MOUTH INFECTION AND TREPONEMA VINCENTI

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It is well known that Vincent's spirochaete (and often the associated fusiform bacillus) is commonly found in smears from unhealthy gums and other mouth lesions, and sometimes from healthy mouths. The aetiological significance of the treponema in these conditions and of its commensal the fusiform bacillus is generally regarded as an unsettled question. Whatever its role may be, Vincent's spirochaete is commonly found in an inflammatory condition of the gums which may in severe cases go on to a gangrenous condition, laying bare both rows of teeth to the roots and leading to a toxic condition and even to a fatal issue.

MacCallum[1] mentions that, besides the mouth, Vincent's infection is found in the pharynx, bronchus and lungs. Park and Williams[2] state that it is found in gangrene and noma, and in the gum margins of dirty teeth. Bailey and Love[3] add acute superficial glossitis. Shera has recorded its presence in the rectum or sigmoid, where it causes a relapsing diarrhoea. The Extra Pharmacopoeia[4] refers to its presence in the eyes, nose and penis. Savill[5] states that these organisms may be found in ulcerative stomatitis, in acute septic scarlet fever, and in a form of acute tonsillitis. From my own observations I can add otitis to the list.

TWO CONSECUTIVE SERIES

To investigate the position in the African population of the Matatiele district of the Transkei, I examined the mouths of 224 consecutive patients. Of these, 141 (63%) showed clinical signs of gum infection. Smears were taken of each of these

and submitted to the Senior Government Pathologist, Durban, who reported that every one of the 141 smears (100%) showed the presence of Vincent's spirochaetes. These 141 patients were not necessarily complaining of mouth trouble. Indeed, only one in the series presented himself with a mouth complaint. Some were parents accompanying sick babies and others were school children and mine workers coming for routine examination.

In a second investigation 141 consecutive African patients were examined.* Of these, 10 were babies and 3 were toothless adults; smears were taken from the other 128 (89.4%), of which 126 (98.4%) were reported by the Government Pathologist as showing Vincent's spirochaetes. Of the 128 patients from whom smears were taken, 100 (78.2%) showed clinical signs of a gum infection (99 of these were positive for Vincent's) and 28 were from patients with clinically healthy mouths (27 of these were positive for Vincent's). The results of this investigation (other than those concerning the state of the teeth) are shown in Table I, which also includes the results of a similar investigation on Coloured patients (48 smears) and on European patients. These investigations show how widespread the clinical condition and the Vincent's infection are in the Matatiele district. Investigations in miners, industrial workers and school children in other places indicate that they may be common in Africans elsewhere in the Union. Coloured and European communities are also affected.

TABLE I. SERIES OF 128 CASES

	Native		*Coloured*		*European*	
	Ratio	%	*Ratio*	%	*Ratio*	%
Infection present (clinical or laboratory)	127/128	99	48/50	96	*	
Vincent's positive	126/128	98	41/50	82	13/22	59
Vincent's negative	2/128	1·5	9/50	18	9/22	40·9
Healthy gums (no clinical or laboratory infection)	1/128	·75	2/50	4	*	
Clinically healthy mouth, Vincent's positive	27/128	21·8	6/50	12	*	
Clinical infection present	100/128	79	25/50	50	*	
Chronic or pyorrhoeal infection (clinical)	14/128	10·9	15/50	30	*	
Chronic or pyorrhoeal infection (clinical), Vincent's negative	0/128	0	3/50	6	*	
Chronic or pyorrhoeal infection (clinical), Vincent's positive	14/128	10·9	12/50	24	*	
Infected gums (clinical), Vincent's negative	1/128	·75	9/50	18	*	
Sub-acute on Chronic forms	*		2/50	4	*	
Diagnostic error (i.e. negative slides returned later as positive	†		2/50	4	3/22	14

The series of Natives and Coloured were consecutive, of Europeans as nearly consecutive as possible.
* Not noted. † Not noted (very small).

