Thrush, plaque, membrane, hyphae-the oral contingent

Commentary

Preface: Candida albicans is a diploid fungus that abounds as a yeast and as filamentous cells, generating opportunistic oral and genital infections. Typically the unicellular yeasts of C. albicans react with the environmental cells and transform into invasive multi cellular filaments, to infect the host tissues, an anomaly known as Dimorphism. Commensal Candida species inhabit the oral cavity as microbes in nearly all the individuals. The carrier phase is non pathogenic, but oral Candida contamination may ensue with pathogenic invasion of the tissue with the Candida micro-organisms. The opportunistic infection with the innocuous microorganism is consequent to the localized or systemic alteration of the host immune response. The implicated organisms frequently are the Candida albicans or uncommonly, the Candida species such as the C. tropicalis, C. glabrata, C. parapsilosis, C. krusei, C. dubliniensis etc. The commonly virulent C. albicans incite a contamination in 70 to 80% instances, whereas the prevalence of C. glabrata infection is roughly 5 to 10%. Candida albicans is a frequent pathogenic fungus, comprising of pseudo hyphae, true, septate hyphae and yeast like structures, in the homo-sapiens. The Oral cavity, Gastrointestinal tract and the Vagina usually lodge the organisms as a commensal.

Keywords: gastrointestinal tract, vagina, fungal infections, candidiasis

Influence and aspects

a. Extended antibiotic or steroid intake, endocrine disorders such as diabetes mellitus, immune suppression due to progressive malignancy, hormonal ingress such as oral contraceptives pre-empt the contamination. Concomitant C. albicans and C. glabrata contamination comprise of a proportionate 80% instances of contamination in individuals.

b. Iatrogenic interventions with Burns, Surgery, Urinary tract catheters and Ulceration of the gastrointestinal tract.

c. The extremities and body when perpetually immersed in water

(1)

Table I Determinant disorders

| Physiological Factors | Old age, Infancy, Pregnancy |
|-----------------------|-----------------------------|
| Local Trauma          | Mucosal irritation, Poor denture hygiene |
| Antibiotics           | Particularly broad spectrum antibiotics |
| Corticosteroids       | Steroid inhalers, Systemic Steroids |
| Malnutrition          | High carbohydrate diet, iron, B12, folate deficiencies |
| Endocrine Disorders   | Hypoendocrine states (hypothyroidism, Addison's disease, diabetes mellitus) |
| Malignancies          | Including Blood disorders (e.g.acute leukaemia, agranulocytosis) |
| Immune Compromised states | Auto immune deficiency syndrome (AIDS), Thymic Aplasia |
| Xerostomia            | Due to irradiation, Drug therapy, Spogren's syndrome, Cytotoxic drug therapy |

Local status: The employment of dentures, a low salivary pH, poor oro-dental hygiene are conditions which enhance the susceptibility to the oral contamination by the micro-organism.

Systemic status: Immune compromised environment such as a human immune deficiency virus ingress or an autoimmune deficiency syndrome (HIV/AIDS), immune-suppression, drug abuse/misuse, antibiotic intake, chemotherapy, immunodeficiency states are disorders which augment the probability of oral infection with Candida sp.

Disease evolution

Opportunistic infections of Candida species may be inhibited by the host on account of

i) The oral epithelium is a locus for cell mediated immune response and impedes the infiltration of microorganisms in the tissues.

ii) Candida sp and co-existent oral microorganisms interactively collaborate, inhibit and counteract the consequences of microbial invasion. The saliva is endowed with mechanical disinfectant attributes and immunogenicity, with the incorporation of the salivary antibodies, which may aggregate the Candida organisms to circumvent the coherence to the epithelial surface and the enzymatic ingredients such as lysozyme, lactoperoxidase, and antileukoprotease. A disintegrated localized and systemic host defence ensures an inherent susceptibility to oral contamination, in conjunction with the virulent Candida micro-organisms and an Oral condition may emerge. A Commensal or a carrier state may not symbolize a disease condition.

Localization of lesions

a. The superficial mucosa or the oral cavity (oral thrush) are frequently invaded.
b. The vagina, epidermal furrows, and nails especially the middle finger constitute the adjunctive, affected sites.

c. **Oral thrush**: A pseudo membrane, comprised of organisms and inflammatory debris, articulates fragile, creamy white coatings, overlying the tongue, soft palate and buccal mucosa. A pin point, vascular, inflamed surface is visible, on extracting the membrane.

d. **Dermo-epidermal**: The common mode of presentation is the acute superficial candidiasis. Chronic mucocutaneous candidiasis is distinguished by a chronic and tenacious mucosal inflammation due to the *Candida* sp and is usually encountered in individuals with immune suppression. Paronychia and Onychomycosis may appear. The dermo-epidermal furrows in the axilla, inguinal region, foci of inflammation, gluteal fold, inter-digital areas and umbilicus are the typical sites of incrimination. Pruritic eczema, peripheral vesicles or pustules may be elucidated.

e. A mutilating cutaneous lesion is a *Candida Granuloma* which develops as a warty, hyperkeratotic papule or plaque.

