Targeted Therapy: Any Place in the Management of Chronic Adenotonsilitis

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Opinion

The mucosal layer of oral cavity and upper respiratory tract has a major role in protecting the human body from infectious agents. Waldeyer’s ring including tonsils and adenoid located at the junction of both the digestive and respiratory tracts provides also an immunological protection [1]. New immune-biological tests have helped in recognition of tonsillar cells responsible for the immunity reactions. Recurrent or chronic adenotonsillar infections mainly occur in children and also may affect healthy subjects. Local dysfunction of the rhino-oropharyngeal epithelium of the host is the main cause of this entity; also some cases may be triggered by the systemic immunological deficiencies. This leads to inappropriate immunity reaction towards the infectious microorganisms that are characterized by recurrent adenotonsilitis [2].

Multiple articles have reported that T lymphocytes located in the tonsillar crypt epithelium may produce pro-inflammatory Th1 type cytokines such: IL-2, IFN-γ, TNF-α and anti-inflammatory Th2 type cytokines: IL-4, IL-5, IL-6, and IL-13 after antigenic stimulation. Production of Th1 type cytokines is usually the dominant. In chronic tonsillitis production of cytokines starts with Th1-type and later on the production of Th2 type cytokines. The role of those cytokines is well understood and heterogeneous [3]. Todorović et al. [3] studied the distribution of cells producing tumor necrosis factor α and interleukin 6 in patients with recurrent tonsillitis and tonsillar hypertrophy. They found that number of TNF-α producing cell were significantly higher in recurrent tonsillitis patients compared with tonsillar hypertrophy patients. The concentration of IFN-γ was three times higher in recurrent tonsillitis patients than in tonsillar hypertrophy patients.

Passali et al. [4] reported that interleukin-1, interleukin-6, and TNF-α were significantly raised in all the specimens of adenotonsillitis patients studied post-operatively. Kheirandish-Gozal et al. [5] did an in vitro study on tonsil and adenoid tissue removed post adenotonsillectomy. In the study the tissue was cultured in corticosteroids and the levels of TNF α, IL-6, and IL-8 were measured. The study found that tonsils and adenoids obtained from children with obstructive sleep apnoea undergoing tonsillectomy and adenoidectomy displayed increased proliferative rates and pro-inflammatory cytokine production. In addition, treatment with corticosteroids resulted in marked reductions in proliferative rates, increased cellular apoptosis and reduced cytokine release.

Sugiyami et al. [6] did a study on the influence of IL-6 on proliferation and differentiation of tonsillar lymphocytes. Tonsillar B cells were cultured in the presence of external interleukin-6, a small portion of them differentiated into plasma cells which seemed to have also IL-6 receptors on their surfaces. The number of plasma cells tended to be greater in the tonsils of children than in the tonsils of adults. Unal et al. [7] reported that serum IL-6 and IL-1β levels were significantly higher in chronic tonsillitis patients than the control. After tonsillectomy, IL-1β and IL-6 levels were significantly reduced. He suggested that IL-1β and IL-6 may be the mediators which play a role in chronic tonsillitis. Desiderio et al. [8] analyzed the structural and immunological aspects of tonsils and adenoids in subjects who underwent adenotonsillectomy because of recurrent adenotonsilitis. He found that serum and tissue interleukin-1β and TNF-α were higher in the adenotonsillar specimens, whereas the IL-6 has a moderate rising.

Esteitie et al. [9] studied the effect of fluticasone on interleukin 6 secretions from adenoid tissues. The study showed a production reduction of IL-6 in adenoid tissue obtained from children complaining of obstructive sleep apnea syndrome treated with fluticasone nasal spray. It has also been reported that the chronic tonsillitis may trigger multiple immunologic disorder such sternocostoclavicular hyperostosis, reactive arthritis, rheumatic fever, palmoplantar pustulosis, erythema nodosum,
IgA Nephropathy, purpura nephritis and psoriasis [10]. There are a lot of controversies about the benefits of tonsillectomy in the previous immunologic disorders, but what about the using of targeted anti-inflammatory therapy in those patients, can we use this therapy also to prevent those autoimmune diseases in chronic tonsillitis patients at high risks.

Targeted anti-inflammatory therapy using anti-IL6, anti-IL4, and anti-TNFα may have a role in the management of chronic adenotonsillitis. It could be tried as a palliative therapy for chronic adenotonsillitis patients who are contraindicated for surgery due other Anastasia or health problems. It could be also tested as adjuvant or alternative to long-acting penicillin protective therapy for the patients of rheumatic fever.

Reference