MEDICO-DENTAL LESIONS OF THE ORAL CAVITY

PART II.

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I intend to limit my description of oral lesions to those accompanying dyscrasias of the haemopoietic system. Fortunately they are not of common occurrence but of very serious prognosis. Their early recognition is, therefore, essential, firstly in order to direct the patient for the proper medical treatment, secondly because any surgical intervention of the mouth may result in fatality.

It may sound too presumptuous to imply that a dental practitioner may diagnose blood disorders by the condition of the oral mucosa; on the other hand I am quite convinced that the Medical Profession do not sufficiently avail themselves of the abundant information that the oral tissues provide in aid to the diagnosis of systemic disease, blood dyscrasias and nutritional deficiencies.

The oral cavity is in intimate relation with the external environment, and its tissues are subjected to mechanical, chemical and bacterial insults which make them unusually vulnerable. Thus, unlike other protected mucosae, that of the mouth may be the first to show the early signs of the subclinical stage of a systemic derangement. Hence the absolute importance to include the thorough examination of the mouth in the diagnosis of systemic diseases.

I will very concisely try to describe the derangements of the corpuscular elements of the blood and the oral lesions accompanying them. Pathological alterations of the red blood corpuscles affect the oral tissues comparatively slightly, but the changes accompanying the macrocytic type of anaemias are worth mentioning.

Pernicious Anaemia:

Pernicious Anaemia is a progressive disease with insidious onset characterised by marked alteration in the red blood corpuscles, namely increase in their size (macrocytosis) and hence called macrocytic, irregularity in their shape (poikylocytosis) and the appearance of nucleated cells in the blood stream (normoblasts, erythroblasts and megaloblasts). The number of reds may fall to 1,000,000 per c. mm. but the colour index is greater than 1 — hence also called hyperchromic. Achlorhydria is present and degeneration of the cord accompanies later stages of the disease.

In 1909, Hunter described the atrophic changes of the tongue in pernicious anaemia and called attention to the relationship between oral sepsis and pernicious anaemia. He believed that the resulting anaemia was caused by the ingestion of purulent material which gave rise to a haemolytic substance. The same trend of thought was long sustained that the septic material swallowed from pyorrhoeic mouths damaged the gastric mucosa and thus the production of Castle's intrinsic factor was impaired. Recent researches about the intrinsic factors are very encouraging.

Hunter's glossitis is a marked feature of this disease and it is due to atrophy of the papillae filiformes and fungiformes giving the tongue a red, smooth, waxy appearance, while desquamation of the epithelial layers makes the condition exquisitely sore.

The oral mucosa presents the greenish yellow colour noted on the skin. This is best seen at the junction of the hard and solf palate. The mucosa of the cheek occasionally shows patches of brownish pigmentation on a pale background.

Since the discovery of the effectiveness of liver therapy by Minot and Murphy 22 years ago, there has been a gradual ad-

vance in the methods of purifying liver extracts. A few years ago folic acid appeared to be the haemopoietic factor but it did not influence favourably the cord lesions of subacute combined degeneration. The isolation of Vit. B 12 in 1948 has provided us with an anti-anaemia factor of great potency which stimulates haemopoiesis and prevents cord lesions.

Because of its craniofacial and dental changes I will now make a short mention of those abnormalities accompanying **Primary Erythoblastic Anaemia — Cooley's Anaemia.** It is usually manifest in the early years of life. The child develops a mongoloid appearance; the frontal and parietal bones are very prominent, the maxillar and malar bones are overdeveloped while the bridge of the nose is depressed. The Skiagram of the skull shows peculiar changes in the structure of the cranial bones. Well calcified spicules appear to be joining the inner and outer tables of the skull and they show in the X-Ray picture as "calcified hairs standing on end".

Diseases of the White Blood Corpuscles.

The Leukaemias.

Leukaemia is a disease in which the leucoblastic tissue proliferates causing a marked increase of White Blood Corpuscles in the circulating blood and in the tissues. According to the types of cells which show an increase we have the Myelogenous, Lymphatic or Monocytic Leukaemia.

The symptoms of acute leukaemia usually simulate an acute infection with marked general malaise, fever, pains, headache. This is especially so in the myelogenous and monocytic types. The first oral symptom is hypertrophy of the gums. In the monocytic variety the mouth lesion may take a very acute form, the gums becoming intensely swollen and in a matter of days they may entirely cover the crowns of the teeth. The hypertrophied tissues often lead to areas of necrosis.

In these patients the gums become intensely swollen and may entirely cover the crowns of the teeth, the whole process only taking a matter of days. The hypertrophied tissues often lead to areas of necrosis and ulceration especially in the upper molar region and the incisor region. The tongue is not usually involved.

