

Oral Medicine

EFFECT OF VITAMIN C ON A SCORBUTIC TYPE GINGIVAL LESION

Report of a Case

R. HARRIS, M.D.S.,* AND G. HUTCHINSON, M.D.S., F.D.S.R.C.S. (ENG.),**
SYDNEY, AUSTRALIA

Introduction

THE significance of changes in the gingival tissues, unless dramatically appearing as in an acute infection or a neoplasm, may be overlooked in present conditions in this country. With full employment and a high living standard, it is rare that a patient is seen with marked gingival conditions which may be definitely diagnosed as scorbutic. The classical picture of scurvy—loose teeth, dark red swollen hemorrhagic gingiva, tender or painful extremities and joints—is rarely, if ever, seen in general practice, and it has been singularly absent in our institutional experience. It was this fact which prompted our report.

That a relationship appears to exist between vitamin C deficiency and the repair of tissue has been recognized; hence, the predisposition for scorbutic lesions to appear in regions of connective tissue activity. Oral lesions thus appear at the gingival margins and the interdental papillae—regions of local inflammation. It is most unlikely for signs of the condition to be found in the edentulous mouth. Deprivation experiments in human subjects, however, have not always resulted in the development of typical oral lesions. The explanation for this is that such subjects have shown an absence of any noticeable degree of inflammation in mouths better cared for than those of the usual clinic patient. The importance of any such deficiency, if marked, cannot be underestimated when surgical intervention is contemplated.

Extraoral lesions may be observed, such as hemorrhage into the skin and mucosa; the hemorrhages may range from multiple, small, perifollicular zones to large ecchymoses having no relation to the amount of trauma.

*Head, Department of Preventive Dentistry, United Dental Hospital, Sydney.

**Clinician, Department of Preventive Dentistry, United Dental Hospital, Sydney.

The requirements of vitamin C are difficult to determine. It has been demonstrated that from a starting point of excellent nutritional status and all-round good health, one may go a long time on a low intake before visible scorbutic changes occur. Sherman and Lanford² report an example of such a deprivation experiment. The level of this vitamin in the blood plasma fell to zero in forty-two days and in the white blood cells in 122 days, while visible symptoms of scurvy appeared in five to six months. The range considered essential for good health is 0.7 mg. per 100 c.c. to 1.40 mg. per 100 c.c.

The recommended daily allowances (intake) are 75* mg. for males, and 70* to 150* mg. for females, but it is important to remember that there is a fairly rapid excretion of vitamin C and that the effective amount of vitamin C in the plasma, after additions to a meal, is dependent upon the original fasting level. If this has been low for a long time, it becomes essential to give large supplements to the diet before a satisfactory level can be maintained.

Diagnosis

The diagnosis of a scorbutic condition has been based upon the presence of extraoral signs (mentioned previously) and changes in the gingivae.

The latter are dark red and swollen, the color tending more toward purple; these changes are probably due to extravasated blood corpuscles. In long-standing cases there may be an anemia. This dark red gingiva shows a ready tendency to bleed and is usually smooth. The teeth in advanced cases are loose and the gingivae are detached. Clinical diagnosis is supported by low plasma and white cell levels and by both a qualitative and a quantitative assessment of the patient's dietary intake. At present, there are no absolute figures for the blood levels, but it has been suggested that below 0.2 mg. per 100 c.c. plasma indicates a severe deficiency,¹ although Krebs³ found that it is unlikely to develop, even where this figure is as low as 0.1 mg. per 100 c.c. plasma. It should be remembered, however, that Crandon was referring specifically to surgical subjects.

Case Report

The patient, H. E., a slightly built man aged 39 years, was first seen Nov. 16, 1953, and appeared undernourished. The only medical history obtainable was that the patient was in receipt of a full pension for a pulmonary disorder, which he stated was not tubercular. He was, at the time, totally unemployed and lived in poor quarters without adequate comfort and in an uninvigorating environment.