**Deliberations**

Immune-deficiency in adults on account of a human immune deficiency virus or an autoimmune deficiency syndrome (HIV/AIDS) and chemotherapeutic protocols may also induce a contamination of *Candida*. Topical or systemic corticosteroids such as those employed for Asthmatics, Active cancer therapy, Chemotherapy, Radiotherapy, Dietary predilections, Malnutrition, Mal-absorption, Nutritional deficiencies especially Iron, B12, Folate may incite the emergence of Oral Candida infection. The host defence and epithelial integrity is compromised, the cell mediated immunity is diminished, as is with iron deficiency anaemia or vitamin A and pyridoxine insufficiency. Augmenting carbohydrates in the everyday diet may impact the growth, adhesion, bio-film composition of the *Candida* sp, all of which may be amplified by the carbohydrate availability such as glucose, galactose, sucrose. Broad spectrum antibiotics eradicate the bacterial flora and disorganize the ecological equilibrium of oral microorganisms. Corticosteroids or Broad spectrum antibiotics (tetracycline) may induce the acute oral candidiasis. Local epithelial conversions due to heavy smoking expedite the migration of *Candida* sp, as the smoke consists of nutritional constituents for *C. Albicans* (Figures 1-8).
Hyperkeratosis and Dysplasia in the mucosa subsequent to site-specific deformities, as encountered with lichen planus, may induce adjuvant mucosal infections, especially with Candida albicans. Physical mucosal aberrations such as a fissured tongue/tongue piercing, may manifest with the fungus. The magnitude and character of saliva is a valuable oral defence against Candida sp. Diminished salivary function or hypo-salivation, as characterized by a reduced rate of salivary flow or altered salivary content, augments the infectivity of the micro-organism. Xerostomia amplifies oral contamination. The viscosity and the flow rate of the saliva, however, may not be altered. Oral microbial agents may be eradicated or modified with the administration of broad spectrum antibiotics/corticosteroids/tetracycline. The transformed composition and the ecological equilibrium of the bacterial flora may determine a full blown acute oral candidial infection. Extensive denture protocol, improper denture hygiene, persistent denture usage, nocturnal denture insertion are factors which augment the oral fungal contamination. Dentures may produce a comparatively acidic, moist, anaerobic environment as the denture capped mucosa lacks the ingress of oxygen or saliva.

Analysis and attributes: The clinical presentation is discriminative. The lesions may be exhibited as hyperaemic red or frosty white or as an intermingling of red and white patches (Table 2).
Candidal infections can be categorized as primary or secondary, acute or chronic, pseudomembranous or erythematous. In primary oral candidiasis, the infection is derived from the contaminated oral cavity, whereas in secondary oral candidiasis, the infection is derived from the contaminated systemic candidiasis. Pseudomembranous and erythematous candidiasis may be distinguished by the appearance of the lesions. Pseudomembranous candidiasis is characterized by a white, cottage cheese-like exudate that is easily removed, leaving a raw, erythematous base. Erythematous candidiasis, on the other hand, is characterized by a red, inflamed appearance without a visible exudate. The presence of pseudomembranes, erythroplakia, and leukoplakia are examples of candidal lesions that may be seen in the oral cavity. These lesions can be asymptomatic or may cause discomfort, and they may vary in color, size, and shape. The presence of these lesions can be determined through clinical examination and histopathological analysis. The identification and treatment of candidal infections are crucial for maintaining oral health and preventing the development of more severe conditions.
dermo-epidermal inflammatory infiltrate with intra-epithelial micro-abscesses.1–5 Fungal components are infrequent. Periodic acid Schiff (PAS), Silver Methanamine (Grocott stain - black fungal hyphae with a green environment and is specific for the deteriorating fungus) aids in delineating the organism.1–5

**Chronic candidiasis:** Prominent hyperkeratosis, pseudo-epitheliomatous hyperplasia, compressed ophthalotaxis and a scaly encrustation may appear.1–6 Fungal spores and hyphae may be delineated in the absence of a Periodic acid Schiff (PAS) stain. Granulomatous dermatitis may also emerge with indeterminate granulomas comprising of lymphocytes, plasma cells, epitheloid cells and sporadic giant cells.5,6

