Case 1 Discussion: Mr. Victor Harrison

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CASE SUMMARY

Mr. Victor Harrison is a 38 year old male who is employed in finance and recently moved to a new area, putting him under high amounts of stress. He is human immunodeficiency virus (HIV) positive although his HIV viral load is undetectable with a CD4 count of 350 cells/μL. Although he has been on antiretroviral therapy (ART) for two years, he has recently stopped taking his medication without consulting with his doctor. This shows that he may have lower health literacy due to his lack of understanding of the importance of ART. In addition, men such as Mr. Harrison are less likely than women to take preventive measures regarding their health. As a result, Mr. Harrison may feel embarrassed or uncomfortable to disclose his current health status as well as previous sexual habits and medication. In addition, due to stereotypes regarding age, older sexually active individuals are less likely to disclose their sexual habits and drug use with their doctors, and doctors are also less likely to inquire older individuals about these concerns. 1

HIV is a viral disease that attacks and destroys the immune system's CD4+ T cells, which are responsible for stimulating other immune cells including B lymphocytes, CD8 lymphocytes, and macrophage induction.2 As a result, cellular activity to sites of infection are increased by recruitment of neutrophils, eosinophils, and basophils.3 Treatment of HIV is not currently available, but the disease can be managed by ART. ART slows the progression of HIV, and reduces the viral load within blood and bodily fluids.4 Unfortunately, if HIV is left untreated by lack of ART, progression into more severe stages of HIV may occur. Currently, Mr. Harrison is in the second stage of HIV, otherwise known as clinical latency or domancy, the stage prior to obtaining acquired immunodeficiency syndrome (AIDS).5 Mr. Harrison's CD4+ count of 350 cells/μL is within the stage 2 range of 200-499 cells/μL according to the Centres for Disease Control and Prevention.6 The spectrum of oral manifestations is very vast in patients with HIV or AIDS and can be categorized into the following: infections (bacterial, fungal, viral), neoplasms (Kaposi's Sarcoma), immune mediated (acute necrotizing stomatitis, aphthous ulcers), others (nutritional deficiency and xerostomia), and oral manifestations as adverse effects of antiretroviral therapy.7

DESCRIPTION OF CLINICAL LESIONS

	FIGURE 1	FIGURE 2	FIGURE 3
MARGINS	isolated	discrete	discrete on superior border, diffuse on inferior border
COLOUR	slightly more erythematous than surrounding tissues, vascular similar to alveolar mucosa	yellow/white, leukoplakia	white in comparison to adjacent tissues (leukoplakia)
APPEARANCE	homogenous	homogenous in colour, non-homogenous in texture	non-homogenous, speckles of white lesion found on top of a background of a white lesion
TEXTURE	concavity, seems ulcerated, fairly smooth	raised papillae present, nodules present, flat lesion	Anterior portion of lesion: smooth, posterior portion of lesion: elevated/wrinkled, fissured
SIZE	10mm x 7mm	10mm x 7 mm	60mm x 30mm

SITE	attached gingiva to alveolar mucosa of inferior to the 31, 41	right lateral border of the tongue, found on posterior third of the tongue	extends from anterior third of left ventral surface of the tongue, to left posterior third of the lateral surface of the tongue

Table 1: Description of clinical lesions presented by Mr. Harrison using margins, colour, appearance, texture, size, and site (MCATSS)

DIFFERENTIAL DIAGNOSIS: (in order of likelihood, refer to Appendix A for rationale)

Figure 1:	***Figure 2 and 3***
- Acute Necrotizing Ulcerative Gingivitis (ANUG)/Acute Necrotizing Ulcerative Periodontitis (ANUP) -Trauma due to External Factors - Periapical Abscess - Chemical Burn	- Oral hairy leukoplakia (OHL)7: - Angular Cheilitis: - Hyperplastic Candidiasis: - Idiopathic Leukoplakia - Dysplasia/Squamous Cell Carcinoma - Hyperkeratosis due to external factors - Chronic Graft vs. Host Disease - Lupus Erythematosus - Geographic Tongue

Table 2: Differential Diagnosis for Mr. Harrison

WORKING DIAGNOSIS

Figure 1: HIV associated periodontal disease: NUG and linear gingivitis erythema (LGE):

Mr. Harrison presents with linear gingival erythema (LGE) which is an early stage of necrotizing periodontitis. 8 This lesion typically occurs within the early stage of HIV positive patients or patients that are immunocompromised. In Mr. Harrison's case, the LGE is localized and presents as a red line two to three millimeters in width adjacent to the free gingival margin. 9Mr. Harrison is unaware of pre-existing periodontal disease and does not complain of pain, eroded gingival margins, bleeding gums, or halitosis which are symptoms that are not associated with HIV-related periodontal disease. In addition, high levels of stress and immunosuppression are etiological factors for HIV associated periodontal disease. The cratered and localized characteristics of this lesion is similar to NUP's progression: rapid loss of supporting bone and tissue. Interestingly, in comparison to Mr. Harrison's CD4+ count of 350/μl, typically HIV patients with NUP have CD4+ counts of less than 200/μl. 7