Clinical Examination.—The patient, when first examined, was lethargic and unkempt. The skin of the face showed multiple small subcutaneous hemorrhages associated with the hair follicles. The oral hygiene was poor; the mucosa and gingivae appeared redder than normal. Some teeth had been extracted a considerable time previously, and there was extensive bone loss in the upper left molar, lower left premolar, and lower right molar

*These figures are from Sherman and Lanford² and the Food and Nutrition Board, National Research Council, Washington, D. C., and represent the amount necessary for saturation, but Krebs³ says, "So long as there is no evidence to support the view that an intake of more than 30 mg. daily has beneficial effects, there is no basis for recommending an intake greater than that amount."

Fig. 1.



Fig. 2.



Fig. 3.



regions associated with the long-standing periodontitis. The teeth in these regions were markedly mobile.

Three distinct and unusual types of lesions of the gingival margins were noted.

1. *Upper left molar and premolar.* On the buccal aspect, an extremely hyperemic swelling, which became blue-black toward the gingival margin with a gray necrotic marginal area (Fig. 1). The palatal gingiva was also hyperemic.

2. *Lower left molar and premolar and lower anterior teeth.* Small blue-black blebs, the largest about 5 mm. in diameter, which bled swiftly and collapsed on pressure with an instrument (Fig. 2).

3. *Multiple granulations.* Dark pink granulations located at the free margin of the gingival papillae at various situations both labial, lingual, and palatal (Fig. 3).

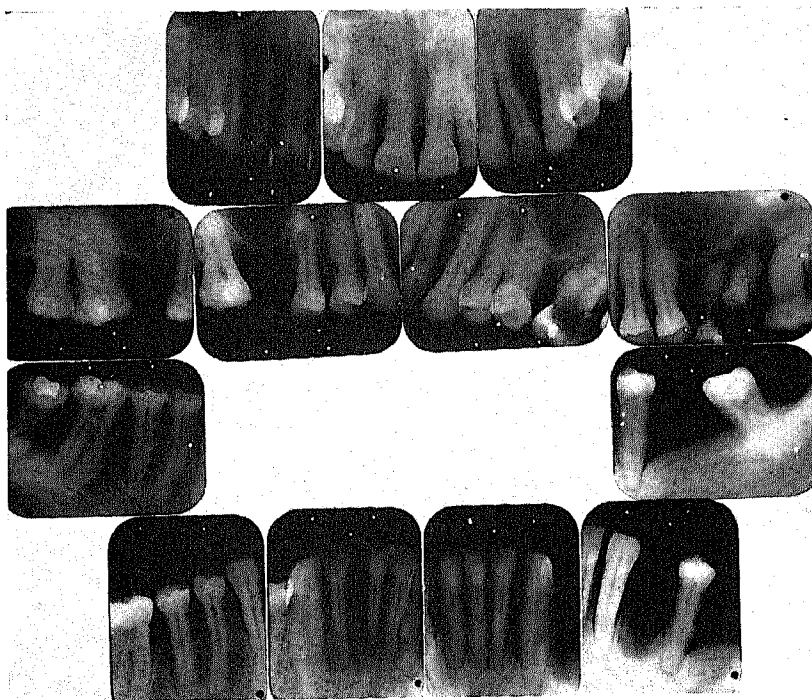


Fig. 4.

Radiographic Examination.—Intraoral radiographic examination revealed extensive bone loss in the upper left molar region with an apical granuloma and probable involvement of the maxillary sinus. This was confirmed by an extraoral radiogram. Other regions of the maxilla and mandible showed areas of bone loss, all associated with intensely inflamed tissue (Fig. 4).

