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Malacoplakia: A rare entity

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Abstract
Malakoplakia is a relatively uncommon chronic inflammatory reaction of unknown etiology. It usually affects the genitourinary tract but may rarely involve the tongue. There are many theories that explain this reaction but it seems to be the answer to an infectious agent in a patient with immunologic deficiency. Microscopically, malakoplakia is characterized by the presence of foamy histiocytes with distinctive basophilic inclusions, which are known as Michaelis Gutmann bodies due to a partially ingested bacteria and their posterior calcification. There are many alternatives to treat this entity.

Keywords: malacoplakia, infectious

1. Introduction

Malacoplakia is a chronic inflammatory disease of unknown aetiology, described initially to be found in the bladder by Michaelis and Gutmann in 1902 [1-3]. The following year, von Hansemann described nodules of yellowish brown soft tissue with central umbilication and coined the term malacoplakia for this condition from the Greek words malakos = soft and plakos = plaque.It is usually reported in genitourinary tract although it has also been reported in different sites such as the gastrointestinal tract, retroperitoneum, and less common in lungs, bones, mesenteric lymphatic nodules, medium ear, larynx, palatine tonsil, parotid gland, temporal bone, neck [2] and tongue [4-7]. The age of presentation varies between 6 weeks of life to 85 years, being more frequent in adult age that in childhood. The distribution is equal in both sexes although in bladder the predilection is for the female sex [4].

Histologically, there is a tissue with numerous macrophages (von Hansemann cells) some of which show eosinophilic cytoplasma; these macrophages have partially digested bacteria due to a defective phagolysosomal Activity and lead to the deposition of calcium And iron resulting in a basophilic inclusion structure, the Michaelis-Gutmann body, considered the hallmark for the diagnosis of malacoplakia [2, 3, 7, 8].

The aetiology remains unknown but three possible theories have been postulated to explain the unusual reaction in this lesion: [3] Microorganisms with unusual toxic properties, immune anomaly that affects the intracellular Death of the microorganisms and enzymatic deficiency of the macrophage to destroy phagocytized bacteria.

Therapy with choline agonist (bethanechol chloride) has been used to correct the decreased cGMP levels that are believed to interfere with complete bacterial killing; ascorbic acid has been used to increase cGMP and cyclic adenosine monophosphate (cAMP) levels in monocytes and to increase synthesis Of collagen fibers;And antibiotics against Gram negative microorganisms (trimethoprim-sulfamethoxazole, quinolone) because of the possible infectious origin and surgical procedures to excise the mass [8].

2. Discussion

Malacoplakia is a disease that rarely involves the mouth. Because the aetiology remains unknown, immune deficiencies are tested in these patients [1, 3, 4] like sarcoidosis, lymphoma, carcinoma, diseases associated to disturbances of the lymphocytes T or immunity mediated by cells, hereditary immunodeficiencies [1] or a history of immunosuppression due to some diseases or long-term therapy with systemic corticosteroids.

Different authors believe that the chronic inflammatory reaction in malacoplakia is an answer to an infectious agent. Electron microscopy studies found intracellular microorganisms forming the matrix of Michaelis-Gutmann bodies.

These studies have also shown remnants products of an incomplete bacterial digestion inside the phagolysosomes due to a decreased intracellular cyclic guanosine monophosphate (cGMP) level [8]. Calcification is initiated in the central zone showing a concentric lamination (owl eyes). Inside the macrophage, one or several intracitoplasmatic basophilic round or sometimes irregular inclusions are observed with PAS stain. Viruses, fungies, bacterias, tuberculous bacilli and other infectious agents have been implied, but none of them show sufficient evidence to be considered causal agents [1]. Some of the most studied infectious agents have been E. Coli and Klebsiella because they have been cultured occasionally in urine, blood and clinical wounds. The serotipification of the E. Coli, does not show microorganisms with greater virulence.The anecdotic cases of focal malacoplakia in chronic periapical periodontitis support the opinion that lesions characterized by macrophage accumulation facilitate the local condition for the production of Michaelis-Gutmann bodies. These could be related to local bacterial antigen load due to a pulpar necrosis [9].

Treatments with antibiotics, such as quinolones and trimetoprim - sulphamethoxazol combined with surgery have better results. Also the combination of surgery with bethanechol (cholinergic agonist) has been used, because it increase levels of cGMP; although vitamin C has been used with an insufficient number of patients as a treatment method, it has employed to increase the cGMP and cAMP levels in monocytes and has the ability to induce collagen fibril synthesis and tissue repair.

3. References

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