Oral candidiasis is most common opportunistic disease of the oral mouth cavity. It is common and under diagnosed among the aging, particularly in those who wear dentures, many cases is avoidable with a good mouth care regimen. It is the also mark of systemic infections disease, such as diabetes mellitus and is a common problem among the immune compromised. Oropharyngeal candidiasis is caused by an overgrowth of the oral cavity by a yeast-like fungus, candida[^23,24].

The important ones are *C. albicans*, *C. glabrata*, *C. tropicalis*, *C. pseudotropicalis*, *C. guilliermondii*, *C. krusei*, *C. lusitaniae*, *C. parapsilosis*, *C. stellatoide*, *C. albicans*, *C. glabrata*, and *C. tropicalis* represent more than 80% of isolates from clinical infection[^25]. Oral candidiasis is the most common human fungal infection[^26,27]. Especially in early and later life, the general population, carriage rates have been reported to range from 20% to 75% without any symptoms. The incidence of *Candida albicans* isolated from the oral mouth cavity has been reported to be 45% in neonates[^28], 45%–65% of healthy children[^29], 30%–45% of healthy adults[^30,31], 50%–65% of people who wear removable dentures, 65%–88% in those residing in acute and long term care facilities[^32-34], 90% of patients with acute leukaemia undergoing chemotherapy[^35] and 95% of patients with HIV[^36]. *C. albicans* is a common commensal of the mouth and generally causes no problems in well people. Overgrowth of candidiasis however can increase to local discomfort, an altered taste sensation, dysphagia from oesophageal overgrowth resulting in poor diet, slow recovery, and prolonged hospital stay. In immunocompromised patients and infection can spread through the bloodstream or upper gastrointestinal tract (GIT) leading to severe infection by significant morbidity and mortality. Systemic candidiasis infection carries a mortality rate of 71% to 79%[^37]. It is important for all physicians looking after older patients to be alert of the risk factors, diagnosis, and treatment of oral candidiasis infection. In a recent study 30% of clinicians said they would prescribe nystatin for oral candidiasis on the call of nursing staff without examination of the oral cavity[^38]. This is unfortunate as other pathology labs may be missed, the diagnosis may be incorrect, and disappointment to address risk factors may lead to recurrence of the oral candidiasis.
There are a number of different types of oral candidiasis including infection acute pseudomembranous, acute atrophic, chronic hyperplastic, chronic atrophic candidiasis infections average rhomboid glossitis, and angular cheilitis \(^{(39)}\). The most discrete injury represents conversion from benign colonisation to pathological overgrowth.

*Pseudomembranous candidiasis or thrush* is characterized by extensive white pseudomembranes containing of desquamated epithelial cells, fibrin, and fungal pseudo hyphae. These white patches occur on the surface of the labial and buccal mucosa, hard, soft palate, tongue, periodontal tissues, and oropharynx, membrane can usually scrape off with a swab to expose an underlying erythematous mucosa and diagnosis is usually straightforward as it is easily seen and one of the commonest forms of oropharyngeal candidiasis accounting for almost a third. \(^{(40)}\) Diagnosis can be confirmed microbiologically examination result either by staining a smear from the affected area and by culturing a swab from an oral rinse. Predisposing factors take in extremes of age, diabetes mellitus, patients who have HIV/AIDS, leukemia, those applying steroid aerosol inhalers, broad spectrum antibiotics, and psychotropic drugs, patients who are terminally ill and other conditions that can give rise to white patches in the oral mouth are lichen plan us and squamous cell carcinoma, lichenoid reaction, and leukoplakia.

Acute atrophic candidiasis is usually associated with a burning sensation in the mouth and on the tongue. The tongue may be bright red similar to that seen with a low serum B12 and low folate, and low ferritin. Diagnosis may be difficult of patients but should be considered in the difference diagnosis of a sore tongue especially in a frail old patient with dentures who has received antibiotic therapy, who is on inhaled steroids. A swab from the tongue and buccal mucosa may help diagnosis.

*Chronic hyperplastic candidiasis infection* characteristically occurs on buccal mucosa or lateral border of the tongue as speckled or homogenous white lesions and the lesions usually occur on the buccal mucosa, lateral borders of the tongue. There is an association with smoking \(^{(41)}\) and complete resolution seen to be dependent on cessation of tobacco smoking. This situation can development to severe dysplasia or malignancy and is sometimes refer to as candida leukoplakia. *Candida* spp. is not always isolated from lesions of mouth leukoplakia, suggested that the finding of *Candida* species in these premalignant wounds is a
complicating factor rather than a causative agent one. This condition may be confused with lichen planus, pemphigoid/pemphigus, and squamous cell carcinoma infection.