Figure 2 and 3: Oral Hairy Leukoplakia (OHL) and Angular Cheilitis

Mr. Harrison also presents with inflamed, dry, and rough corners of the lips, which may suggest he is experiencing angular cheilitis, a candida fungal infection. Mr. Harrison's stress and HIV induced immunosuppression are possible etiological factors for this opportunistic infection. In regards to the lesions found on the lateral and ventral borders of the tongue, they match the clinical description of OHL: painless, bilateral, non-removable, white corrugated textured patches. Furthermore, OHL may also spread onto the ventral surface of the tongue, where they usually appear flat, as seen in figure 3. However, OHL commonly occurs among individuals whose CD4+ lymphocyte count is less than 200/μl, which is lower than Mr. Harrison's 350/μl count.7, 10 This lesion caused by the Epstein-Barr virus is most likely a secondary infection due to the client's immunosuppressed state among patients with failing or lack of

ART.10 The client did not present with these lesions in his previous 6 month recall, and recently arrested ART, suggesting that his choice in stopping ART is the etiology of this lesion. Furthermore, candida albicans can be found in association with many OHL lesions, where this trend can be seen with Mr. Harrison's angular cheilitis.

TREATMENT AND CLIENT MANAGEMENT:

A. Treatment of Necrotizing Ulcerative Gingivitis (NUG):

In managing NUG, phase I therapy should include urgent treatment under local anesthesia to resolve the client's discomfort and pain while stopping the progression of disease. The second step of treatment would include periodontal irrigation of the affected area to treat the underlying conditions, as well as eliminating or reducing the etiological factor: immunosuppression. Prior to ultrasonic periodontal debridement, a diluted solution of hydrogen peroxide could be applied to the lesion for antimicrobial and antiseptic reasons.11 After treatment, the client should be prescribed an oral antibiotic (metronidazole) as well as an antibiotic mouth rinse (0.12% chlorhexidine). This phase of treatment would also include referring the client to periodontics for a potential gum graft to treat the ulcerated and cratered gingiva, a well as addressing the possible localized loss of alveolar bone. Phase III includes maintenance and rehabilitation of the area to ensure post treatment healing of the gingiva.12

B. Treatment of Oral Hairy Leukoplakia (OHL) and Angular Cheilitis:

OHL is asymptomatic and usually does not require treatment unless there are aesthetic concerns. However, treatment using antiviral medications to eliminate the Epstein-Barr virus, such as oral acyclovir, and other options such as topical podophyllum resin, retinoids, and surgical removal can eliminate its presence.10 Furthermore, the link between candida albicans and OHL patients warrants the consideration of antifungal treatment.10 Angular cheilitis is a common inflammatory condition that can exist as a bacterial infection, oral thrush, or viral infection. In this case, Mr. Harrison's immunosuppression due to a decreased CD4+ lymphocyte count predisposes him to developing a secondary fungal infection. His current condition, oral candidiasis could be the cause of angular cheilitis. The interaction between bacteria and candida may play a role in the etiology of the lesions present, therefore, treating both bacteria and fungi should be involved in managing HIV-associated lesions. Treatment of angular cheilitis could include placing topical antifungal, steroid, and antibiotic ointments on the localized area. Local antiseptics could be used as well during non-surgical debridement to reduce discomfort.13 See Appendix B for antifungal treatments and dosages.

The responsibilities of a dental hygienist are to continue periodontal treatment as well as consideration and monitoring of client's intake of medications to minimize drug interactions. MCATSS should be recorded prior to every appointment to assess the progress of treatment on the lesions. After treatment in the oral cavity, Mr. Harrison should be referred to a nutritionist in regards to his stress and dietary habits as this could negatively impact his NUG. Furthermore, it is crucial to collaborate with other health care professionals such as the client's general practitioner to stay updated with his medications and current conditions. Most importantly, communication between Mr. Harrison's health professionals are crucial in regards to the increased risk of HIV progression to AIDS; beginning with explaining the importance of continuing ART. It is imperative to address that HIV positive clients with OHL are more likely to acquire AIDS than HIV positive patients without OHL.10 HIV is a lifelong illness that could increase the risk of other diseases as one ages, including an increased risk for cardiovascular disease, bone

loss, and cancers.1 As a result, client centered care, increased awareness of systemic risks, as well as preventative efforts must be considered to increase the client's quality of life.