Laboratory Examination.—

1. Hematologic examination:

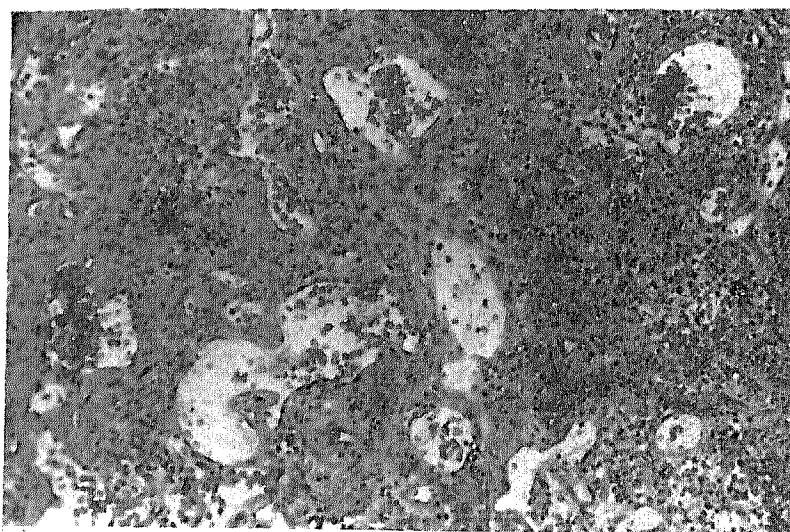
- (a) Blood coagulation time, four minutes.
- (b) Hematologic examination for red and white cells was within the normal range.
- (c) Wassermann reaction and Kahn flocculation test were both negative.
- (d) Blood sedimentation rate was increased at 20 mm. (normal for males 0.9 mm.). (Wintrobe)

2. Plasma ascorbic acid: 0.26 mg. per 100 c.c.

3. Urinalysis: Negative.

4. Biopsy material was obtained from the upper left molar region. *Pathologist's report*: (Upper left molar region) "The section shows a cellular, ulcerating hemangioma. The line of excision does not go beyond the lesion." (Fig. 5, *A*.) A section through the epithelium (Fig. 5, *B*) shows lack of keratinization and the usual peg formation (noted also by Orban⁴).

A.



B.

Fig. 5.—Magnification, $\times 100$.

(See the findings of Orban and his associates,⁴ who reported in their case "[the tissue] is rich in polymorphonuclear leucocytes. . . . The inflammatory reaction is acute, characterised by the presence of a large number of leucocytes in the blood vessels as well as in the tissue. The intercellular substance contains few fibres and is infiltrated by fluid [edema].")

