

Oral lesions in scurvy

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Abstract

Scurvy is the nutritional deficiency state associated with lowered levels of ascorbic acid (vitamin C). Lack of ascorbic acid leads to suppression of collagen synthesis and the synthesis of defective collagen among other metabolic derangements. Weakening of vascular walls results in signs and symptoms mimicking other diseases such as bleeding diatheses and deep vein thrombosis.

Scurvy is rarely encountered in Western countries where there is a broad community understanding of the importance of nutritional requirements and where foods containing ascorbic acid are readily available.

As a result of these factors early diagnosis may be hampered where it is not considered in the differential diagnosis, and consequently, prolonged suffering of the patient. Scurvy is easily treated with high doses of oral ascorbic acid, although recurrences may occur.

Education of health care providers in recognizing the signs and symptoms of scurvy therefore cannot be over emphasized, particularly in societies in which nutritional deficiencies are considered uncommon.

A case of scurvy presenting primarily with oral manifestations is reported here.

Key words: Ascorbic acid, case report, gingivitis, oral medicine, scurvy, vitamin C.

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INTRODUCTION

Guinea pigs, primates and humans cannot synthesise ascorbic acid (vitamin C) due to deficiency of L-gluconolactone oxidase.¹ Therefore, a diet not including ascorbic acid will lead to clinically manifested scurvy. The history of scurvy, a nutritional deficiency with significant morbidity, includes at risk groups as diversified as sailors on long intercontinental voyages and infants receiving proprietary foods provided by their affluent parents in the late nineteenth century.¹

Ascorbic acid, once isolated, has been manufactured synthetically for over 50 years. As a result of this and an awareness in the population that nutritional intake

of ascorbic acid containing foods was essential, the prevalence of scurvy has been very low.

A small number of individuals in any community develop scurvy and clinicians should still be aware of this disease and investigate and manage appropriately. Symptoms of scurvy include haemorrhage related symptoms (including epistaxis, haemorrhage into joints, muscles and deeper tissues), hyperkeratosis of hair follicles and anaemia. In order to highlight this, a case is presented here in which the primary symptom leading to diagnosis and management was gingival haemorrhage.

CASE REPORT

A 19-year-old male was referred to the Oral Medicine Clinic, The Royal Dental Hospital of Melbourne with a three week history of 'bleeding gums'. Onset had been acute, but he had noted some improvement since commencing using a proprietary mouthwash (Listerine). He was aware of having to expectorate blood over the preceding few weeks. He had experienced loss of appetite and general lethargy over a corresponding period of time, particularly at his workplace, where he was employed as a carpenter.

His medical history was essentially unremarkable. He experienced asthma occasionally, which was relieved by salbutamol. He was a non-smoker. Recently he had taken a course of oral penicillin for tonsillitis.

On examination the patient was pale (claiming to be feeling ill following excessive alcohol consumption the previous evening). A non-tender submandibular lymph node was palpable on the right side. Intraorally, the labial free gingival margins adjacent teeth 13, 12, 11, 21, 22, 23, 32 and 42 were erythematous with spontaneous haemorrhage (Fig 1, 2). A moderate amount of plaque was noted. The rest of his oral mucosa was somewhat pale, but no other specific mucosal lesions were noted. No periodontal pocket depths of >3mm were noted. A provisional diagnosis of chronic marginal gingivitis with an acute exacerbation, possibly associated with a blood dyscrasia, was made and the patient was given oral hygiene instruction, prescribed chlorhexidine gluconate mouthwash and referred for haematological investigations. In addition to a full blood examination, serum iron, ferritin,

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Fig 1. A clinical photograph at the time of presentation showing erythematous spontaneous bleeding from the labial gingival margins adjacent teeth 13, 12, 11, 21, 22 and 23.



Fig 2. A clinical photograph at the time of presentation showing erythematous spontaneous bleeding from the labial gingival margins adjacent teeth 32 and 42.

vitamin B12, ascorbic acid and red cell folate were requested. Reported abnormalities included a mildly elevated mean corpuscular haemoglobin concentration and a markedly reduced serum ascorbic acid level (14µmol/L, range 40-120µmol/L) (Table 1).

Two weeks later, he was reviewed when the results of haematological investigations were available. He had noted some improvement in the interim, which was attributed to the use of chlorhexidine. On examination gingival erythema and spontaneous haemorrhage were noted, particularly on the anterior maxillary labial aspects between teeth 13 and 23. He was given dietary advice and instructed to increase his ascorbic acid intake. One month later, a marked resolution had occurred and the gingivae appeared healthy. Haematological investigations at that time showed a marked increase in his serum ascorbic acid level (56µmol/L). Subsequently, he was lost to follow-up.

DISCUSSION

Ascorbic acid is present in cow's milk, kidney, liver, fish and in many fruits and vegetables. However, it is destroyed by cooking and prolonged storage. Foods rich in ascorbic acid are abundant in Australia, but some groups of individuals may not maintain sufficient dietary intake. This includes some 'vegetarians'

Table 1. Results of haematological investigations.

Date	22/12/95	09/02/96	Range	Units
Haemoglobin	148	146	130-180	g/L
Leukocytes	10.4	6.4	4.0-11.0	x10 ⁹ /L
Platelets	211	215	150-400	x10 ⁹ /L
Red cells	5.07	5.02	4.50-6.50	x10 ¹² /L
PCV	0.42	0.43	0.40-0.54	
MCV	82.2	84.7	80.0-96.0	fL
MCH	29.2	29.1	27.0-32.0	pg
MCHC	355H	344	300-350	g/L
RDW	13.7	13.5	11.0-15.0	%
MPV	10	10.6	7.4-10.4	fL
ESR	*****	7	2-10	mm/hr
Iron	10.8	19	7.0-35.0	umol/L
Transferrin	2.93		1.91-3.22	g/L
Transferrin sat	27		20-60	%
Ferritin	209	110	20-400	ug/L
Ascorbic acid	14L	56	40-120	umol/L

including vegans, substance abusers and young adults lacking nutritional education. In addition, those dependent on the care of others may be at risk of developing a deficiency state.

As a disease, scurvy may mimic a number of medical conditions including vasculitis, systemic bleeding disorders and deep vein thrombosis.² The disease may present as haemarthrosis,³ purpuric rash,⁴ refractory anaemia⁵ and inability to walk.⁶ Gingival lesions associated with scurvy have rarely been reported in the last 30 years.⁷⁻¹¹ Oral lesions of scurvy have also been reported following withdrawal from a megavitamin diet.^{12,13} The historical importance of oral lesions associated with scurvy have also been reviewed.¹⁴

To confirm a diagnosis laboratory tests include serum ascorbic acid estimation, white cell or platelet estimation,^{15,16} although these latter tests are not available in all laboratories. Frequently, the diagnosis is based on clinical grounds and the response to dietary intake of ascorbic acid.

Frequently, oral lesions of scurvy precede other manifestations of disease. Therefore, early recognition may facilitate early treatment.¹⁷ Rapid response to ascorbic acid supplementation, as occurred in this case, is a strong indicator of correct diagnosis. Although rare, patients with scurvy with a complaint of 'bleeding gums', may present in general dental practice. Scurvy should be considered in a differential diagnosis along with leukaemia, dermatoses, blood dyscrasias and primary herpetic gingivostomatitis. Touyz^{7,18} reported that of 762 cases fully investigated for periodontal disease in a South African study, five cases (0.66 per cent) of oral scurvy were diagnosed. However, periodontal disease is far more common. It is not recommended to routinely screen all patients presenting with the complaint of 'bleeding gums'.

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