**Chronic atrophic candidiasis** also called as “denture stomatitis” is characterised by localized infection chronic erythema of tissues covered by dentures. Lesions commonly occur on the palate, upper jaw but may also affect mandibular tissue. Diagnosis requires removal of dentures and watchful inspection, swabs may be taken for confirmation. It is quite most common with incidence rates of up to 65% reported.

**Median rhomboid glossitis** is a chronic infection symmetrical area on the tongue of mouth anterior to the circumvallate papilla it’s made up of atrophic filiform papillae. Diagnosis by Biopsy of this area usually yields Candida in 85% of cases. It tends to be associated by smoking of nicotine and the use of inhaled steroids.

**Angular cheilitis** is an erythematous fissuring at one otherwise both corners of the oral and is usually associated with an intraoral candidal infection. Further organisms implicated are staphylococci, streptococci gram positive bacteria. In the case of staphylococci gram positive bacteria the reservoir is usually the front area of the nostrils and spread to the angles of the oral has been confirmed by phage typing. Facial wrinkling at the corners of the mouth and with the nasolabial fold especially in old persons leads to a chronically moist environment that predisposes to this lesion. This wrinkling is worse in long period denture wearers because near is resorption of bone on which the dentures rest lead to a reduction in height of the lower face area when the mouth is closed. Another factors implicated in the etiology of this condition are iron deficiency anemia, vitamin B12 deficiency.

### RISK FACTORS

**Pathogen**

Candida is a fungus and was first isolated in 1844 from the sputum of a mycobacterium tubercle bacilli patient. Like other fungi, they are non-photosynthetic, eukaryotic cell organisms with a cell wall that lies external to plasma membrane. There is a nuclear pore complex within the nuclear membrane and the plasma membrane contains large quantities of sterols, usually ergosterol. A part from a few exceptions and the macroscopic and microscopic cultural characteristics of the different candida spp. are similar. They can metabolise glucose under both aerobic situation and anaerobic environments. Temperature influences their growth with high temperature such as 37°C that are present in their potential
host and promoting the growth of pseudohyphae. They have been isolated from animal and environmental sources. They can be found on or in the man body with the gastrointestinal tract, the vagina, and skin being the common sites and *C. albicans* being the most common isolated from these sites. They require environment sources of fixed carbon for the development. Filamentous progress and apical extension of the filament and formation of lateral branches are show with psedohyphae, mycelium, single cell division is associated with yeasts [49].

Several studies have demonstrated that infection with candidiasis is associated with certain pathogenic variables and adhesion of candida to epithelial cell walls, an main step in initiation of most infection, is promoted by certain fungi cell wall components such as mannose, C3d receptors, mannoprotein, and saccharins [50-52]  The degree of hydrophobicity [53] and ability to bind to host fibronectin [54] has also been reported to be important in the initial stages of infection and Other factors implicated are germ tube formation, presence of mycelia and persistence within epithelial cells and endotoxins, induction of tumour necrosis cells factor, and proteinases [55-60]. Phenotypic switching which is the ability of certain strains of *Candida albicans* to change between differ morphologic phenotypes has also been implicated [61].

Oral candidiasis can be three types classified as follows:

1. Acute oral candidiasis
   - Acute pseudomembranous candidiasis (thrush) infection.
   - Acute atrophic (erythematous) candidiasis infection.

2. Chronic oral candidiasis
   - Chronic hyperplastic candidiasis (candidal leukoplakia) infection.
   - Denture induced candidiasis chronic atrophic (erythematous candidiasis) infection.
   - Median rhomboid glossitis infection.

3. Angular cheilitis (stomatitis)

Pathogen has peculiar properties that increase its infectivity rate in the correct environment.

- Host factors could be local and/or systemic infection.
• Local factors include wearing dentures and impaired salivary gland function, inhaled steroids, and oral cancer infection.
• Systemic factors take in extremes of age, smoking, diabetes mellitus, Cushing’s syndrome, immunosuppression disease, malignancies, nutritional deficiencies, and use of antibiotics.

Local factors

*Impaired salivary gland function* can predispose to oral candidiasis[^62]. Secretion of saliva causes a dilutional effect, removes microorganisms from the mucosa. Antimicrobial proteins in the saliva of mouth such as lactoferrin, sialoperoxidase, lysozyme and histidine-rich polypeptides, and specific anti-candidiasis antibodies, interact with oral mucosa and prevent overgrowth of candida. Therefore situations such as Sjögren’s syndrome and radiotherapy of the head/neck or drugs that reduce salivary secretions can lead to an increased risk of oral candidiasis.