The pathological changes in the gum tissues account for the lesions described. The microscopical findings in a section of the leukaemic gum are oedema of these tissues with marked extravascular accumulation of abnormal leucocytes. Multiple thrombi are formed in the smaller blood vessels causing extensive tissue necrosis.

In leukaemia, radiotherapy may delay the fatal end. Recent advances in biochemistry and biophysics have provided us with certain chemotherapeutic agents which are only pointers for further lines of research.

Urethane has been clinically tried in leukaemias. It has no effect on the acute types, but it has been found effective in chronic cases of both myeloid and lymphatic types (at a dose of 2-4 gms daily given orally and a maintainance dose of 1.5 gms daily).

Sir Stanford Cade mentions the case of a woman who, during a period of remission under treatment for chronic lymphatic leukaemia, became pregnant and was delivered of a healthy child by Caesarean section and was still alive after 2 years at the time of his writing.

Aminopterin is another substance on trial for acute leukaemia with a similar, but more dangerously pronounced, cytotoxic action and it causes severe ulcerative lesions of the mouth.

Having described the mouth lesions accompanying the malignant proliferation of the white corpuscles let us now consider shortly the mouth lesions occuring when the white granular cells suffer a numerical reduction.

Agranulocytosis.

The syndrome known as Agranulocytosis was first described by Schultz in 1922. The initial clinical lesion is commonly found in the mouth. The extraction of teeth is contraindicated and may cause fatal results. Valuable time in treatment may be lost if the lesion is mistaken for a fusospirochaetal infection.

The pathological condition is brought about by toxic material damaging the bone marrow. The injury to the leucoblastic tissue causes an arrest in maturation of the myeloid series which do not develop further than the megaloblastic level and do not migrate into the blood stream.The haematologic findings, therefore, include a decrease in the number of granulocytes and at times their disappearance.

The damaging substance may be bacterial in origin or drug administration. The most inculpated drug is certainly amidopyrine, which enters into the combination of a legion of proprietary products. Cumroe (1936) attributes the syndrome to an allergy to the drug. Gold salts and arsenic preparations may cause this drop in the whites. Finally, one should not forget that Agranulocytosis may result from the prolonged administration of the sulphonamides.

The oral lesion is accompanied by a general feeling of malaise, fever etc., out of proportion to the extent of the mouth lesion. The typical oral lesion consists in irregular-shaped necrotic areas. The ulcers have a dirty gray appearance with little inflammatory reaction around the margins. The lack of reaction around the lesion has been attributed to the absence of defensive granulocytes. The foul odour is characteristic of decomposing tissue. Ptyalism is common.

The oral lesions are the result of the haematologic changes. The blood does not afford adequate cellular defence (phagocytic etc.) to the moist tissues of the mouth and the ulcerations are caused by the ever present bacteria of the oral cavity. Thus, with the suspension of the offending drug, penicillin treatment would control the secondary infection and allow the spontaneous regeneration of the leucocytes.

The differential diagnosis should include:

(a) acute follicular tonsillitis with ulcerations.

(b) fusospirochaetal lesions including angina.

(c) noma.

(d) infectious mononucleosis.

(e) diphtheria.

The Purpuras.

The purpuras fall into two main groups, (a) those in which the blood platelets are numerically reduced i.e. the Thrombocytopoenic Purpuras (b) and those where the platelets show no deficiency, but the capillary walls are damaged i.e. the Vascular Purpuras.

The oral manifestations consist of capillary oozing from the entire marginal gingivae which produces the foetid odour of decomposing blood; small reddish spots appear beneath the oral mucosa, often more evident at the junction of the soft with the hard palate. They do not blanch on pressure. Ecchymotic areas may be present and blood-filled blebs may form. The diagnosis is made by low platelet values, prolongation of bleeding time and failure of blood clot to retract. Rumpel-Leeds tourniquet test is positive.

In both types of purpura, there is a great danger of haemorrhage after extraction of teeth or mouth operations.

Gentlemen,

It has been a great pleasure to address such an elect and mixed audience lending a listening ear to the description of Dentistry on the border. I have endeavoured to show that Dentistry can offer great contributions to Medical Science. It is time that we evaluated how Dentistry and Medicine could better assist each other

and together contribute towards the welfare of our patients. The dental practitioner is better trained to assess the condition of the oral mucosa and early to detect any abnormal alterations. The physician, therefore, who seeks the co-operation of the dental practitioner is simply gaining and very wisely. On the other hand, it is incumbent on students of the Dental Science to build their knowledge on the biological understanding of anatomy, physiology and pathology in order that Dentistry will secure its honourable place among its elder sister specialities of Medicine and Surgery.