

HIV: THE IMPACT ON DENTISTRY*

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AIDS (Acquired Immune Deficiency Syndrome (AIDS)) was first observed in 1981 in male homosexuals, though there is now evidence that sporadic cases existed as long ago as 1959 in Africa and the United Kingdom. Human immunodeficiency viruses (HIV) are almost certainly the causal agent of AIDS.

HIV damages the immune system, especially certain T lymphocytes (CD4 cells), and the nervous system, and thus presents particularly with opportunistic infections and neurological upset. *Pneumocystis carinii* pneumonia and oral candidosis are especially common. Neoplasms, especially Kaposi's sarcoma are also seen.

HIV-1 was first reported in the United States of America and Africa and has now spread to all continents and a further virus HIV-2, which originated in West Africa – has also now spread to Central Africa, Europe, United States of America and South America.

The World Health Organisation (WHO) has estimated that during the 1990's at least 40 million persons will have become HIV-infected so that, by the year 2000, 10 million adults and 5 million children will have developed AIDS.¹ In parts of Africa 1 in 5 adults have already been infected with HIV and in the Ivory Coast and some cities in North America for example, AIDS is now the leading cause of death of certain groups of adults.

It is currently estimated that in the United States of America some 60–70,000 persons will now be diagnosed per year as having AIDS² and that HIV infection will continue to spread into rural

communities³. By May 1990 one million persons were estimated to have been infected with HIV-1 in the USA, nearly 135,000 persons had had AIDS, and over 50% of these had died.

The number of persons in the United Kingdom reportedly infected with HIV in 1993 is about 30,000–40,000 but this is certainly a gross underestimate.⁴

Worldwide, 75% of all infection has been acquired through sexual intercourse, mostly transmitted heterosexually, and there is currently a male : female ratio of 3 : 2¹. Only about 10% of HIV infections have been linked to injecting drug use (IDU), 10% were transmitted perinatally and the remaining 5% were transmitted through blood, principally by transfusion. HIV is thus no longer solely a problem of male homosexuals, and is a rising problem among heterosexuals particularly those who have unprotected sexual intercourse.⁵

Persons infected with HIV, especially those who abuse drugs intravenously or who are sexually promiscuous may also be coinfecting with other agents including hepatitis viruses B and D (rarely C), human T lymphotropic viruses I and II (HTLV-1 AND HTLV-2), hepatitis viruses and other sexually transmitted diseases.

1. ASPECTS OF TRANSMISSION OF HIV RELEVANT TO DENTISTRY

There has been considerable concern about a possible occupational hazard from HIV⁶⁻⁹. However, HIV is not transmitted by social contact, contact at work, or even by living in the same household, provided there is no sexual contact or other high risk activity.

Whole saliva from some patients with HIV disease may contain viruses including HIV¹⁰⁻¹⁶. Other viruses and HIV-like particles may be present in salivary glands of patients with AIDS¹⁷⁻¹⁸. However, epidemiological and laboratory animal data indicates that salivary transmission of HIV is unlikely although transmission of HIV via orogenital routes has been suggested¹⁹⁻²². HIV antigens and antibodies²³ have also been demonstrated in gingival crevicular fluid. Aerosol transmission of HIV has never been documented.^{24, 25}

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HIV transmission during dental surgery

The first health care worker (HCW) reported to have been responsible for the transmission of HIV to patients in clinical practice was a homosexual dentist practicing in Florida, United States of America who has now died from AIDS²⁶. The precise mode of transmission of HIV cannot be certain²⁷ but 6 patients were thought to have been infected and all had invasive procedures²⁸. A sharp injury is a possible route of transmission as the dentist had, like many HCW²⁹⁻³¹, sustained needlestick injuries.

That HIV transmission appears to have occurred during dental treatment is clearly alarming, but it must be tempered by the knowledge that worldwide, in the decade or so of the recognised HIV pandemic, only these 6 patients in a single dental practice are thought to have become HIV-infected. Even this incident has been questioned³² and a look-back study on nearly 80% of locatable patients treated by another United States HIV-infected dentist has failed to show transmission³³. Indeed, thus far, none of over 19,000 patients treated by HIV-infected HCW (including 16 dentists/dental students) have been found to have acquired HIV through dental or other operative treatment^{31, 34-39}.