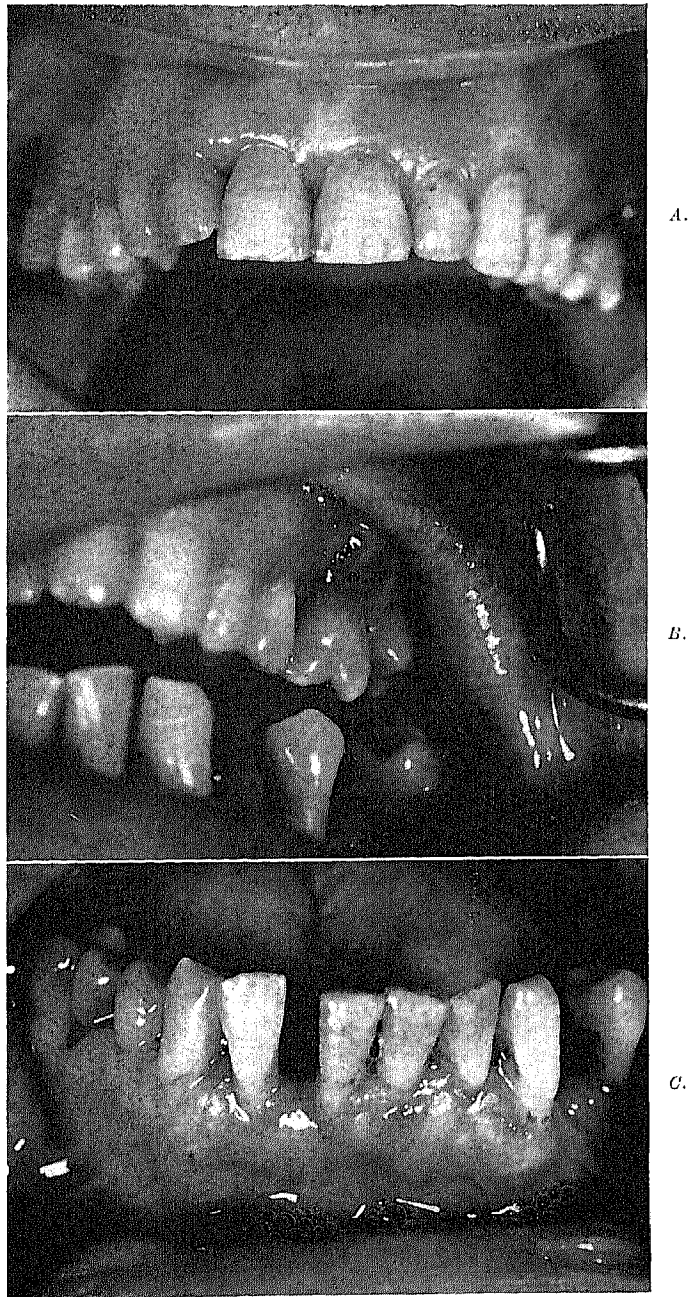


Fig. 6.

Thomas⁷ says there is hyperemia of the papillary vessels and extravasation of the erythrocytes may dominate the picture. In the more advanced cases there is destruction of the epithelium by ulceration and secondary infection. Stones⁶ refers to the absence of collagen fibers and infiltration with inflammatory cells.

Dietary Analysis.—A seven-day detailed dietary record was instituted immediately and revealed the almost total absence of fruit and vegetables, with an average of twenty-six slices of white bread per day, often supplemented by biscuits and cake. On inquiry, the patient stated that the record was a fair sample of his usual diet program for many months past. It is somewhat similar to the case recorded by Orban⁴ in that it has a high carbohydrate content.

Therapy.—Commencing Nov. 25, 1953, the patient was given daily ascorbic acid, 700 mg. orally for three days to obtain saturation, followed by 200 mg. daily for two weeks.

Nov. 30, 1953. The small granulations had disappeared and the wound at the site of biopsy showed all appearance of healing tissue. The oral mucosa and gingiva generally were much paler and the patient appeared more alert than when first seen.

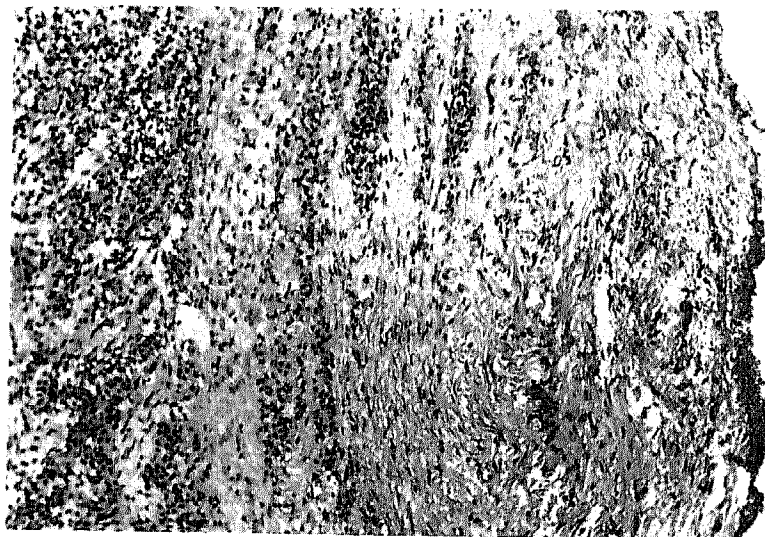


Fig. 7.—Magnification, $\times 100$.

