Case Report:

Scorbutic gingivitis – a case report

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Abstract:
Scurvy, a rare disease of dietary deficiency of vitamin C is mostly seen in malnourished person and a diet devoid of citrus fruits and green vegetables. The classical picture of scurvy consists of loose teeth, dark red swollen hemorrhagic gingiva, swollen joints and haemorrhages. Histopathologically it shows signs of nonspecific inflammation with hyperaemia of papillary vessels and decreased no. of collagen fibres. A rare case of scorbutic gingivitis is reported where diffuse generalised erythematous gingival enlargement with hemorrhagic tendency and severe generalised mobility of teeth was present.

INTRODUCTION:
Scurvy is a nutritional deficiency disease caused by a prolonged inadequate uptake of Vitamin C.1 Vitamin C is water-soluble substance, very sensible to heat, ultraviolet or oxygen exposure. The total pool in the body is 1500-2500 mg. Its absorption occurs in the ileum but is not stored in the body, so the dietary intake is indispensable. To prevent scurvy the daily intake must be minimum 10 mg/day.

Vitamin C functions, both as a reducing agent and as an antioxidant, is necessary for many physiologic functions, including metabolism of iron and folic acid, resistance to infection, and integrity of blood vessels. Vitamin C is required for prolyl and lysyl hydroxylase activity and is essential for collagen synthesis. Defective collagen composition compromises skin, joint, gums, mucosal, bone, and vascular integrity.

Extraoral lesions may be observed, such as hemorrhage into the skin and mucosa; which may range from multiple, small, perifollicular zones to large ecchymoses having no relation to the amount of trauma. Scorbutic gingivitis is an important feature of this disease. The gingival tissues are swollen, spongy, and bleed easily. Ecchymoses may be present in the palate, cheeks, or floor of the mouth. Necrosis and infection may be further complications. A pronounced hypochromic anemia and hyperkeratosis of hair follicles is often present. Biochemical assay of the plasma ascorbic acid level is a valuable test. The range considered essential for good health is 0.7mg per 100c.c. to 1.40mg per 100 c.c. The recommended daily allowances (intake) are 75mg. for males, and 70 to 150mg for females.

Microscopically, the gingiva is oedematous and hemorrhagic. There is marked collagen degeneration, together with acute and chronic inflammatory infiltration. In most cases the response to vitamin C administration as recommended is prompt and dramatic. The prognosis...
of scurvy is good as the disease is readily amenable to treatment with ascorbic acid.\(^1\)

The case of scorbutic gingivitis is presented in a 19 year old female with classical oral manifestations which is rarely seen in contemporary patients.

**CASE REPORT:**

A 19 year old female patient reported to the Oral diagnosis and Radiology department, G.D.C.H, Ahmedabad with the chief complaint of swelling in relation with upper left posterior region since 2 months with associated complaint of bleeding, swollen gums and mobility of all teeth since 3 months.

The patient was asymptomatic 3 months back. Then she noticed swelling in the gums with slight bleeding tendency especially on brushing teeth and mobility of teeth. Subsequently she noticed swelling in relation with palatal gingiva of upper posterior teeth which was very small initially. Gradually it increased upto the present size. She went to various private dentists where medications were given and incision and drainage was done but to no avail. She had experienced loss of appetite and general lethargy over a corresponding period of time.

Her medical and family history was essentially unremarkable. Patient was in a habit of chewing unilaterally on the right side without any other oral destructive habit.

On examination the patient was pale. Intraorally, there was generalized gingival enlargement with boggy and diffuse erythematous appearance involving interdental papilla, marginal gingiva and attached gingiva of all teeth. No disturbance of occlusion was present. (Fig. 1a) There was a single well defined localised growth of gingiva on left side of hard palate of size about 3x3 cm extending from mesial surface of 26 to distal of 27 anterioposteriorly and from marginal gingiva of 26 and 27 to 3 cms towards the midline of palate mesiodistally. The overlying mucosa was erythematous, shiny, and smooth. (Fig. 1b) On palpation the palatal growth and the enlarged gingiva was nontender, soft to firm in consistency.

Heavy deposits of subgingival plaque and calculus were noted in relation with 26 and 27 while other teeth showed moderate deposits. There was 3 degree mobility of 16, 26, 27, 37 and 47 and 1-2 degree mobility along with bleeding on probing in relation with all teeth.

The above clinical findings were in favour of scorbutic gingivitis with pyogenic granuloma in relation with left side hard palate. Hormonal gingival enlargement as well as juvenile periodontitis can be considered in differential diagnosis considering the age, sex and clinical appearance of the lesion.

Various investigations such as radiographs and blood investigations were carried out to confirm the diagnosis and to exclude other possibilities. Intraoral periapical views (fig.2a,b), and orthopantamograph (fig. 3) revealed severe generalised horizontal bone loss in relation with all teeth while 26 and 27 showed more extensive bone loss with floating tooth appearance.

In addition a full blood examination was requested. Reported abnormalities included low haemoglobin concentration of 8 gm% and slightly raised ESR of 14mm at 1 hour and 30mm at 2 hrs. Ascorbic acid assay was reviewed and found to be reduced to about 0.04 mg/dl. (normal range - 0.20 to 2.0mg/dl). The clinical diagnosis of scorbutic gingivitis was thus confirmed.

The patient was treated with vitamin C supplements in the form of 1 gm/day orally divided into 2 equal doses and was given dietary advice to
increase her vitamin C uptake. Along with this ferrous sulphate (200mg BD) and multivitamins were given. Anti-inflammatory (diclofenac 50mg BD), antibiotic (metronidazole-400mg TDS) and chlorhexidine mouthwash were given to subside inflammation, prevent secondary infection and for oral hygiene maintenance respectively. Follow up after 7 days showed marked improvement in the patient’s condition and the gingival tissues appeared firm, with decrease in erythema and bleeding tendency. Acute features of gingival involvement subsided within 2 weeks of same therapy.