Transmission of HIV to HCW

If dentistry were to be a significant source of HIV transmission between patients and dental staff, it is likely that several dental staff would have acquired HIV by now: in fact, only one out of over 7,000 tested dental health care staff including those from areas of high HIV prevalence, may have acquired HIV during dental practice⁴⁰⁻⁴². Furthermore dentists have the same risk of occupational acquisition of HIV as do other health care workers. As with all surgical procedures, there can be areas of weakness in infection control in dental practice. Other infections have indeed been transmitted but current data indicates that the risk of HIV transmission during dental treatment is very small indeed.

Some cases of HIV infection in HCW (including the New York dentist) may well represent occupational transmission^{40, 43-51}. However, there is great difficulty in such instances – including that of the dentist – in proving that the HIV transmission was occupational, although most infected HCW, including the dentist, had sustained needlestick or sharp injuries with potentially HIV-infected material.

Virtually all HIV-affected patients have recognised risk factors for HIV infection that are non-occupational, and the HIV positive group with no identifiable risk activity does not contain an unduly high proportion of clinicians⁵²⁻⁵⁴. Cross-sectional studies on HCW have revealed little difference in HIV-seropositivity rates compared with those in non-clinicians⁵⁵⁻⁵⁷ though, of course, where

such studies are carried out on a volunteer basis, there may be some bias. Nevertheless, of some 7,000 dental clinical personnel from several countries serologically examined for HIV antibodies, only 1 (the New York dentist previously mentioned) has tested positive^{40, 41, 58-65}. Many of those tested and shown to be HIV-negative had treated HIV-infected persons for several years.

From many parts of the world reported prospective studies on HCW occupationally-exposed to HIV-infected persons (ie HCW with documented sharp injuries or mucous membrane exposures involving HIV-infected blood/body fluids) have shown a risk to HCW of being infected occupationally with HIV at less than 0.5% per such exposure to HIV-infected blood^{30, 48, 66-77}. Even mouth to mouth resuscitation of a patient with AIDS bleeding from the mouth did not appear to transmit HIV to any of the 4 nurses involved.⁶⁸

2. ORAL MANIFESTATIONS OF HIV DISEASE

An overall picture of oral disease in the whole spectrum of HIV infection and the acquired immune deficiency syndrome (AIDS) has now emerged. There are a few fairly common lesions such as candidosis, hairy leukoplakia, Kaposi's sarcoma and possibly gingival and periodontal disease⁷⁹⁻⁸⁷

Most HIV infected patients have head and neck manifestations at some stage⁸⁸⁻⁹⁰ and oral lesions are often fairly early signs⁹¹⁻⁹⁸. However, oral mucosal lesions are not uncommon very early after acquisition of HIV⁹⁹ and are most common when the CD4 lymphocyte count falls. A wide range of oral and perioral lesions may be seen: this paper discusses only the more important of these.

i. Lymph node enlargement

Cervical lymphadenopathy is almost invariably in all stages of HIV infection^{100, 101}.

ii. Candidosis

(a) Significance

Oral candidosis is the most common oral manifestation of HIV infection¹⁰²⁻¹⁰⁵, may imply the concurrent presence of oesophageal candidosis¹⁰⁶⁻¹⁰⁹ and can be a predictor of liability to other opportunistic infections¹¹⁰⁻¹¹⁵ and later may predict early progression to AIDS¹¹⁶.

(b) Clinical types of oral candidosis

Thrush (pseudomembraneous candidosis) is one of the most obvious oral lesions in HIV infection, but other types of candidosis may also be seen, especially erythematous, hyperplastic, angular stomatitis (cheilitis) and median rhomboid glossitis¹¹⁷⁻¹¹⁹. The erythematous form of candidosis may well be the

most common early oral manifestation of HIV infection¹²⁰ and presents as pink or red macular lesions typically on the palate and dorsum of tongue.

(e) Management of oral candidosis

Diagnosis is clinical, possibly supported by identification of blastospores and pseudohyphae in smears from a lesion, examined by Gram stain, potassium hydroxide, or a fluorobrightener such as calcofluor white. Early treatment is warranted not only because of the discomfort but also because the foci may act as reservoirs for local spread of disease¹⁰⁷. Prophylaxis should also be considered.