*Drugs* such as inhaled steroids have been exposed to increase risk of oral Candidiasis[^63] by possibly suppressing cellular immunity and phagocytosis and the local mucosal immunity reverts to usual on discontinuation of the inhaled steroids[^64].

*Dentures* predispose to infection with candida spp. in as many as 65% of elderly publics wearing complete upper dentures. Wearing of dentures creates a microenvironment conducive to the growth of candida spp. with low oxygen, low pH, and an anaerobic environment and this may be due to enhanced adherence of Candidiasis to acrylic, reduced saliva of the mouth flow under the surfaces of the denture fittings, improperly fitted dentures, and poor oral hygiene. *Other factors* are oral cancer/leukoplakia and high carbohydrate diet. Growth of candida in saliva is enhanced by the occurrence of glucose and its adherence to the oral epithelial cells is enhanced by a high carbohydrate diet[^65].

Systemic factors

*Extremes of life* predispose to microorganism infection because of reduced protection. *Drugs* such as broad spectrum apply antibiotics alter the local mouth flora creating a suitable environment for candida to proliferate[^66]. The normal mouth flora is restored once the antibiotics are discontinued. Immuno-suppressive medicines such as the antineoplastic agents
have been seen in several studies to predispose to oral candidiasis infection by altering the oral flora, disrupting the mucosal oral surface and altering the character of the saliva \cite{67, 68}.

*Other factors* are tobacco smoking, diabetes, Cushing’s syndrome, immunosuppressive patients conditions such as HIV infection, malignancies such as leukaemia, deficiencies of nutrition — vitamin B deficiencies occurs have been particularly implicated. Ninane found that 15%–60% of people suffering with malignancies cells will develop oral candidiasis while they are immunosuppressed \cite{69}.

In those with HIV infection rates of between 7% to 48% have been quoted and more than 90% has been report in those with advanced infection. Relapse rates are between 30% and 50% on completion of antifungal infection treatment in severe immunosuppression \cite{70}.

**MANAGEMENT**

Taking a history followed by a thorough examination of the oral mouth, looking at the soft and hard palate, examining the buccal mucosa in those wearing dentures after they have been erase are usually good starting points and Predisposing factors are identified as notice above and resolved if possible, and the type, severity, chronicity of the infection are assessed. The correct diagnosis is usually made on finding the characteristic lesion, ruling out other possibilities, the response to antifungal infection treatment. Acute pseudomembranous and chronic atrophic oropharyngeal candidiasis can be treated based on clinical features but growth culture and sensitivity testing should be undertaken if primary therapy is unsuccessful. Imprint cultures, where sterile foam pads dipped in Sabouraud’s broth media are placed for 30 seconds on the lesion and then placed on SDA agar containing chloramphenicol for an hour after which they are incubated, have and used for identification of *C*.spp. Acute atrophic infection and chronic hyperplastic infection forms may mimic other lesions, by biopsy is recommended in addition to empirical treatment to rule out more serious lesions such as squamous cell carcinoma.

Oral hygiene and topical antifungals agent are usually adequate for uncomplicated oral candidiasis. *Oral hygiene* involves cleaning teeth, buccal cavity, tongue, and dentures, if present, every day. Dentures should be cleaned and disinfected day-to-day and left out overnight or for at least 6th hours daily. The dentures should be soaked in a denture cleaning solution agent such as chlorhexidine as this is additional effective in eliminating candida than
This is because dentures have asymmetrical and porous surfaces to which candida spp. easily adheres and brushing alone cannot take out them. When rinsing the mouth with the topical antifungal and dentures should be removed proper to allow contact between the mucosa and the antifungal. The patient should confirm that the whole mucosa is coated with the antifungal agent and held in the mouth for a few minutes. The incorporation of antifungal with a denture liner has been recommended for infectious person with dentures who find it hard to hold the antifungal in their mouth for a few minutes. Also the mucosal surface should be brushed regularly uses with a soft brush. Later disinfection, dentures should be allowed to air dry as this also kills adherent candida agra on dentures. Chlorhexidine can discolour both dentures and natural dentition if not removed adequately later disinfection. A referral to a dentist might be required for those with poorly fitting dentures as these predispose to fungal infection by break down the epithelial cell barrier. Other denture cleaning methods not routinely used but shown to be in effect are ultrasonic cleaning tanks and with a suitable solution.

Regular oral and dental hygiene with periodic oral checkup will prevent most cases of oral candidiasis in those with dentures. Combining drugs nystatin with chlorhexidine digluconate, an antiseptic apply to disinfect dentures, inactivates both drugs therefore this combination should not be applied. The dentures should be removed each one time the mouth is rinsed with the oral antifungal agent preparation in established fungal cases of denture stomatitis and the dentures soaked in chlorhexidine before putting them back in the mouth area.