Vitamin c (Ascorbic acid) plasma level was evaluated after 2 weeks of systemic therapy of vitamin C supplementation which was 1.08 mg/dl.. Drastic response with vitamin C therapy confirmed the diagnosis as scorbatic gingivitis. Surgical excision of pyogenic granuloma along with extraction of 26 and 27 was done under local anaesthesia after 1 month. The specimen was sent for histopathological examination and was consistent with the diagnosis of pyogenic granuloma with fibroepithelial polyp in relation with left side of hard palate. [Figure 4] The tissues healed well postoperatively. [Figure 5] The patient is currently under maintenance therapy of 100mg vitamin C daily in the form of oral tablets for 3 months.

**DISCUSSION:**

Scurvy is a nutritional deficiency disease of vitamin C characterised by purpura, bleeding, loose teeth and gingival abnormalities rarely with musculoskeletal abnormalities, found mainly in infants, elderly and persons whose diet lack in fresh fruits and vegetables, poor oral hygiene and malnourishment. The present case was of a 19 year old female patient with generalised diffuse gingival enlargement with bleeding tendency and loose teeth and pyogenic granuloma on left side of hard palate.

Clinically, the signs of scurvy develop after 1-3 months of inadequate vitamin C intake, depending on existing body stores. Frequently, oral lesions of scurvy precede other manifestations of disease characterised by gingival swelling, ecchymoses, bleeding gums and loosening of the teeth. Cutaneous manifestations like haemorrhages in the form of petechiae, ecchymoses or hematomas are late findings. The present case demonstrated the early manifestations of scurvy with classical oral signs; however the patient was without any systemic or cutaneous manifestation which occurs late in the disease.

Along with the oral signs of scurvy, there was a single well defined localised growth of gingiva on left side of hard palate in relation with 26, 27 teeth, with erythematous, shiny, and smooth overlying gingival due to habitual unilateral chewing on the opposite side and heavy deposits of plaque and calculus on the left side.

Radiographically, there is widening of periodontal ligament space in early stages with marked interdental bone loss and loss of lamina dura in severe cases. The present case demonstrated similar changes which is a late feature of the disease however the patient already had generalized periodontitis having heavy deposits of subgingival plaque and calculus. It is likely that vitamin C deficiency was a factor in accelerating the periodontal destruction. There was more bone destruction in the region of pyogenic granuloma in relation with 26, 27 due to severe deposits of local irritants, more inflammation and vitamin C deficiency.
Biochemical assay of the plasma ascorbic acid level is a valuable test in scurvy which is below 0.2mg per 100c.c. and there is marked improvement in the plasma level once the ascorbic acid stores are restored. The present case also showed normal plasma levels of vitamin C after it was supplemented orally.

Anemia is another hallmark of scurvy. Ascorbic acid improves iron absorption by reducing it to the more absorbable ferrous state. The present case also showed classical signs of iron deficiency anaemia with pale conjunctiva, labial mucosa, skin and nails having longitudinal ridges and fatigue on exertion. Aggressive periodontitis with inflammatory gingival enlargement can be considered as a differential diagnosis because of severe generalised mobility of teeth in an adolescent female. However, in aggressive periodontitis there is angular bone loss and absence of local factors. Heavy deposit of subgingival calculus and horizontal bone loss rule out the possibility of aggressive periodontitis in this patient.

Histopathological section of gingival growth on the palate showed hyperplastic epithelium with endothelium lined vascular spaces and fibroblasts between sheets of collagen fibres consistent with the diagnosis of pyogenic granuloma with fibroepithelial polyp. Finally the best evidence of scurvy is the resolution of the manifestations of the disease after ascorbic acid treatment. Treatment consists of a regimen of vitamin C supplementation. Recommendations are that 1 to 2 grams of vitamin C be administered daily for the first 2 to 3 days followed by 500 mg per day for the next week and 100mg daily for next 1-3 months with oral hygiene maintenance. The treatment of scurvy in our case was administration of vitamin C 500 mg twice a day for 2 weeks followed by maintenance therapy. This resulted into marked resolution of the symptoms. For the pyogenic granuloma, surgical excision was performed after 1 month so that tissues can heal once the normal levels of Vitamin C was achieved.

Frequently the diagnosis of scurvy is based on clinical grounds and the response to dietary intake of ascorbic acid not necessitating histopathological confirmation. The typical appearance of swollen, spongy and bleeding gums with loose teeth as in our case and prompt response to vitamin C therapy confirmed the diagnosis of scurvy.

CONCLUSION:
Adult scurvy is a specific dietary deficiency disease which is rarely seen nowadays. The very striking oral features, should suggest vitamin C deficiency. A heightened awareness for a detailed clinical and radiological examination is needed to avoid unnecessary tests and procedures and to be able to implement treatment for a potentially fatal but easily curable disease.

REFERENCES:


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Figure 1. (a) Intraoral photograph showing teeth in occlusion and erythematous and boggy gingival enlargement of anterior teeth

Figure 1. (b) Intraoral photograph showing gingival growth in relation with26, 27.

Figure 2 Intra Oral Periapical view of (a)24,25,26,27 (b)36,37,38 showing interdental horizontal bone loss in relation 26, 27,36,37,and floating tooth appearance of 27.
Figure 3. Oral pantomograph showing generalised horizontal interdental bone loss with floating tooth appearance of 27.

Figure 4. Photomicrograph showing hyperplastic epithelium with endothelium lined vascular spaces and fibroblasts between sheets of collagen fibres.

Figure 5. Intraoral photograph showing healing ulcer after gingivectomy and extraction in relation with 26 and 27.