PLASMA CELL CHEILITIS

To the Editor,

An 18-year-old male student presented with asymptomatic, bilaterally symmetrical, well circumscribed, soft, slowly progressive lower lip swelling of 4 years duration. It involved the vermilion surface and mucosa of lip with red glistening surface without ulceration. There was no history of using dentures, smoking, chewing gum, mouth wash, drugs, radiation, trauma, contact allergy, candidal infection, prolonged light exposure, facial palsy or lingua plicata.

Investigations including haemogram, urinalysis, blood urea nitrogen, serum alkaline phosphatase, calcium, phosphorous, X-ray chest, VDRL test and scraping for KOH examination were within normal limits. Histopathological examination revealed parakeratosis, acanthosis with intercellular oedema, elongated rete ridges and an upper dermal predominantly plasma cell infiltrate in a band like pattern clinching the diagnosis of plasma cell cheilitis.

Plasma cell orificial mucositis is a benign idiopathic condition of the orificial mucous membranes, characterised histopathologically by a dense plasma cell infiltrate. Although this phenomenon was first described by Zoon in 1952, as occurring on the glans penis; conditions similar to plasma cell orificial mucositis involving vulva, buccal mucosa, palate, nasal aperture, gingiva, lips, tongue, epiglottis, larynx and other orificial surfaces have been reported under various names.[1] European authors have designated the process when it occurs on the lip as "cheilitis plasma cellularis".[2] Scheuermann in 1960 and Luders in 1972, simplified the nomenclature by grouping these anatomic variants under the titles "plasmacytosis circumorificialis" and "plasmacytosis mucosae".[1] It repesents a stage in the immune response to any one of a variety of stimuli, benign or malignant.[2]

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References

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HYPERVITAMINOSIS -A

To the Editor,

A 10-year-old boy was being treated for night blindness with injectable and oral vitamin A for more than 3 months. He presented to us with well circumscribed itchy, dry, scaly, follicular lesions over knees, elbows and knuckles present for 15 days. Scalp hair was sparse, thin and dry. He complained of headache. There was no history of chest pain, palpitation or lymphadenopathy. There was no neurological deficit. Fundoscopic examination did not reveal any abnormality. Routine blood and urine analysis were within normal limits. Skeletal X-rays showed mild periosteal reaction. On histopathological examination moderate hyperkeratosis and focal inflammatory cell infiltration were seen.

Hypervitaminosis A occurs in an acute form due to ingestion of a large single dose of vitamin A and in a chronic form due to prolonged ingestion of more than 50000 iu of vitamin A.[1] Though serum vitamin A levels could not be done in our patient due to lack of facilities, the remarkable clinical improvement on vitamin A and restriction of vitamin A rich diets without any other medications pointed towards the clinical diagnosis of

hypervitaminosis A.

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PREHERPETIC NEURALGIA MASQUERADING AS ANGINA

To the Editor,

A 46-year-old male patient presented to the emergency department with continuous dull pain on the left side of chest radiating to the medial aspects of left arm, forearm and hand. The pain started in the evening when the patient was engaged in indoor activity. After 4-6 hours the intensity of pain increased and it was associated with epigastric discomfort, sweating, palpitation and fear of impending death. There was no history of dyspnoea, unconsciousness, or any past history of similar complaints. He was a chronic alcoholic and tobacco chewer. The patient was admitted to intensive care unit about 12 hours after the onset of pain with the provisional diagnosis of angina pectoris. Physical examination revealed tachycardia. There was no other abnormal physical finding. His following tests were within normal limits: total and differential leukocyte counts, erythocyte sedimentation rate, cardiac enzymes and electrocardiogram.

With the diagnosis of angina pectoris, he was prescribed analgesics, isosorbide dinitrate, and aspirin. The pain improved slightly but it did not disappear.

Thirty-six hours after the onset of pain, the patient developed vesicles in groups on erythematous base over the left side of chest, medial aspect of arm, forearm and medial two finger involving C8-T3 segments of cutaneous innervation. A Tzanck smear from the lesions showed characteristic multinucleated giant cells. The diagnosis was changed to herpes zoster with neuralgia. Antianginal drugs were stopped and the patient was given acyclovir 800 mg 8 hourly. Pain reduced considerably within a day and in 2 weeks the lesions healed completely. This case is being reported to highlight the need of keeping preherpetic neuralgia in the differential diagnosis of cardiac pain.

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