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A CASE OF OESOPHAGEAL CANDIDIASIS IN PREGNANCY

Obstetrics & Gynaecology

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ABSTRACT

Candida organisms are the most common oesophageal infection in the immunocompromised AND HIV. Candida albicans accounts for vast majority. Candida esophagitis is included in the differential diagnosis of refractory nausea, dysphagia and odynophagia, vomiting and weight loss
Reporting a case of A 35years,G4P1L1A2 with 15 weeks 5days gestational age presented with complaints of epigastric burn and oral ulcers since one month and difficulty to take oral feeds both solids and liquids since 15days. No significant family history. Patient is a k/c/o hypothyroidism since 9years, on tablet Thyronorm 100mcg OD
On examination – mild pallor was present and Oral cavity showed multiple white plaques on buccal mucosa, stomatitis, cheilitis and geographic tongue present.
Endoscopy - Multiple superficial to deep ulcers seen starting from 20cm from incisors teeth extending up to GEJ, multiple biopsies taken. Antral erosions seen and oesophageal candidiasis present. Endoscopic biopsy – non specific ulcer
Treated with injection pantoprazole infusion over 24hours (200mg in 100ml NS) and tablet flucanazole 150mg for 14days

KEYWORDS

Esophageal candidiasis, Candida albicans, Epigastric burn.

INTRODUCTION:

Esophageal candidiasis is the most common cause of infectious esophagitis. Candida albicans causes 88 percent of infectious esophagitis, herpes simplex virus causes ten percent, and CMV- two percent. Candida infections of the oesophagus are opportunistic infections that affect immunocompromised patients the most. Candida can be found in the mouth's regular flora. When the host's defence mechanisms are compromised, candida multiplies on the oesophageal mucosa and creates adherent plaques. Antifungal medicines, both oral and intravenous, can be used to treat esophageal candidiasis.[1,2]

CASE REPORT:

A 35years female, G4P1L1A2, with 15 weeks 5days gestational age presented with complaints of epigastric burn and oral ulcers since one month and inability to take oral feeds both solids and liquids since 15days. No significant family history. Patient is a k/c/o hypothyroidism since 9years, on tablet thyronorm 100mcg OD.

EXAMINATION:

Patient was poorly built and nourished, conscious and oriented Vitals stable, no signs of dehydration BMI – 16.89kg/m2. On examination – mild pallor was present.

Oral cavity showed multiple white plaques on buccal mucosa, stomatitis, cheilitis and geographic tongue present (FIGURE 1).

Breast, thyroid and spine clinically appeared normal RS, CVS, CNS - normal P/A – uterus 14-16weeks size, FHS present, Pfannenstiel scar present, healed by primary intention.

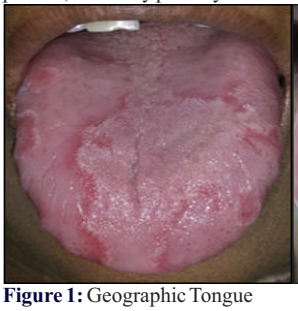

Figure 1: Geographic Tongue

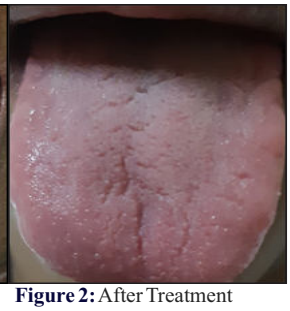

Figure 2: After Treatment

Investigations:

Haemoglobin–8.6g/dl
Peripheral smear – microcytic hypochromic anemia
Thyroid profile – T3 – 104.5; T4 – 4.03; TSH - >100
Iron profile, coagulation profile, serum vitamin b12 and serum folic acid were within normal limits
Serology – negative
ANA – positive

Endoscopy Done On 3/5/2021 -

Multiple superficial to deep ulcers seen starting from 20cm from incisors teeth extending up to GEJ, multiple biopsies taken. Antral erosions seen and oesophageal candidiasis present.

