DIFFUSE SCLEROSING OSTEOMYELITIS OF MAXILLA AND MANDIBLE: REVIEW OF THE LITERATURE AND CASE REPORT.

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Sclerosing osteomyelitis of the mandible and maxilla has been described as a special form of chronic osteomyelitis, which generally starts from a low grade infection and manifests itself in the form of multiple osteosclerotic areas(1). The disease is usually classified into focal and diffuse forms(2,3). The focal type is more common and known as condensing osteitis(3). The diffuse type is uncommon and usually confined to the mandible(4).

Diffuse sclerosing osteomyelitis (DSO) is also known as: primary chronic osteomyelitis(5), nonsuppurative osteomyelitis(6), osteomyelitis sicca(7), reactive hyperplasia of bone(8), sclerosing osteitis(9), osteosclerosis, enostosis, bone whorls, bone eburnation, hyperostosis and ossifying osteomyelitis(10,11).

The disease has a pronounced chronic course with repeated episodes of pain, swelling and may be accompanied with trismus and often resist antibiotic treatment. Mild suppuration and chronic

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ulceration of the covering mucosa may occur. Systemic manifestations are usually absent (1,2,9, 12).

The roentgenographic features of this condition are rather characterized with ill defined osteolytic zones, irregularly scattered in more or less dense sclerotic bone. Periosteal deposition of bone can be found especially during acute episodes and in younger persons (13). The radiopaque area may involve a considerable portion of the jaw, with indistinct border between sclerotic and normal bone (3,8). Lebland and Leacock (9) followed several cases for a long period of time and described in detail the roentgenographic and clinical changes occurring in this disease. They noted that the early lesions show, on roentgenographic examination, a region of dense osteosclerosis with rounded or lobulated margins and no signs of osteolytic changes. These changes tend to occur at a level in bone corresponding to the roots of teeth. In the intermediate lesions, there is expansion of the alveolar crest. Radiographic examination reveals irregular density and no osteolytic changes. In the late lesions, further alveolar expansion occurs and infection develops, leading to osteolytic changes separating the lesion from surrounding bone and eventual sequestration of the affected bone.

Microscopic examination of these lesions reveals marked proliferation of dense, irregular bone trabeculas. Some areas show active osteoblasts, but «elastic» cells were not observed. There is a reduction in the marrow spaces, which are filled with loose fibrous tissue containing variable numbers of inflammatory cells (3,8).

The etiology is unknown, but is generally thought to be an infection of low virulence, together with high host resistance (1,12).

The reaction of bone to injury is influenced by a number of factors; including age of the patient, local blood supply, tissue resistance.
and degree of injury. Occasionally, bone will react in an abnormal way to injury; Robinson (14) has termed this altered reaction "ossaceous dysplasia".

Shafer (2) is of the opinion that chronic sclerosing osteomyelitis is a form of osseous dysplasia, the injurious agent being a form of low-grade, chronic, nonspecific infection.

Diffuse sclerosing osteomyelitis is relatively a rare disease and occurs most frequently in middle-aged and older persons. Females are more frequently affected than males. It may occur in any race, however, most of the reported cases were Negroes. The disease had been reported to affect the mandible more frequently, but rarely the maxilla may be involved (1-3, 8, 9, 11-13, 15). Bell (1) in his review of 5 cases, he noticed that they were all Negro females, in three cases the mandible only was affected, while in the other two cases both mandible and maxilla were involved.

Jacobson (4) followed up 21 cases of diffuse sclerosing osteomyelitis they were all affecting the mandible. Waldron (8) followed 38 cases, he reported only 3 of them involving maxilla.

Treatment therapy remains a problem because of insufficient understanding of the etiology. Different lines of treatment have been tried, including antibiotics (3, 6), decortication (5), and hyperbaric oxygen (16).

Case report:

A 40-year-old dark lady was referred to the outpatient clinic of Oral Surgery Department of Faculty of Oral and Dental Medicine, Cairo University at June 1986. Her chief complaint was pain swelling in the upper left premolar region. These symptoms had been getting