It would appear that the infection with *Treponema vincenti* mainly occurs in the mucous membranes, and that in severe cases it extends more deeply, sometimes resulting in gangrene.

* These were also investigated for dental conditions.

The alternative hypotheses are that this organism is the primary invader and the cause of the diseased condition; or that it is associated with some other primary invader (e.g., a virus), either as a virulent ally or as a harmless commensal. It is found in persons who appear to be in normal health and in what appear to be normal gums, and another theory is that it lies dormant (or as a saprophyte) to become active under certain conditions, such as malnutrition and ill-health. Possibly the gums become sensitive to its presence and react in a similar manner to an impetiginous eczema about a discharging ear. Whatever the role of this organism, it is important to recognize the gravity of the gum condition (and other diseases) with which it is associated, both in view of their extremely wide distribution and their severity in many cases.

SYMPTOMS AND SIGNS

The following are prominent symptoms and signs in this widespread condition of gingivitis. Sucking of the teeth or pressure on the gums (e.g. with a swab) will draw blood. This is sometimes observed in the absence of apparent inflammation. Spitting of blood after the use of a toothbrush is suggestive.

Taste. There is usually a foul taste of stale blood or pus. The taste of fresh blood also occurs, as well as a salty taste from the blood. The smell of the mouth depends on the severity of the disease, the habits of oral hygiene, the state of health, fever, etc. Africans do not generally complain of foul breath or bad tastes.

Pain. There is a dull neuralgic ache as a rule, which varies

in intensity. It is not often complained of. It is due to infected gums, carious teeth, infected jaw-bone, and loose teeth. Other forms of pain and throbbing also occur—from the pressure of chewing, contact with cold air or water, the eating of sweets, etc.; and from abscess formation. As the gums recede the exposed teeth are painful like the quick of a nail.

Oedematous gums, overlapping the teeth somewhat and producing pockets discharging mucoid pus, and flaps. This is more marked in the lower jaw. The gum is hyperaemic and red, according to the severity of the inflammation, but in some cases does not look very different from normal gum. The affected gum recedes to a certain extent with treatment, but never altogether recovers from its inflammatory state.

Receding gums are seen in older persons in whom the disease has persisted for many years. The oedematous soft tissue of the earlier years is replaced by fibrous tissue, which retracts and does not bleed so easily. This shrinking causes the gums to recede and the teeth become 'long' with 'windows' between.

VARIOUS FORMS OF THE DISEASE

No one appearance can be described as typical. Gums which appear to be healthy give positive smears as well as obviously inflamed gums. Where inflammatory process is present, the outer gum-margin is always more affected than the inner. Various gum states can be distinguished as follows:

1. *Apparently healthy gums*, clinically indistinguishable from the normal, but giving smears showing the Vincent organisms, which may be regarded as latent.

2. *Mild form.* A thin pink line is seen along the gum margin, or the gum may look normal, though it bleeds on pressure or on sucking. In a slightly more advanced stage this line becomes thicker and redder, sometimes presenting a striated appearance. Pain is slight or negligible, or there may be mild neuralgia. There is often satisfactory general health in spite of prolonged infection.

3. *Sub-acute oedematous form.* The inflammatory process is deeper and the gums redder and thicker (i.e., more oedematous), with flaps overlapping the teeth. The base of the gum becomes undermined and separated from the tooth, forming pockets, while the edge of the gums shows overgrowth of granulations (proud flesh). The infection does not extend to the rest of the mouth, though sometimes (not commonly) a tonsillar ulcer may occur, with sore throat. Africans usually do not complain of the pain unless it is very severe, and then they seek dental extraction rather than medical attention. Often they are only seen for some unrelated condition, e.g. dysmenorrhoea. If the infection becomes deeper a low-grade osteomyelitis of the jaw results in continuous pain, with some malaise and fever. Even in this state the patient may seek dental extraction rather than medical treatment. Sometimes the inflammation of the gum is more acute locally, about a stump or a carious tooth or tartar. The sub-acute inflammatory form is commoner than the acute toxic form (no. 6) and is usually found in young adults.