APPENDIX A

Figure 1:

- Acute Necrotizing Ulcerative Gingivitis (ANUG)/Acute Necrotizing Ulcerative Periodontitis (ANUP): (see working diagnosis for rationale)
-Trauma due to External Factors: Client does not place lozenges, cigarettes, chewing tobacco, partial dentures, or any foreign objects for long periods of time near the lesion. It is unlikely that exposure and chronic friction to foreign objects would likely cause localized recession of this lesion. Mr. Harrison also does not use smoke or smokeless tobacco products, despite the moderate attrition present on S5 and hyperkeratinized, opaque lesion on labial mucosa opposite to recession. These are clinical signs of possible frictional tobacco keratosis. Client states there is no trauma associated with these areas.
- Periapical Abscess: in order to confirm this differential diagnosis, an anterior periapical radiograph of S5 is required. Lack of tooth vitality and periapical abscess formation could result in inflammation, recession, and rapid increase in periodontal depth readings. Evidently, radiographs, tooth vitality test, and periodontal assessments are required.
- Chemical Burn: unlikely, being client does not recall applying aspirin or any other chemical agent directly on soft tissues. Client states there is no trauma associated with these areas.

Figure 2 and 3

- Oral hairy leukoplakia (OHL): (see working diagnosis for rationale)
- Angular Cheilitis: (see working diagnosis for rationale)
- Hyperplastic Candidiasis: Candidiasis are typically observed as an initial manifestation of HIV and observed at CD4 counts of < 300 /microliter. Although Mr. Harrison may not be currently taking antiretroviral medication, his previous xerostomia state paired with his immunosuppression caused by stress and stage 2 HIV could allow for the opportunistic fungal infection to thrive. Although hyperplastic candidiasis are non-removable plaques, similar to the client's lesions, they are found over mucosal surfaces and not lateral borders of the tongue. Diagnosis requires histopathological analysis and identification of hyphae, branching filaments that make up the mycelium of C. albicans. Candidiasis was the most common oral lesion, amongst 217 patients infected with HIV followed by hairy leukoplakia.
- Lichen Planus: bilateral leukoplakia, most likely plaque lichen planus, resemble leukoplakia, elevated. However, found on dorsum of tongue and not ventral surface of tongue
- Idiopathic Leukoplakia
- Dysplasia/Squamous Cell Carcinoma: client is unaware of duration or progression in size of lesion, therefore monitoring is required. Leukoplakia dysplasia is ___ more harmful? However, Mr. Harrison does not present with any risk factors associated with lifestyle habits that may increase his risk of developing oral squamous cell carcinoma: tobacco use, alcohol, family history of cancer, chronic infection, age (fairly young, 38 y/o).

- Hyperkeratosis due to external factors: being Mr. Harrison is experiencing stress, bruxism as well as cheek and tongue biting may be a habit. This is further supported by the presence in moderate attrition to sextant 5 incisal surfaces. However, the ventral location of the lesion in figure 3 is unlikely to be in the range of occlusal forces.
- Chronic Graft vs. Host Disease: Although chronic graft vs host disease's oral lesions have leukoplakia lesions on the dorsum and ventral of the tongue and buccal mucosa, similar to figure 2 an 3, it is more generalized. However, it is unlikely that Mr. Harrison recently received donated bone marrow, as well as no other lesions present on Mr. Harrison's body.
- Lupus Erythematosus: Unlikely, being there are no other systemic lesions and oral lesions are found as erythematous lesions with delicate white striae found on the buccal mucosa, gingiva, and vermillion border. No ulcerations present in Mr. Harrison's lesions presented.
- Geographic Tongue: unlikely, being lesion involves atrophy of filiform and fungiform papillae on the dorsum of the tongue, rather than keratinized growths on lateral surface of the tongue. Lesion also has not been noted to move.

Appendix B: Dosages of Antifungal treatment:

https://www.iasusa.org/sites/default/files/tam/13-5-143.pdf

Table 3: Topical and Systemic Agents for Oral Candidiasis

	Agent	Dosage and Frequency
Topical Agents	**Clotrimazole troches**	10 mg each, dissolve 1 troche in mouth 5x/d for 14 days
	Nystatin oral suspension	500,000 units: swish 5 mL orally as long as possible, 4x/d for 14 days
	Nystatin pastilles	100,000 units: dissolve 1 in mouth 4x/d for 14 days
Systemic Agents	**Fluconazole**	100 mg: 2 tablets on day 1, 1 tablet/d for remainder of 14 days
	Itraconazole oral suspension	10mg or 10 mL: swish and swallow 10 mL/d for 7-10 days, without food
	Voriconazole	200 mg: 1 tablet 2x/d for 14 days or at least 7 days after resolution of symptoms

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linear gingival erythema from HIV-positive patients. J Periodont Res 1995;30(5):355-359.

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