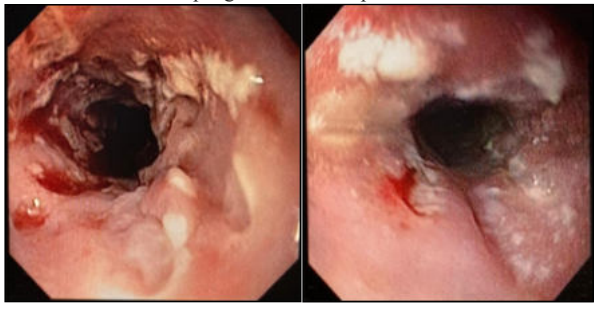

Figure 3 And **Figure 4** – Endoscopic Changes

HPE – Multiple biopsy bits lined by stratified squamous epithelium exhibiting acanthosis. Few of the bits show inflammatory granulation tissue response comprising of proliferating vessels and dense inflammatory cell infiltrate comprising of neutrophils, plasma cells, lymphocytes and karryorhectic debris. No atypical cells seen. Impression– non specific ulcer

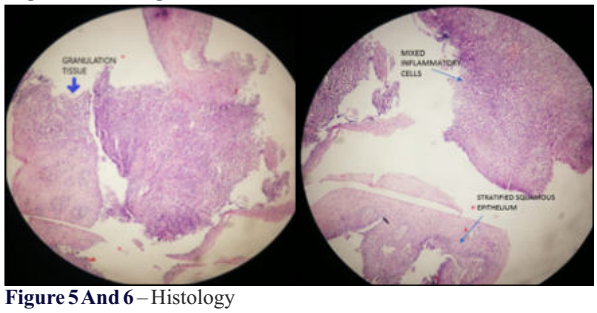

Figure 5 And 6 – Histology

Past History:

Patient gives history of oral ulcers on and off since 10years, and was on proton pump inhibitors and local gel.

Endoscopy done on 21/11/2014 –Oesophagus shows multiple superficial ulcers in lower third, ?pill related, ?post viral CMV/ Herpes. Multiple biopsies taken. No stricture.

Endoscopic biopsy on 21/11/2014 – non specific ulcers probably drug induced.

TREATMENT GIVEN:
Mucaine gel
Zytee gel
Inj pantoprazole
Candid mouth paint
Tab Flucanazole 150mg OD for 14days
Inj ferric carboxymaltose 500mg IV
Multivitamin injection – 5doses
Tablet thyronorm 125mcg OD

DISCUSSION:
Candida oesophagitis has been linked to advancing age, HIV infection, and the use of corticosteroids and no gender difference, median age of 55.5 years. Incidence rate in the general population ranges from 0.32 to 5.2 percent. HIV-positive people have a 9.8 percent prevalence rate. Non-HIV individual rates appear to be rising, presumably as a result of co morbidities like diabetes mellitus or drugs like antibiotics, proton pump inhibitors, and corticosteroids. Some studies relates smoking tobacco predisposes to oesophageal candidiasis.[2,3,4]

Pathophysiology:
Candida albicans can be found in the mouth as part of a healthy flora. The oesophagus epithelial layer is prone to infection and colonisation by Candida due to a lack of cell-mediated immunity. Candida overgrows and attaches to the mucosa of the oesophagus, creating white-yellow plaques. Upper endoscopy can reveal the plaques, which do not wash away with water irrigation. These plaques can be found throughout the oesophagus or in specific areas such as the upper, middle, or distal oesophagus.[2]

Histopathology:
The gold standard for diagnosing Candida is histologic confirmation. Biopsy or cytology stained with hematoxylin and eosin shows pseudohyphae, diagnostic for oesophageal candidiasis. Desquamated parakeratosis is a type in which clusters of squamous cells have detached or are in the process of detaching from the main squamous-lined tissue. This observation, however, is not unique to oesophageal candidiasis. Acute inflammation and/or intraepithelial lymphocytosis may be seen on pathology.[2]

Patients with oesophageal candidiasis are asymptomatic or may experience a variety of symptoms like dysphagia, odynophagia, and retrosternal chest discomfort. Odynophagia is the most common symptom. Abdominal pain, heartburn, weight loss, diarrhoea, nausea, vomiting, and melena are some of the other symptoms.[1,2]

The only finding may be an associated infection of the oropharynx with candida.