Dec. 9, 1953. The perifollicular hemorrhages in the skin had disappeared; the mucosa of the mouth was normal in color; the intense hyperemia of the lesion in the upper left molar region had subsided; and the gingival tissues as a whole had lost all sign of the granulations previously noted (Fig. 6).

During the therapy a serious attempt was made to influence the patient in the hope that an appropriate dietary schedule could be instituted. Although the patient readily promised to implement the suggestions, we had very little confidence that the effort could be seriously maintained.

On the second and the third day a vitamin C saturation test of the urine was made (Harris and Abbasy²). This test was made six hours after the massive daily dose was given, and the results were as follows:

Second day—36 mg. vitamin C excreted.

Third day—73 mg. vitamin C excreted.

Normally, 50 mg. vitamin C is excreted by the second day and this rather indicates a pre-existing deficiency in the vitamin.² The plasma ascorbic acid had risen to 1 mg. per 100 c.c. on the third day.

Jan. 9, 1954. At this stage appropriate periodontal treatment was instituted. It is important to stress that no local treatment to the gingival condition had been carried out previously.

Jan. 12, 1954. The patient was referred to the Oral Surgery Department. His upper left molars were removed, and at the same time a radical antrostomy was effected through an opening in the alveolus which included the area of the molar socket. An opportunity was taken for further material for biopsy from that region. Healing was completed and patient was discharged from the Oral Surgery Department on Feb. 23, 1954.

The pathologist now reported as follows: "Upper left molar region (material removed at time of surgical treatment)—Section of chronically inflamed gingival tissue." (See Fig. 7, second biopsy.)

A reduction in vascularity and an increase in connective tissue is to be noted.

Comment

The lesions, though not having all the classical and marked signs of scurvy, had, in fact, certain features closely simulating some of them. There were the dark purplish blebs on the gingiva, perifollicular hemorrhages, very low plasma level, and an indication of a severe deficiency. However, it was above the level set by Krebs³ and Crandon¹ as consistent with a clinical diagnosis of scurvy.

It is significant, however, that the peculiar lesions described rapidly resolved following specific therapy and supports our contention that the clinical diagnosis of scorbutic gingivitis had a reasonable basis. This was further supported by the lack of any sharp rise in the urinary excretion of vitamin C until the third day, despite the administration of 700 mg. of ascorbic acid daily.

This case demonstrates the need for thorough investigation of any lesion found in the mouth, the correlation of these findings with those elsewhere, and adequate laboratory examination. On the basis of these findings, an appropriate therapy can be instituted which has every prospect of success. It is important to note that in cases of prolonged avitaminosis there is danger of a regression developing if the patient is unable to cooperate and maintain an adequate dietary level for the appropriate nutritional factor.

Permission to use material presented in this case report was granted by the Superintendent, United Dental Hospital, Sydney. N.B. Illustrations for the intraoral lesions are from Kodachrome originals.

References

1. Crandon, J. H., Mikal, S., Landeau, B. R.: *Proc. Nut. Soc.* 12: 2, 1953.
2. Harrison, G. A.: *Chemical Methods in Clinical Medicine*, ed. 3, London, 1947, J. & A. Churchill, Ltd.
3. Krebs, H. A.: *Proc. Nut. Soc.* 12: 237, 1953.
4. Orban, B., Martin, W. B., and Hehn, R. M.: *J. Periodont.* 18: 95, 1947.
5. Sherman, H. C., and Lanford, Caroline S.: *Essentials of Nutrition*, ed. 2, New York, 1943, The Macmillan Company, p. 181.
6. Stones, H. H.: *Oral and Dental Diseases*, ed. 1, Edinburgh, 1948, E. & S. Livingstone, p. 49.
7. Thoma, K. H.: *Oral Pathology*, ed. 2, St. Louis, 1944, The C. V. Mosby Company, p. 1206.