Predisposing factors such as smoking and xerostomia should be controlled first. Underlying xerostomia should be treated with, for example, bethanecol⁸¹.

Antifungal therapy is reviewed elsewhere¹²¹. Topical treatment of candidosis with nystatin, amphotericin or clotrimazole is often successful initially with about 14 days but relapses are common. Fluconazole is the mainstay of systemic antifungal therapy.

iii. Viral infections

Oral or peri-oral infections with herpesviruses (especially herpes simplex virus (HSV)) and Epstein-Barr virus (EBV), are fairly common in HIV infection: varicella-zoster virus (VZV) infection is less common. Hairy leukoplakia is the most common viral infection.

Hairy leukoplakia (HL) is an adherent white patch usually seen on the parakeratinised mucosa on the lateral margins of the tongue¹²²⁻¹²⁵ and occasionally elsewhere¹²⁶. Originally described in HIV infection, and recorded in all risk groups¹²⁷, HL was generally regarded as pathognomonic of HIV disease¹²³. HL is seen in at least one quarter of HIV-infected persons¹²⁸. The lesions of HL are often corrugated or have a shaggy or "hairy" appearance¹²⁹, are mostly symptomless and unlike some oral keratoses, have no known pre-malignant potential¹³⁰.

HL is now known not to be absolutely specific for HIV, rather is usually a manifestation of chronic immunosuppression¹²¹. Nevertheless, HL in HIV is a predictor of poor prognosis³²⁻¹³⁴.

Management of hairy leukoplakia

Hairy leukoplakia (HL) is usually symptomless and though it may be the source of concern or some discomfort to the patient, specific treatment is rarely indicated. HL in HIV-infected individuals may occasionally improve spontaneously¹²⁵, or with zidovudine¹³⁵⁻¹³⁷ or DDI¹³⁸.

iv. Gingival and periodontal disease

HIV-related gingivitis (HIV-G) and periodontitis (HIV-P) as well as necrotizing gingivitis in HIV may be seen¹³⁹⁻¹⁴¹. The different periodontal lesions are detailed below:

i. **Non specific gingivitis** is indistinguishable from plaque-related gingivitis affecting non-HIV infected individuals.

ii. **HIV gingivitis** (linear gingivitis, previously termed generalised atypical gingivitis (ATYP)). In this, there is gingivitis with erythema and oedema and it does not respond to improved oral hygiene. The erythema can manifest as an intense linear band at the gingival margin, as petechiae-like red patches on the attached and/or non-attached gingivae, or as a generalised redness affecting the attached and/or unattached gingivae. Spontaneous gingival bleeding is common¹⁴²⁻¹⁴⁴. While some workers believe that no similar condition occurs in non-HIV infected individuals¹⁴⁴, it is known that neutropenic children can have profound gingival erythema which may affect the free and/or attached gingivae.

iii. **Acute necrotizing ulcerative gingivitis (ANUG)** essentially indistinguishable from ANUG in non-HIV infected patients, giving rise to gingival pain, ulceration, pseudomembrane formation, and interdental gingival cratering. Loss of crestal bone may occasionally occur as can sequestration^{142, 144}.

iv. **HIV-associated periodontitis** is characterised by rapid localised or generalised periodontal destruction giving rise to pain, tooth mobility, infrabony pocketing and, rarely, involvement of adjacent structures such as the maxillary antrum^{142, 145}.

v. **Necrotising stomatitis (necrotising periodontitis)** characterised by extensive soft tissue and bony necrosis with sequestration¹⁴⁶.

v. Malignant neoplasms

The most common malignant tumours in HIV disease are Kaposi's sarcoma (KS: sometimes termed epidemic KS or EKS), which accounts for 83% of neoplasms, and non-Hodgkin's lymphomas which account for 13%¹⁴⁷. KS currently affects up to 40% of those with AIDS; lymphomas affect up to 10% or more¹⁴⁸.

