LETTERS TO THE EDITOR
CASE REPORT

Elderly male complaining of life-threatening oral swelling

Dear Editor,

As the prevalence of oral lesions has increased in older adults, there is a strong interest in the oral health of older adults. In this vast group, there are several conditions more characteristic for the aging and elderly. In this context, there are conditions ranging from the non-neoplastic type, generally represented by inflammatory and reactive lesions, through to true neoplasias that might be of a benign or malignant nature. We aimed to present an aberrant case of life-threatening gingival maxillary pyogenic granuloma in an elderly patient.

The patient, a 68-year-old man, sought attention complaining of “a large lump in the cheek that prevented him from eating”. Anamnesis revealed a painless growth that had developed over a period of 8 months. The extraoral exam showed a swelling in the left masster-Buccinator region causing extensive facial deformity. The oroscopy showed an edentulous patient, presenting an increased gingival mass with its largest diameter measuring approximately 9 cm (Fig. 1). Histopathological examination of the fragment removed by incisional biopsy revealed a fragment of conjunctive tissue devoid of epithelial lining and covered by granulocytic membrane, presenting dense leukocytic inflammatory infiltrate mixed with polymorphonuclear granulocytes, intermingled with neutrophils and numerous blood vessels of various calibers, featuring the diagnosis of pyogenic granuloma. The immunohistochemical analysis showed evidence of intense marking for CD31, CD34 and muscle-specific actin (HHF35) in blood vessels walls, demonstrating high vascular proliferation, characteristic of reactive lesions. The lesion was completely removed by local surgical excision in a hospital environment under general anesthesia because of the patient’s age and the large dimensions of the lesion. The present case reinforces the importance of a detailed differential diagnosis of gingival lesions in elderly patients. Among the non-neoplastic proliferative processes that affect the gingiva in the maxillary and mandibular region, we could include the peripheral giant cell granuloma, peripheral ossifying fibroma, plasma cell granuloma and pyogenic granuloma. In addition, the history of relatively rapid growth, reaching large dimensions in just 8 months, in association with minimal bleeding on trauma, led us to include other lesions with more aggressive behavior in the differential diagnosis: ossifying fibromixoid tumor, epithelioid hemangioendothelioma and angiosarcoma. The pyogenic granuloma is usually treated by surgical excision. Similarly to the procedure carried out in the present case, in addition to removing the lesion, it is fundamental to include a small safety margin in depth, including the periosteum, as well as removing the irritant factor, with the purpose of preventing recurrences.

The present report, despite being rare because of the lesion dimensions, should warn professionals who routinely make diagnoses of different oral lesions to be permanently alert to the different clinical nuances that the most varied benign and malignant diseases might present in elderly patients, considering that the incorrect diagnosis of a lesion could lead to unsatisfactory results from an aesthetic and functional point of view, and impact negatively on the quality of life of these elderly individuals.

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Dear Editor,

Arterial aging is a dynamic and systemic process characterized by structural and functional changes of blood vessels that exceed the physiological adaptations of the arteries over time. The chronic exposure to cardiovascular (CV) risk factors dramatically accelerates age-associated arterial burden. In Western countries, diabetes mellitus and metabolic syndrome (MetS) have mounted to epidemic proportions, particularly in the older population.1 Cognitive and depressive disorders are among the leading conditions causing disability in older individuals, as either are tightly associated with decreased quality of life, deterioration in daily living activities, social relationships reduction, sleep disorders, and poorer medical outcomes.

Mood disorders, especially those with late-onset, and cognitive impairment/dementia, are likely to overlap. Such association is not surprising, as a growing body of evidence has reported that there could be common underlying biological mechanisms.1 Mood and cognitive disorders in late life were attributed to age-associated neuronal degenerative changes, until this paradigm was challenged by observations that small vessels subcortical injury and CV risk factors are potent independent predictors of late-onset depression1,2 and cognitive disorders,3,4 even of sporadic Alzheimer’s disease (AD), the most common form of dementia worldwide, classically attributed to a pure idiopathic etiology.

Several pieces of evidence have reported that depression with onset in late life is closely associated with systemic atherosclerotic vascular diseases, even at a preclinical stage, as well as with the presence of classical CV risk factors. The presence of diabetes and even more of MetS, with its nexus of metabolic and CV features, are risky for the brain, especially if accompanied by activated systemic inflammation. Nowadays, C-reactive protein is considered as an independent CV risk factor, and its concentrations are typically raised in patients with MetS.1,2 Several investigations have reported that patients suffering from mood disorders, especially in late life, are more likely to show higher plasmatic inflammatory activity,1,2,5 which can directly affect mood by impacting on different pathways, such as those regulating neurotransmission and modulating neuronal plasticity. At the same time, depressive symptoms occur more frequently in patients with MetS1,2,7 making it conceivable to assume that inflammation could represent a main broker between CV diseases and depressive disorders, especially in the elderly.

Likewise, evidence for an independent association with CV risk factors has been provided for cognitive impairment and many forms of dementia, even in patients free of vascular brain lesions.6 CV risk factors are potent predictors of AD, as well as of non-dementia cognitive impairment. Also, in this case, MetS has been shown to predict cognitive impairment, although others have suggested that such association might cease to be significant after adjustment for the presence of silent white matter lesions.7 In marked contrast to this opinion, we have previously described that patients with MetS, free of clinical CV diseases, show poorer cognitive functioning even in the absence of cognitive


References

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