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Opinion Article

Squamous Metaplasia Mechanisms of Epithelial Adaptation and Clinical Implications in Human Tissues

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DESCRIPTION

Squamous metaplasia is a pathological process in which a normal specialized epithelial tissue is replaced by stratified squamous epithelium as an adaptive response to chronic irritation, inflammation, environmental stress, or nutritional deficiencies. In ophthalmology, squamous metaplasia is an important manifestation of chronic ocular surface disease and the transformation of the normal non-keratinized conjunctival epithelium into a keratinized squamous epithelium. Although this adaptation initially serves as a protective mechanism against persistent injury, prolonged epithelial transformation normal physiological functions of the ocular surface, including lubrication, tear film stability, and protection against microbial invasion. Progressive squamous metaplasia may ultimately result in visual impairment, chronic discomfort, and irreversible ocular surface damage if not identified and managed appropriately. The healthy ocular surface consists of the cornea, conjunctiva, tear film, and associated glands that function together to maintain transparency, optical clarity, and protection. The conjunctival epithelium contains numerous goblet cells that secrete mucins, essential components of the tear film responsible for maintaining ocular surface hydration and smoothness. In squamous metaplasia, chronic epithelial injury leads to the gradual loss of goblet cells, decreased mucin production, increased epithelial keratinization, and replacement of normal columnar epithelial cells by flattened stratified squamous cells. These structural alterations impair tear film integrity and increase susceptibility to inflammation, infection, and epithelial breakdown. Vitamin A deficiency is one of the most significant systemic causes of ocular squamous metaplasia. Vitamin A plays a critical role in maintaining normal epithelial differentiation and goblet cell function. Deficiency results in progressive epithelial keratinization, xerosis, and loss of mucin-producing cells, eventually leading to conjunctival and corneal squamous metaplasia. In severe cases, persistent epithelial damage may progress to keratomalacia, corneal ulceration, and permanent blindness. Early recognition and nutritional supplementation remain essential for preventing irreversible ocular complications associated with vitamin A deficiency. Several ocular disorders are associated with the development of squamous metaplasia. Dry eye disease is among the most common causes, as chronic tear film instability exposes epithelial cells to continuous desiccation and mechanical stress. Long-standing ocular surface inflammation in conditions such as Stevens-Johnson syndrome, ocular cicatricial pemphigoid, chemical burns, thermal injuries, trachoma, and chronic allergic conjunctivitis can also induce epithelial transformation. Patients with limbal stem cell deficiency experience impaired epithelial regeneration, allowing abnormal squamous differentiation to replace healthy corneal epithelium. Chronic use of preserved topical medications, especially those containing benzalkonium chloride, has also been implicated in promoting epithelial toxicity and squamous metaplastic changes. Environmental factors significantly influence the development of ocular squamous metaplasia. Prolonged exposure to ultraviolet radiation, air pollution, smoke, dust, industrial chemicals, and dry climates increases oxidative stress and chronic inflammation on the ocular surface. Individuals working outdoors or in dusty environments frequently experience repeated epithelial injury that contributes to abnormal epithelial adaptation. Contact lens misuse, poor ocular hygiene, and chronic mechanical irritation from eyelid abnormalities further increase the likelihood of developing squamous metaplasia. Clinical manifestations depend on the extent of epithelial involvement and the underlying disease process. Early stages may remain asymptomatic or produce mild ocular irritation. As squamous metaplasia progresses, patients commonly complain of dryness, burning sensation, foreign body sensation, redness, photophobia, excessive tearing, blurred vision, and recurrent episodes of ocular discomfort. Tear film instability results in fluctuating visual acuity, particularly during prolonged reading or digital screen use. Advanced keratinization may cause recurrent epithelial erosions, filamentary keratitis, persistent epithelial defects, corneal vascularization, and scarring that significantly compromise visual function. Control of ocular surface inflammation represents a fundamental component of treatment. Short-term topical corticosteroids may reduce acute inflammatory

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activity, while immunomodulatory agents such as topical cyclosporine and tacrolimus provide long-term suppression of chronic inflammation with fewer adverse effects than prolonged corticosteroid therapy. In conclusion, squamous metaplasia is a significant adaptive epithelial response to chronic ocular surface injury by replacement of normal conjunctival epithelium with keratinized stratified squamous cells. Although initially protective, persistent epithelial transformation disrupts tear film function, reduces goblet cell density, impairs ocular surface integrity, and may lead to progressive visual impairment. Chronic inflammation, oxidative stress, vitamin A deficiency, environmental exposure, autoimmune disorders, and dry eye disease represent the principal factors contributing to its development.