4. *Chronic*, or *pyorrhoeal*, or *fibro-purulent* form. This is a later stage, common in the elderly. The gums become more fibrous and less oedematous (not bleeding so readily) and recede, laying bare the teeth. The tooth bed is undermined and the pockets thus formed exude frank pus in place of the previous sticky mucoid discharge. The teeth eventually become loose. Mild pain is generally present. As a rule the patients enjoy fair health.

5. *Sub-acute on chronic* form. Less commonly acute inflammation supervenes on the foregoing (4). The gums become hyperaemic and bleed readily and the gum pockets literally drip mucoid pus, which runs down the side of the teeth.

6. *Acute toxic, inflammatory* and *gangrenous* form. This rarer form is observed in children only. The gums, tongue and mouth become inflamed and painful, bleeding readily, with oral ulcers associated. The gums sometimes become oedematous to the point of overlapping the teeth. Later a necrosis commences and a grey slough appears and, as the flesh decays, windows appear between the roots of the now

TABLE II. AGE INCIDENCE

Age (years)	*No.*	+	−	*H and* −	*I and* −	*H and* +	*I and* +	*H*	*I*
1-2* ..	14	2 (14%)	12	8	4	1	6	9	5 (36%)
3.. ..	12	11 (92%)	1	1	0	6	11	7	5 (42%)
4.. ..	12	9 (75%)	3	1	1	5	11	6	6 (50%)
5.. ..	12	11 (92%)	1	0	1	5	12	5	7 (57%)
6.. ..	12	12 (100%)	0	0	0	5	12	5	7 (57%)
7.. ..	12	11 (92%)	1	0	1	1	12	1	11 (92%)
8.. ..	12	8 (67%)	4	0	4	3	12	3	9 (75%)
9.. ..	10	9 (90%)	1	0	1	1	10	1	9 (75%)
10-12 ..	12	7 (57%)	5	2	3	2	10	4	8 (67%)
13-15 ..	12	10 (83%)	2	1	1	3	11	4	8 (67%)
16-19 ..	14	13 (93%)	1	0	1	5	14	5	7 (50%)
20-30 ..	12	9 (75%)	3	1	2	0	11	1	11 (92%)
31-40 ..	12	12 (100%)	0	0	0	1	12	1	11 (92%)
41-50 ..	12	10 (83%)	2	0	2	1	12	1	11 (92%)
60-75 ..	12	10 (83%)	2	0	2	1	12	1	11 (92%)
Total ..	182	144 (79%)	38	14	23	46	168	54	126 (69%)

No.= Number of patients.
\+ = Smear positive for Vincent's spirochaete.
− = Smear negative for Vincent's spirochaete.

H = Gums clinically healthy.
I = Gums clinically infected.
* 13-24 months.

loosened teeth. Fever, pain and loose teeth prevent eating. The child's condition, resembling that in cancrum oris, is highly toxic, with tachycardia, fever, sunken eyes, dehydration and exhaustion. Death may occur, but on treatment the child as a rule recovers rapidly. The teeth remain loose and fall out when the gums recede, and the gums remain in a chronic inflammatory state. The buds of the second dentition are not destroyed by the gangrene.

Age Incidence. Experience at Matatiele is at variance with Savill's statement[6] that 'gingivitis due to Vincent's spirochaete is common before the middle-age' and that of Bailey and Love[3] that it is essentially a disease of young adult life and it is exceptional to find it after the age of 35. In our patients the clinical condition is common at all ages and its percentage incidence tends to increase with age (Table II). Nevertheless the inflammatory process tends to be more active in younger people. If no teeth are present the organism as a rule is not found in the gums.