*Topical antifungal treatment* is the recommended first line cure for uncomplicated oral candidiasis and where systemic treatment is needed topical therapy should carry on as this reduces the dosage and duration of systemic treatment required. The systemic adverse effects and medicine interactions that happen with the systemic agents do not occur with topical agents. Treatment in early part of the twenty century was with gentian violet, an aniline dye, but because of drug resistance developing, side effects, such as stain of the oral mucosa, it was replaced by a polyene antibiotic, nystatin, medicine discovered in 1951 and the amphotericin B, find out in 1956. They act by binding to sterols in the cell membrane of fungal and altering cell membrane permeability. Nystatin and amphotericin drug are not absorbed from the gastrointestinal tract and used by local application in the mouth. Miconazole and an imidazole, can be apply as a local application in the oral cavity but its
apply in this way is limited because of potential side effects such as symptom vomiting and diarrhoea. Other medicine belonging to this class is clotrimazole, ketoconazole. Nystatin is the most widely apply topical agent for the treatment of oral candidiasis (thrush). It is available as a mouth rinse, pastille, and suspension. It should be used as a rinse 4th times a day for two weeks. It can cause nausea, vomiting, and diarrhoea. The mouth rinse contains sucrose and is useful in edentulous effecting infections patients and those with xerostomia such as infectious patients receiving radiotherapy and those with HIV infection [79]. Clotrimazole troche can be an alternative for those infectious patients who find nystatin suspensions unpalatable.

Systemic antifungal drug therapy in oropharyngeal candidiasis is appropriate infectious patients intolerant of or refractory to topical treatment use and those at high risk of developing systemic infections. Both nystatin mouth rinses and clotrimazole troches have a high sucrose content and if mouth tooth decay is a concern or oral candidiasis is complicated by diabetes and steroid use or an immune-compromised state, triazoles which include fluconazole or itraconazole/day has been found to be effective in these cases [80]. Ketoconazole is also as effective as fluconazole and itraconazole but its apply in elderly patients is not recommended due to medicine interactions and side effects, which include hepatotoxicity.

Fluconazole is a potent and choosy inhibitor of fungal enzymes include in the synthesis of ergosterol and important constituent of the plasma cell membrane. Therefore disrupts cell wall development increasing to leakage of cellular contents and cell die. It is well absorbed by the gastrointestinal tract (GIT) and 90% of the levels achieved with intravenous administration and the levels in saliva.

- Diagnosis is generally made on clinical grounds with lab. Testing and to exclude potentially other serious oral mouth lesions especially squamous cell carcinoma.
- Pre-disposing factors should be treated or eliminated where feasible.
- Best mouth hygiene is important.
- Topical antifungals medicine given for two weeks is usually effective.
- Systemic antifungals should be given in certain circumstances.
Sputum is also similar to that in plasma. It is preferred and does not have the same hepatotoxicity as the imidazoles. It’s now recorded in the dental practitioners’ formulary as well as the British National Formulary and is therefore broadly apply both in dental as well as medical practice but there are problems with resistance.

Itraconazole has a wider spectrum of action than fluconazole and it is therefore valuable in salvage treatment of the immune-compromised patients with fluconazole resistant candida. Increasing resistance to antifungals has become increasingly most common since the introduction of fluconazole especially in the patients with advanced immunocomprise disease and recurrent and extended term treatment [81-82].

Angular cheilitis is treated with antifungal steroid creams and ointments any concurrent intraoral lesion is also treated at the same time and nutritional deficiencies should be excluded and cured if found. Failure to respond to therapy particularly in chronic atrophic candidiasis is usually due to non-compliance with treatment.

Prophylaxis with antifungal managers reduces incidence of oral candidiasis in patients with cancer undergoing treatment [83] and fluconazole has been found to be more effective than topical polyenes [84]. Prophylaxis on either a daily or weekly basis with antifungal reduces the incidence of oropharyngeal candidiasis in patients with HIV reductions being most marked in those with low CD4 counts and recurrent oral candidiasis [85-88]. The use of a chlorhexidine rinse only in bone marrow transfer patients as prophylaxis was found to be very effective [89].

PROGNOSIS

The prognosis is bad for oropharyngeal candidiasis with in appropriate and in effective treatment. Relapse when it occurs is more often than not due to weak compliance with therapy, failure to erase and clean dentures appropriately, or inability to resolve the underlying/predisposing factors to the infections.