

IMMUNOHISTOCHEMICAL CHANGES IN THE THYROID GLAND
UNDER POLYPHARMACY WITH ANTI-INFLAMMATORY DRUGS:
A LITERATURE REVIEW

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Abstract. Immunohistochemical methods provide additional information for evaluating drug-induced changes in the thyroid gland beyond conventional histological examination. The available literature indicates that inflammation, oxidative stress, and apoptosis are important biological processes involved in structural alterations of thyroid follicular cells. Cyclooxygenase-2 expression has been associated with inflammatory and pathological processes in thyroid tissue, while activation of the Fas/FasL pathway may contribute to apoptosis of follicular cells. Therefore, the combined assessment of morphological changes and immunohistochemical markers may provide a more comprehensive understanding of the tissue-level effects of anti-inflammatory drug polypharmacy on the thyroid gland.

Keywords: thyroid gland, polypharmacy, immunohistochemistry, COX-2, Fas, FasL, apoptosis, inflammation.

Introduction. Immunohistochemical examination is widely used in thyroid pathology to characterize cellular phenotypes and investigate molecular processes associated with inflammation, proliferation, and apoptosis [1]. Accordingly, immunohistochemistry may complement conventional morphological assessment when studying the effects of prolonged or combined pharmacological exposure on thyroid tissue.

Oxidative stress is closely associated with thyroid hormone biosynthesis because reactive oxygen species are physiologically generated during hormone

production. Excessive oxidative stress, however, may exceed the antioxidant capacity of thyroid cells and contribute to cellular injury [2]. NADPH oxidases, including DUOX and NOX4, participate in hydrogen peroxide generation within the thyroid and are involved in the regulation of thyroid redox homeostasis [3].

Cyclooxygenase-2 is of particular interest as a potential marker of inflammatory activity. Studies of human thyroid tissue have demonstrated increased COX-2 expression in several pathological conditions compared with normal thyroid tissue [4]. Therefore, evaluation of COX-2 immunoreactivity may provide additional information concerning inflammatory processes occurring in thyroid tissue.

Apoptosis represents another important mechanism of thyroid follicular cell injury. Experimental studies have demonstrated that combinations of inflammatory cytokines can enhance Fas-mediated apoptosis in thyroid follicular cells [5]. These findings support the potential value of investigating apoptotic pathways when evaluating thyroid tissue responses to prolonged or combined pharmacological exposure.

Thus, conventional histological examination alone may not fully characterize the molecular mechanisms underlying thyroid alterations associated with polypharmacy. A combined assessment of tissue morphology and immunohistochemical markers related to inflammation, oxidative stress, proliferation, and apoptosis may provide a more comprehensive evaluation of the effects of anti-inflammatory drug combinations on the thyroid gland.

Conclusion. The literature indicates that inflammation, oxidative stress, and apoptosis may contribute to structural alterations in thyroid tissue under various pathological and pharmacological conditions. Immunohistochemical assessment of relevant molecular markers, combined with morphological and morphometric analysis, represents a promising approach for elucidating the

mechanisms underlying thyroid tissue responses to polypharmacy with anti-inflammatory drugs.

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