(a) Kaposi's sarcoma

KS is discussed fully elsewhere¹⁴⁹. Epidemic KS is more aggressive than the classical endemic form, or that related to iatrogenic immunosuppression¹⁵⁰. KS in western countries is now found mainly in HIV-infected homosexual or bisexual men¹⁵¹ or those who have had sexual relations with these^{152, 153} but is also occasionally seen in such sexually active groups who are not infected with HIV^{154, 161}. This suggests a

sexually transmitted causal agent.

The prevalence of KS in AIDS has been decreasing during the recent past¹⁶², and it is now perhaps a less common oral manifestation than, for example, oral candidosis and hairy leukoplakia. KS is oral or perioral in 50% or more of patients with mucocutaneous KS and is often an early manifestation of severe HIV disease¹⁶³.

Early lesions typically present in the palate, usually as a pigmented macule. Sometimes the tongue, gingiva, or other sites are affected. Later, KS may present, as red, bluish, or purple patches or nodules, that are sometimes ulcerated¹⁶³. Some oral KS is nondiscoloured¹⁶⁴⁻¹⁶⁶.

Management of KS

Biopsy may well be indicated. Treatment is discussed further elsewhere¹⁶⁷. Neither local or systemic treatment of KS have been shown to alter the ultimate course of the disease but they may cause a disappearance or reduction in size of lesions and thereby alleviate discomfort¹⁶⁸. Because the natural course of KS disease progression is highly variable, evaluating the long term efficacy of systemic treatment has been difficult: no data show that treatment improves survival. Systemic management of HIV with zidovudine (azidothymidine) may cause oral KS to regress¹⁶⁰.

(b) Lymphomas

Lymphomas are fairly common in HIV disease – particularly in intravenous drug abusers^{170, 171}. High-grade non-Hodgkin's lymphomas are the most common lymphomas in HIV-infected persons. The incidence of Hodgkin's disease overall appears not to be significantly increased¹⁷²⁻¹⁷⁴ but there is an increase of the mixed cellularity type of Hodgkin's disease¹⁷⁵.

The onset of lymphomas is preceded, in one-third of cases, by persistent generalised lymphadenopathy. Enlargement of pre-existent palpable lymph nodes or a rapidly enlarging mass in the head and neck in HIV disease is always an indication for a biopsy to exclude malignant lymphoma. At initial presentation, the lymphoma is typically widely disseminated, with extranodal sites of disease in 65% to 98% of patients.

Oral lymphomas are now a recognised, but uncommon, complication of HIV and have been recorded thus far mainly in homosexual/bisexuals¹⁷⁶⁻¹⁷⁸. Typically presenting in the fauces, gingiva, or elsewhere, as a rapidly growing mass, an ulcer, or tooth mobility, they are being increasingly recognised and reported¹⁷⁹⁻¹⁸⁴. Burkitt's lymphoma may also present orally¹⁸⁵, there is an increase in salivary gland lymphomas in HIV disease¹⁸⁶⁻¹⁸⁸ and occasionally cases of oral Hodgkin's disease are

being reported¹⁸⁹.

Management of lymphomas

Management of oral lymphomas is typically by radiotherapy¹⁹⁰ unless there is widespread systemic lymphoma, when chemotherapeutic intervention is warranted.¹⁹¹

Other oral lesions

A wide range of other oral disorders can arise in HIV disease, particularly in AIDS. (Table 1) Unlike the above mentioned lesions, most are uncommon and simply reflect the degree and type of immunodeficiency associated with late HIV disease.

Xerostomia and Sicca type syndrome
HIV-embryopathy
Submandibular lymphadenopathy
Hyperpigmentation
Granulosum annulare
Exfoliative cheilitis
Lichenoid reactions

Table 1 - Classification of oral lesions associated with HIV infection

Association of lesions with HIV infection	Lesions
Strongly associated	Candidosis; hairy leukoplakia; HIV-gingivitis and periodontitis; Kaposi's sarcoma; non-Hodgkin's lymphoma
Less commonly associated	Atypical ulceration; salivary gland disease; thrombocytopenic purpura; viral infections (other than Epstein-Barr virus)
Possibly associated	Bacterial infections (excluding gingivitis or periodontitis); fungal infections other than candidosis; melanotic hyperpigmentation; neurologic disturbances; osteomyelitis; sinusitis

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