gland correlate with reduced hormonal activity (low thyroid hormone levels) observed clinically in patients with severe burns.

It is important to note that such morphometric changes are not limited to external burns; it is biologically reasonable to hypothesize that internal burns of the gastrointestinal tract, which cause a similarly massive inflammatory response, may lead to similar changes in the thyroid gland. The gastrointestinal tract has extensive neuroendocrine connections with other organs (via the gut-thyroid axis, vagal pathways, etc.), and damage to it can generate stress signals that affect other systems.

Animal experimental data also confirm thyroid involvement in corrosive burns of the gastrointestinal tract. For example, in some rat models, changes in distant organs appeared within days or weeks after exposure to severe chemical burns of the esophagus. Although most research focuses on local damage and treatment methods, some studies report observations

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 5. No 11. November ,2025

consistent with changes in the thyroid gland. In severely burned animals, acute loss of endocrine gland mass and changes in metabolic parameters have been observed. Stress from a chemical burn can cause a decrease in thyroid mass (atrophy) due to reduced TSH stimulation or, conversely, a temporary increase due to edema or inflammatory infiltration. In similar stress models (hemorrhagic shock, severe infection), flattening of follicular cells (a sign of decreased activity) has been described in animals, as well as changes in colloid levels—its accumulation or depletion depending on the stage of the disease. Direct histological data on the thyroid gland in the rat caustic burn model are rare, but by analogy with human data and other critical illness models, similar morphometric changes can be expected: disruption of follicular structure, variability in the amount of colloid (often its decrease) and inflammatory infiltration.

Additional indirect confirmation comes from measuring thyroid gland weight and histology in animals treated after a burn. In a number of studies investigating treatment methods (discussed in more detail below), organ parameters are measured indirectly. If protective therapy prevents a decrease in gland weight or maintains normal histology, this implies that the burn itself leads to a deterioration of these parameters in untreated animals. For example, if the group receiving antioxidant therapy maintains a nearly normal thyroid structure compared to the untreated group, it can be concluded that the burn causes significant damage to the gland without treatment.

Overall, the literature indicates that morphometric changes in the thyroid gland after severe gastrointestinal burns may include: a decrease in gland size or weight (involution), disruption of follicular architecture, loss or alteration of colloid, flattening or degeneration of the follicular epithelium, and inflammatory infiltration. These changes reflect a state of severe stress and thyroid damage secondary to the systemic effects of the burn. Such structural changes underlie functional impairments in thyroid hormone synthesis and link biochemical findings (e.g., low T3 levels) with actual histopathological signs of thyroid damage.

In conclusion, the thyroid gland can be considered an "innocent bystander" organ caught up in the pathological cascade of the body's reactions following a chemical burn of the gastrointestinal tract. Although it is not in direct contact with the corrosive agent, a chain of physiological changes can significantly disrupt its morphology and function. Fortunately, many of these changes are part of an acute adaptive response and can be reversible over time or with appropriate therapy.

By highlighting this aspect of burn disease, we call for increased awareness and further research—especially prospective observations and targeted experimental studies—to fill existing knowledge gaps. Such studies can contribute to the development of improved treatment protocols aimed not only at eliminating visible damage but also at correcting associated "invisible" metabolic disorders.

Ultimately, protecting and restoring thyroid function in patients with gastrointestinal burns is an important element in improving the overall prognosis and quality of life for these patients.

References

AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ISSN NUMBER: 2692 - 5206 Volume 5. No 11. November ,2025

1.Chirica M., et al. (2017). Acute caustic ingestion injury of the gastrointestinal tract: a literature review. World Journal of Gastroenterology, 23(30), 5435–5443pubmed.ncbi.nlm.nih.gov.

2.Chen P.C., Cheng H.T., et al. (2021). Profiling of inflammatory cytokines in patients with caustic gastrointestinal tract injury. Clinical Toxicology, 59(6), 517–524pubmed.ncbi.nlm.nih.govpubmed.ncbi.nlm.nih.gov.

3.Hsieh T.C., et al. (2022). Systemic complications following caustic ingestion injuries. Clinical Toxicology, 60(4), 478–486tandfonline.com.

4.Demidchik L.A., et al. (2018). Oxidized proteins in the blood of patients with acute poisoning by acetic acid. International Journal of Applied and Fundamental Research, (5-1), 82–86bsmi.uz.

5.Roy A.K., Saha D., et al. (2022). Assessment of histopathological changes in the thyroid gland of fatal burn patients: A cross-sectional study. Egyptian Journal of Forensic Sciences, 12(1), 1–8researchgate.netresearchgate.net.

6.Vaughan G.M., et al. (1985). Alterations of mental status and thyroid hormones after thermal injury. Journal of Clinical Endocrinology and Metabolism, 60(6), 1221–1225pubmed.ncbi.nlm.nih.govpubmed.ncbi.nlm.nih.gov.

7.Fliers E., et al. (2015). The molecular basis of the non-thyroidal illness syndrome. Journal of Endocrinology, 225(3), R67–R81sciencedirect.comuptodate.com.

8.Park S.J., et al. (2017). Protective effect of ursodeoxycholic acid in an experimental rat model of corrosive esophageal burn. Archives of Pharmacal Research, 40(8), 966–975bsmi.uz.