Tonsillar Infection. After gums the commonest site for Vincent's infection is the tonsils. In Table III are shown the results of the examination of smears taken from the tonsils of

TABLE III. EXAMINATION OF TONSILS FOR VINCENT'S SPIROCHAETE

	Total	*Vincent positive*	*Vincent negative*
Consecutive infants (age 3-10 months) without teeth	12	0	12
Consecutive infants (age 12-24 months) with teeth	12	1	12
Consecutive 'normal' tonsils	11	3	8
Consecutive acute tonsilitis (follicular membrane etc.)	17	1	16
Consecutive enlarged tonsils	11	4	7
Consecutive symptomatic sore-throats (N.A.D. on examination)	5	0	5
Consecutive cases without teeth and with tonsils present	10	1	9
Consecutive cases with teeth present and with tonsils absent (pharyngeal swab)	7	1	6
Case with no teeth and no tonsils (pharyngeal swab)	1	1	0
Total	86	12	74

86 patients in consecutive groups. In 12 cases Vincent's infection was reported (14%).

Wassermann Reaction. I have not found the Wassermann test to be an indication of infection with *Treponema vincenti.* For example in 11 related patients of good moral standards, whose Wassermann results were negative, the gum smears were all positive for Vincent's spirochaete. Similarly, 38 patients were tested for Wassermann. Of these 10 had clinical syphilis with positive Wassermann; 6 gave gum smears which were Vincent-positive and 4 Vincent-negative; in 2 the clinical diagnosis of syphilis was doubtful and the Wassermann reaction was negative; both these showed positive Vincent's infection. The remaining 26 were non-syphilitics with negative Wassermanns; 25 of them showed positive Vincent's infection and one (with inflamed gums, however), negative.

TREATMENT

Both from the preventive and curative point of view it is necessary to attend to the general health and to oral hygiene in the treatment of this widespread form of gingivitis.

In the African, nutrition is a factor of first importance, and the correction of malnutrition must take a high place in the combating of the disease. The question of diet is also involved in oral hygiene.

Loose and decayed teeth which are beyond repair should be extracted. A warning, however, should be sounded against the unnecessary extraction of teeth which are sound or can be repaired; so far as these are concerned treatment should be limited to the gums, and attention to the general health. Tartar deposits should be removed and also 'grit' and stains on the teeth. The tooth-brush should be soft. Hard tooth-brushes, especially when the gums are soft and friable, may do more harm than good. In some cases it is probably better merely to rinse the mouth. Tooth-pastes that are chemically irritant or unduly abrasive should be avoided.

Antibiotics. Aureomycin, given systemically, I find to be the most successful drug to subdue oral infection. The local application of aureomycin is less successful. Penicillin may also be used systemically and locally. A one-shot injection of 600,000 units is of great help in subduing acute oral inflammation, but in the toxic and gangrenous forms it is wiser to continue the treatment.

With antibiotic treatment the Vincent's infection, after disappearing, reappears in a few days. With aureomycin the disappearance tends to last somewhat longer. The gums never entirely heal under this treatment, but inflamed gums are not found to flare up again.

Sulphonamides. Sulphatriad has been found to improve the gum state, particularly in the more acute forms. Given with penicillin it produces a good response. The organism, however, returns and the gum never quite heals.

Mouth Lotions. Potassium chlorate may be used as a mouth wash or as a lozenge. It has a still more beneficial effect if taken orally, particularly in the more acute forms, especially when given with Sulphatriad, though the toxic or gangrenous forms respond quicker and more dramatically to penicillin. Hydrogen peroxide and mercurochrome are useful too. With these lotions, also, the gum never quite heals. Antibiotic lozenges have their use, but with long administration they make the gums and tongue sore.

Vitamins. Vitamin B_{12} and B complex, nicotinic acid and vitamin C have all been used, orally and by injection, with varying results. The improvement is usually limited.

Conclusion. The disease should only receive antibiotic treatment if the gum condition has got out of control and the infection has become acute. Antibiotics have taken the place of arsenicals, local and systemic, which the earlier text-books recommended.

SUMMARY

An account is given of an investigation into the occurrence of gingivitis and Vincent's infection in two series of cases

(chiefly Africans) at Matatiele, Transkei. Both the glossitis and Vincent's infection are shown to be extremely widespread.

A clinical account is given of the various forms of this condition.

The aetiology and treatment are discussed.

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