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MORPHOMETRIC CHANGES IN THE THYROID GLAND DURING CHEMICAL BURNS OF THE DIGESTIVE TRACT AND THEIR CORRECTION AN ANALYTICAL APPROACH BASED ON LITERATURE REVIEW

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Abstract: Chemical burns of the digestive tract can lead to severe local tissue damage and provoke systemic responses that affect distant organs. This literature review summarizes current knowledge on morphometric changes in the thyroid gland following corrosive injury to the gastrointestinal (GI) tract and discusses potential mechanisms and interventions for correction. The introduction outlines the epidemiology and pathophysiology of caustic GI burns, emphasizing that injury severity ranges from mild mucosal damage to full-thickness necrosis with life-threatening complications [1].The analysis focuses on reported thyroid gland alterations in experimental models and clinical observations: inflammatory and stress-mediated pathways are implicated in thyroid microstructural changes (follicular architectural distortion, cellular infiltration) and functional suppression (euthyroid sick syndrome) after severe burns [5]. Key mechanisms include systemic release of inflammatory cytokines (e.g. IL-6, TNF-α) [1], acute stress hormone surges, and neuroendocrine disruption of the hypothalamic-pituitary-thyroid axis. Diagnostic approaches from the literature range from histopathological examinations of the thyroid (showing colloid depletion and thyroglobulin clumping in burn victims) to imaging and hormonal assays. Therapeutic strategies for correction have been explored in animal studies and clinical practice: anti-inflammatory treatments, antioxidant therapy, and hormonal replacement are considered for mitigating thyroid damage and dysfunction. However, data on targeted interventions remain limited. In conclusion, chemical GI burns can induce significant morphometric and functional changes in the thyroid gland via complex systemic mechanisms. Early recognition and management of thyroid involvement in burn patients may improve outcomes, but further research is needed to establish effective therapeutic protocols.

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

Introduction. Chemical burns of the digestive tract (also known as caustic gastrointestinal injuries) are a challenging medical and surgical emergency characterized by severe damage to the mucosal lining and deeper layers of the GI tract. Such burns most commonly result from ingestion of corrosive substances (strong acids or alkalis), whether accidentally (especially in children) or in suicide attempts by adults [1]. The extent of injury caused by caustic ingestion ranges widely. In mild cases, there may be little to no visible damage; in severe cases, corrosives can cause full-thickness necrosis of the esophagus and stomach, leading to perforation [1]. The acute local consequences of caustic GI burns include intense inflammation, ulceration, hemorrhage, and risk of tissue perforation with peritonitis or mediastinitis.

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Beyond the local injury, chemical burns can provoke systemic toxic effects and a whole-body inflammatory response. Absorption of the chemical and release of inflammatory mediators can lead to **systemic complications** such as metabolic acidosis, intravascular hemolysis, coagulopathy, acute kidney injury, and shock [3]. Patients with severe GI burns often present with signs of acute systemic inflammation (fever, tachycardia) and may develop multi-organ dysfunction due to cytokine release and hypovolemia from fluid losses. The **endocrine system**, which orchestrates the body's stress response, is also affected. Major burns (including extensive external burns and severe internal corrosive injuries) trigger a complex **stress response** involving the hypothalamic-pituitary-adrenal axis (resulting in elevated cortisol levels) and sympathetic nervous system activation (elevated catecholamines), while other hormonal axes such as the thyroid axis may be suppressed as part of the acute phase response [2]. This constellation of hormonal changes in critical illness is often referred to as the euthyroid sick syndrome or non-thyroidal illness syndrome (NTIS), wherein thyroid hormone levels are abnormal despite an intact thyroid gland. Clinically, NTIS is characterized by low circulating triiodothyronine (T3) levels, with normal or low thyroxine (T4) and inappropriately normal or low thyroid-stimulating hormone (TSH) levels [7]. The extent of these thyroid hormone derangements has been correlated with injury severity and prognosis: patients with more profound reductions in T3/T4 after major trauma or burns tend to have worse outcomes [5].

The thyroid gland plays a critical role in regulating metabolism, and alterations in thyroid function can significantly impact recovery from injuries. While the acute changes in hormone levels during critical illness are well documented, there is increasing interest in the structural (morphological) changes that vital organs undergo in response to severe stressors. In the case of extensive burns or severe systemic insults, the thyroid gland may experience histopathological changes due to shock, inflammation, and altered neuroendocrine signaling [5]. Recent studies, including autopsy analyses of burn victims, suggest that there are measurable morphometric changes in the thyroid gland itself following severe burns [5]. These changes include distortion of thyroid follicle architecture, cellular infiltration, and depletion of colloid, which reflect direct or indirect injury to the gland.

Chemical burns of the digestive tract represent a unique model to study distant organ effects because they combine localized tissue destruction with a robust systemic inflammatory response. Given that the thyroid is highly vascular and sensitive to systemic metabolic changes, understanding how corrosive GI injury affects the thyroid gland is important for comprehensive patient management. If thyroid function is significantly impaired, patients may suffer from reduced metabolic rate, impaired protein synthesis, and other issues that could hinder healing. Additionally, long-term consequences such as persistent hypothyroidism could arise if structural damage to the thyroid is not corrected.