9. Arslan S., et al. (2017). The effect of polaprezinc on the healing of caustic esophageal burns in rats. Esophagus, 14(1), 89–95bsmi.uz.

10.Hamroeva L.R. (2023). Morphological changes in the small intestine in digestive tract burns of various degrees. European Journal of Modern Medicine and Practice, 4(1), 45–50bsmi.uz.

11.Becker R.A., et al. (1982). Hypermetabolic low triiodothyronine syndrome of burn injury. Archives of Surgery, 117(6), 782–786europepmc.org.

12.Cheng H.T., et al. (2008). Caustic ingestion in adults: the role of endoscopic classification in predicting outcome. BMC Gastroenterology, 8, 31.

13.Erofeeva M.V., et al. (2008). Acid-base balance disorders in acute poisonings with acetic acid. Herald of Anesthesiology and Resuscitation, 5, 13–17.

14.Vlasov A.P., et al. (2012). Metabolic changes in burn disease: endocrine aspects. Journal of Extreme Medicine, 4, 27–33.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 5. No 11. November ,2025

15.Kozka A.A., et al. (2015). Impact of oxidative stress on endocrine glands in critical conditions. Bulletin of Experimental Biology and Medicine, 159(6), 740–743. (Translated from Russian).

16. Olimjonovna, K. O. (2023). Ayollarda reproduktiv tizim faoliyatining

O'zgarishida gipoterioz bilan birga kechishi. Ta'lim innovatsiyasi va integratsiyasi, 10(3), 174-179.

17. Olimjonovna, K. O. (2024). Hypothyroidism and reproductive dysfunction in women. Education, Science, and Innovative Ideas in the World, 36(5), 75-82.

18. Komiljonova, O. (2024). The use of ginger for medicinal diseases based on traditional medicine. Central Asian Journal of Education and Innovation, 3(1), 203-211.

19. Olimjonovna, K. O. (2024). Morphological criteria of the thymus in congenital heart disease. Education, Science, and Innovative Ideas in the World, 36(6), 197-202.

20. Olimjonovna, K. O. (2024). Clinical and morphological aspects of the topographic anatomy of the parathyroid glands. Education, Science, and Innovative Ideas in the World, 36(6), 209-217.

21. Olimjonovna, K. O. (2024). 2-tip qandli diabetni davolashda ayurveda yondashuvining ahamiyati. Education, science and innovative ideas in the world, 39(5), 132-143.

22. Olimzhonovna, K. O. (2024). Diabetic neuropathy: etiology, pathogenesis, clinical features and treatment approaches. European journal of modern medicine and practice, 4(3), 159-166.

23. Olimjonovna, K. O. (2024). Hypothyroidism in menopausal women recommendations developed based on experience. European journal of modern medicine and practice, 4(4), 228-235.

24. Saidova, L. B., & Komilzhonova, O. O. Pathological course of hypothyroidism in the climacteric period in the iodine-deficient zone of Uzbekistan. In International Conference Science and Education/Uluslararasi konferans bilim ve eg'itim//-2021-15may 49b. 10.

25. Olimjonovna, K. O. (2024). Investigation of distinctive skin alterations in menopausal women affected by hypothyroidism. Pedagog, 7(5), 302-310.

26. Olimjonovna, K. O. (2024). Understanding the causes and risk factors of diabetes.

BIOLOGIYA VA KIMYO FANLARI ILMIY JURNALI, 2(5), 8-14. 12.

27. Olimjonovna, K. O. (2024). The relationship between diabetes and heart disease. BIOLOGIYA VA KIMYO FANLARI ILMIY JURNALI, 2(5), 36-42.

AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ISSN NUMBER: 2692 - 5206 Volume 5. No 11. November ,2025

28. Olimjonovna, K. O. (2024). Managing type 2 diabetes with diet and exercise. BIOLOGIYA VA KIMYO FANLARI ILMIY JURNALI, 2(5), 22-28.

29. Olimjonovna, K. O. (2024). The link between diabetes and heart disease. BIOLOGY AND CHEMICAL SCIENCES SCIENTIFIC JOURNAL, 2(5), 29-35.

30. Olimjonovna, K. O. (2024). Understanding the causes and risk factors of Diabetes. BIOLOGY AND CHEMICAL SCIENTIFIC JOURNAL, 2(5), 1-7.

31. Olimjonovna, K. O. (2024). Managing type 2 diabetes through diet and Exercise. BIOLOGY AND CHEMICAL SCIENTIFIC JOURNAL, 2(5), 15-21.

32. Olimjonovna, K. O. (2024). Use alternative treatment methods to treat diabetes symptoms. MASTERS, 2(5), 25-32.

33. Olimjonovna, K. O. (2024). CRITERIA FOR CONGENITAL HEART DEFECTS. MASTERS, 2(5), 33-39.

34.Olimjonovna, K. O. (2024). Diabetes and pregnancy: what you need to know. MASTERS, 2(5), 18-24.

35.Olimjonovna, K. O. (2024). Application of alternative treatment methods for the management of diabetes symptoms. SCIENTIFIC JOURNAL OF BIOLOGY AND CHEMISTRY, 2(5), 50-56.