EVALUATION:
Upper endoscopic examination is done to diagnose oesophageal candidiasis. The presence of white plaques or exudates on the oesophageal mucosa is diagnostic. Plaques and exudates cling to the mucosa and are not washed away by irrigation. Mucosal fractures or ulcerations are also possible.[1]

KODSI grading on endoscopy:
Grade 1 – a few white plaques upto 2mm without ulceration.
Grade 2 – multiple white plaques >2mm without ulceration.
Grade 3 - linear, nodular elevated plaques with frank ulceration.
Grade 4 – grade 3 + increased friability of the mucous membranes and occasional narrowing of the lumen.

MANAGEMENT:
Unlike oropharyngeal candidiasis, oesophageal candidiasis requires systemic antifungal treatment rather than topical treatment. Oral fluconazole 200 to 400 mg per day for 14 to 21 days is recommended for oesophageal candidiasis. If patients are unable to take oral preparations, intravenous Fluconazole 400 mg daily can be given, followed by de-escalation to oral Fluconazole once the patient is able to take oral medications.

Recurrent oesophageal candidiasis can be treated with fluconazole 100 to 200 mg three times per week. Micafungin 150 mg IV was found to be equivalent to fluconazole 200 mg daily in clinical trials. Other treatment options includes 200 mg of itraconazole or 200 mg of Voriconazole twice day for 14 to 21 days. In patients with resistant candida oesophagitis, amphotericin B deoxycholate 0.3 to 0.7 mg/kg daily can be given, it has substantial pharmaceutical side effects and should be avoided if feasible. Posaconazole 400 mg twice daily has been effective in refractory oesophageal candidiasis.[5,6,7] Caspofungin is preferred over amphotericin.

Oesophageal candidiasis is an opportunistic infection that most commonly affects immunocompromised people, the cause of the immunosuppression should also be identified and treated.[8,9,10]

Patients with odynophagia or dysphagia who are immunocompromised and where there is no definitive diagnosis, can be treated empirically with a 14-21 day course of antifungal medication. Upper endoscopy should be performed if symptoms do not improve after 72 hours of medication.[11]

The dose of the azole agents does need modification in patients with renal insufficiency.

Azoles are considered teratogenic, so in pregnant patients with oesophageal candidiasis, Amphotericin B is preferred.
Other common causes of oesophagitis are as follows:[1,12,13]
· Cytomegalovirus
· Herpes simplex virus
· Eosinophilic esophagitis
· Pill-induced esophagitis
· Gastroesophageal reflux disease
· Radiation esophagitis
· Bacterial esophagitis (from *Lactobacillus*, B-hemolytic streptococci, *Cryptosporidium*, *Pneumocystis carinii*, *Mycobacterium avium* complex, *Nocardia*, *Mycobacterium tuberculosis*, *Leishmania donovani)*

PROGNOSIS:
There are no studies that specifically address the prognosis of oesophageal candidiasis. Antifungal medicines are usually effective in treating it. Infections that are resistant or refractory to therapy may require different medications or long-term antifungal treatment to prevent recurrence.[7]

COMPLICATIONS:
Oesophageal candidiasis can cause oesophageal ulcers, which can lead to oesophageal perforation and upper gastrointestinal haemorrhage, as well as weight loss, malnutrition, sepsis, candidemia, oesophageal stricture and bronchial tree fistula formation.[14,15,16,2]

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