**Diffuse candidiasis**

**Systemic GI tracts**

a. The oesophagus and gastric mucosa comprise of punctuate, erosive or ulcerated mucosa, enveloped by a pseudo-membrane, containing the micro-organism, with possible infiltration of the mucosa and sub-mucosal blood vessels.1,5

b. The contamination of the urinary tract induces cystitis and ascending pyelonephritis, renal papillary necrosis, necrotic debris or fungal aggregates configuring “fungal balls” which may incite ureteric obstruction or hydronephrosis.5–6

c. Haematogenous dispersal may elucidate military, necrotic foci.5

**Central nervous system**

a. A frequent fungal contamination of the central nervous system is induced by Candida spp. Numerous micro-abscesses accompanied with non caseating granulomas and restrictive meningitis may be demonstrated.1,5

b. Pulmonary Contamination: Inhalation of the fungal micro-organisms may incite a broncho-pneumonia. A haematoengous dissemination may induce bilateral haemorrhagic nodules.1,5–6

**Oral candidiasis in immune deficiency:** Immune deficient conditions or latent infections such as autoimmune deficiency syndrome or infection with the human immune deficiency virus (HIV/AIDS) elucidates a disastrous course.1,6 With a CD4+ helper T cell count in excess of 500 cells/µl, the occurrence or oral fungal contamination is exceptional and the disorder is frequent when the CD4+ helper T cell counts decline below 100 cells/µl.1,6 The lesions are a therapeutic challenge to contain.

**Cultivation of fungi (candida):** May be possible with solid media such as a Hypertonic Xylose Agar medium or a liquid broth. Designated investigations may include microscopic examination of oral swabs, oral rinse or oral smears, in order to isolate the fungus.1,4,6

Molecular analysis of Candida employs a Real time Polymerase Chain Reaction (RT-PCR). Monoclonal antibodies besides the Rapid agglutination tests (RLA) may also be applicable. Debatable conditions such as a “Candida Leukoplakia” mandate an evaluation of a histological specimen.1,6 Imprint smears and tissue material may be analyzed with the Periodic acid Schiff’s (PAS) stain. The carbohydrates in the fungal cell wall stain magenta. Gram Positive reactivity on Gram’s stain may be elucidated by the micro-organism.1,2,5

Serological armamentarium comprises of a Whole cell agglutination, Immune-fluorescence, Immune-enzymatic assays employed for the demonstration of Immunoglobulin G antibodies delineated against the Candida micro-organism, besides Radio-immunological analyses.1,5 Real time Polymerase Chain Reaction (RT-PCR) analyzes the Candida de-oxo ribonucleic acid (DNA) in order to detect the Candida micro-organism in the oral cavities of a high risk population.1,2,4 The methodology also segregates the fungi Candida Albicans from the adjuvant species such as C. glabrata, C. krusei and C. Parapsilosis.1,6 Anti Candida albicans antibodies may be demonstrated on immune–histochemistry (IHC) and immune fluorescence may also be employed for arriving at a conclusion.1,5–6

**Therapeutic interventions:** Therapy for concomitant ailments, appropriate diet, pro-biotic employment is advocated. Oral Candida infection may be ameliorated by the augmenting the oral hygiene or with an antimicrobial mouthwash.1,2 Topical antifungal agents such as Nystatin, Miconazole, Gentian violet Amphotericin B are considered efficacious for the immune-competent patients.1,5 Immune-compromised individuals, patients with autoimmune deficiency syndrome or infection with the human immune deficiency virus (HIV/AIDS) or those on Chemotherapy necessitate Systemic Antifungal agents.1,5–6

**Disease outcome:** Oral Candida contamination following a topical or systemic therapy depicts an excellent prognosis.1 However, with the latent or concomitant predicaments, a declining salivary flow or with adjuvant immune deficient disorders, the oral contamination may not be remediable.1,6–11 Contamination with Candida is a hallmark of an intrinsic disease process, thus the comprehensive prognosis may depend upon the primary derangement.

**Conclusion**

Pathogenic invasion of the tissue with the innocuous Candida micro-organisms, frequently the Candida albicans is enunciated as an opportunistic infection, consequent to the localized or systemic alteration of the host immune response. Extended antibiotic or steroid intake, endocrine disorders such as diabetes mellitus, immune suppression due to progressive malignancy, hormonal ingress such as oral contraceptives pre-empt the contamination. Real time Polymerase Chain Reaction (RT-PCR) offers a better evaluation and analysis of candida species. Monoclonal antibodies besides the Rapid agglutination tests (RLA) may also be applicable. Debatable conditions such as a “Candida Leukoplakia” mandate an evaluation of a histological specimen. Therapy for concomitant ailments, appropriate diet, pro-biotic employment or augmenting the oral hygiene or an antimicrobial mouthwash is advocated. Oral Candida contamination in combination with the latent or concomitant predicaments, a declining salivary flow or with adjuvant immune deficient disorders, may not be remediable.1,6 Contamination with Candida is a hallmark of an intrinsic disease process and the ultimate prognosis may depend upon the primary derangement.

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None.

**Conflict of interest**

The author declares that there is none of the conflicts.
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