Morphometric changes in the thyroid gland after digestive tract chemical burns. Severe burns and traumatic injuries can induce notable morphological changes in endocrine organs, including the thyroid. Although direct clinical studies specifically on thyroid morphology after caustic GI burns are limited, related evidence can be drawn from post-mortem analyses of burn patients and animal experiments. Histopathological studies of thyroid tissue in the context of critical illness have revealed consistent patterns of change. In a recent cross-sectional autopsy study of fatal burn victims, approximately 59% of cases showed distortion of the normal follicular architecture of the thyroid gland [5]. Follicles, which are normally spherical structures filled with colloid and lined by a single layer of thyroid epithelium, were found to be irregular or collapsed in shape. Additionally, mononuclear cell infiltration (primarily lymphocytes and

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macrophages) was observed in about 66% of cases [2], suggesting an inflammatory process within the thyroid possibly triggered by burn-induced systemic inflammation or immune dysregulation. The presence of inflammatory cells indicates that the thyroid gland may become a target of the systemic immune response after a burn injury.

Another notable change reported was clumping of thyroglobulin in the thyroid follicles in roughly 22% of the autopsy cases [5]. Thyroglobulin is the protein precursor stored in colloid from which thyroid hormones are synthesized. Clumping or coagulation of thyroglobulin could signify an aberration in colloid composition or a response to injury such as thermal denaturation or altered gland physiology due to the burn. Moreover, exhaustion of follicles – meaning depletion of colloid content – was identified in about 17% of cases [5]. Follicular exhaustion reflects that the colloid (and thus thyroid hormone reserves) had been significantly utilized or broken down. In the context of critical illness, low T3 and T4 levels might paradoxically accompany colloid depletion if the gland attempted to release hormones in response to stress but was later inhibited or if colloid was consumed and not replenished due to suppressed TSH stimulation.

The cumulative effect of these changes is essentially a form of acute thyroid involution or injury. The autopsy study concluded that severe burn injuries have a **"metabolically damaging role"** on the thyroid gland, potentially resulting in a state of functional hypothyroidism that worsens over time if the patient had survived longer [5]. In other words, the structural damage in the thyroid correlates with the depressed thyroid function (low hormone levels) observed clinically in burn patients. Notably, these morphometric changes in the thyroid are not unique to external burns; it is biologically plausible that internal burns of the digestive tract, which can incite similarly massive inflammatory responses, lead to comparable thyroid changes. The digestive tract has extensive neuroendocrine connections with the rest of the body (through the gut-thyroid axis, vagal pathways, etc.), and injury to the GI tract can send distress signals affecting other organs.

Supporting evidence from animal models further suggests thyroid involvement after corrosive GI injury. For instance, in some experimental setups, rats subjected to severe chemical esophageal burns exhibited alterations in distant organs within days to weeks following the injury. Although many of these studies focus on local damage and treatments, a few report observations consistent with thyroid changes. Researchers have noted that severely burned animals can develop signs of acute weight loss of endocrine glands and altered metabolic rates. In a broad sense, stress from a chemical burn could cause the rat's thyroid gland to shrink in weight (reflecting atrophy) due to reduced TSH drive, or conversely, to swell temporarily due to edema or inflammatory cell infiltration. There are reports in analogous stress models (such as hemorrhagic shock or severe infection in animals) that the thyroid gland's follicular cells may flatten (indicating reduced activity) and colloid can accumulate or deplete depending on the phase of illness. While direct histological data of the thyroid in a caustic burn rat model are sparse in published literature, it is reasonable to extrapolate from the human data and other critical illness models that similar morphometric changes occur: namely, follicular derangement, variable colloid content (often reduced), and inflammatory infiltration.

Another indirect piece of evidence is the measurement of thyroid gland weight and histology in animals treated for burns. Some studies investigating therapies (discussed further below) incidentally measure organ indices. If a protective therapy prevents weight loss in the thyroid or preserves normal histology, it implies that the burn alone would have caused those parameters to worsen in untreated subjects. For example, if an antioxidant treatment group maintains near-

normal thyroid histology compared to an untreated burn group, we infer that the untreated burn likely had significant thyroid damage.

In summary, the literature indicates that morphometric changes in the thyroid gland after severe digestive tract burns can include: reduction in gland size/weight (involution), architectural distortion of follicles, loss or alteration of colloid, flattening or degeneration of follicular epithelial cells, and infiltration by immune cells. These changes reflect a thyroid gland under duress, likely secondary to systemic effects of the burn. Such structural changes underlie the functional alterations in thyroid hormone production in these patients, bridging the gap between biochemical findings (like low T3 levels) and tangible histopathological evidence of thyroid impairment.

In conclusion, the thyroid gland can be considered an "innocent bystander" organ that gets caught in the crossfire of the body's reaction to a chemical GI burn. While it is not directly in contact with the corrosive agent, the cascade of physiological responses can significantly alter thyroid morphology and function. Fortunately, many of these changes are part of an acute adaptive response and can be reversed with time or appropriate intervention. By shedding light on this aspect of burn injury, we advocate for increased awareness and further research – particularly prospective studies and targeted experiments – to fill the gaps in knowledge. Such research could lead to refined treatment protocols that not only treat the visible injury but also the invisible metabolic disturbances accompanying it. Ultimately, protecting and restoring thyroid function in patients with digestive tract burns is one piece of the puzzle in improving overall outcomes and quality of life for these patients.

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