

Case Study on an 86-Year-Old Patient with Esophageal Candidiasis

Nandini P^{1*} and V Mahender Reddy²

¹Department of Pharmacy Practice, Jyothismathi Institute of Pharmaceutical Sciences, Karimnagar, Telangana, India

²Professor, Department of Pharmacy Practice, Jyothishmathi Institute of Pharmaceutical sciences, Karimnagar, Telangana, India

*Corresponding author: Nandini P, Department of Pharmacy Practice, Jyothismathi Institute of Pharmaceutical Sciences, Karimnagar, Telangana, India, Tel: 096181 53535; E-mail: nandinichowdary2002@gmail.com

Received: August 22, 2025; Accepted: September 16, 2025; Published: September 26, 2025

Abstract

The most typical esophageal infection is *Candida esophagitis* (CE). Although non-*albicans* species like *Candida glabrata* and *Candida tropicalis* are becoming more common, *Candida albicans* is the primary cause. In up to 20% of cases, the gastrointestinal commensal *Candida albicans* colonizes the esophagus. *Candida* species can cause serious systemic infections such bacteremia in addition to local mucosal membrane infections of the esophagus, oropharynx, and vagina. Dysphagia and odynophagia, or difficulty swallowing, are common symptoms of CE patients and are frequently localized to one specific retrosternal region. Oral "thrush" frequently coexists with immunosuppressed people (such as those suffering from acquired immunodeficiency syndrome, or "AIDS"). Even though many CE patients may not have any symptoms, the diagnosis can be aided if a patient experiencing esophageal symptoms has oropharyngeal candidiasis.

1. Introduction

1.1 Candidiasis

The fungus *Candida* is the source of the opportunistic infection known as candidiasis. Only when the right circumstances are present do they turn pathogenic. It may impact the penis, vagina, oral cavity, or other bodily parts. Thrush is the commonly used term for oral candidiasis. One of the most prevalent fungal illnesses that affects the oral mucosa is oral candidiasis. These sores are brought on by the yeast *Candida albicans*. About 30% to 50% of people have *Candida albicans*, which is one of the elements of typical oral microbiota. As the patient gets older, the rate of carriage rises. Sixty percent of dentate patients over 60 have *Candida albicans* in their mouths [1].

Citation: Nandini P, Reddy VM. Case Study on an 86-Year-Old Patient with Esophageal Candidiasis. Clin Case Rep Open Access. 2025;8(3):348.

©2025 Yumed Text.

1.2 Candida-induced esophagitis

Even while *Candida* is thought to be a normal part of the human digestive system, an imbalance can lead to illness. As common flora, the yeast organism *Candida* establishes itself in the outermost epithelium of the urogenital and alimentary canal systems in healthy humans. A prevalent infection, oropharyngeal candidiasis is more common in patients receiving antibiotics, chemotherapy, radiation therapy to the head and neck, and those with immune deficiency conditions like acquired immunodeficiency syndrome (AIDS). It is also more common in older persons or newborns who wear dentures. Retrosternal pain during swallowing, often known as odynophagia, is a hallmark of *Candida* esophagitis. Immunosuppression, prior esophageal illness, and a few additional diseases that promote the formation of *Candida* in the digestive tract are the primary risk factors for esophageal candidiasis that have been identified to date [2].

1.3 Epidemiology

Esophageal candidiasis is the most common cause of infectious esophagitis. Eighty-eight percent of patients with infected esophagitis have *Candida albicans*, ten percent have herpes simplex virus, and two percent have CMV. An individual experiencing esophageal candidiasis is 55.5 years old on average. Esophageal candidiasis incidence rates in the general population range from 0.32 to 5.2%, according to several studies. The prevalence among patients with HIV is 9.8% [3,4].

1.4 Pathophysiology

The nonkeratinized stratified squamous epithelium, a protective innate immunological mechanical barrier, naturally lines the mucous membrane of the esophagus.



As a result, in certain people, *Candida albicans* may make up around 20% of the commensal that colonizes the esophagus.



The growth and colonization of *Candida albicans* might result from immune system-compromising processes and localized lesions in the upper cortex of the esophagus.



Candida then settles to the mucosal membrane and develops white, yellow spots. The plaques are visible during upper endoscopy, and water irrigation is unable to remove them from the mucosa.



These plaques may be concentrated in the upper, middle, or distal esophagus, or they may be dispersed over the entire esophagus.

1.5 Risk factors

According to numerous studies, the prevalence of esophageal candidiasis in the general population ranges from 0.32% to 5.2%.

1. Gender
2. Age
3. Comorbidities: comorbid conditions including diabetes mellitus, peptic ulcer disorders, or drugs like corticosteroids and antibiotics used to recipients of organ transplants.
4. Use of Proton-Pump Inhibitors
5. Smoking.

1.6 Diagnosis

Isolating candida from sputum and stool specimens cannot diagnose a candida infection because it is a normal mycotic flora in the oral and gastrointestinal system; histological evidence is frequently required for this diagnosis. Multiple abscesses and an immediate inflammatory response are the pathologic characteristics of the endoscopic biopsy tissue. Fungal spores and pseudohyphae are evident, and neutrophils predominate.

Endoscopy: The preferred method of diagnosing candida esophagitis is esophagoscopy. The existence of white plaques or exudates that are adhered to the mucosa and cannot be removed by water irrigation is confirmed by direct sight of the esophagus mucosa. Ulcerations or fractures in the mucosa can occasionally occur.

Histology: Histological investigation is the gold standard for diagnosing candida esophagus. During endoscopy, the esophagus mucosa is biopsied or brushed, and hematoxylin and eosin staining is performed. One of the most crucial bases for diagnosing esophageal candidiasis is the almost standard appearance of Candida yeast as pseudohyphae.

Radiological Examination: According to the degree of esophageal mucosal destruction, the disease was separated into four phases, with lumen stenosis emerging in the fourth stage. When diagnosing candida esophagitis at stage 4, barium examination is a highly helpful non-invasive method that can be utilized in place of endoscopic examination. The typical signs of esophageal stenosis are shown on a barium swallow esophagogram; a few sources refer to this condition as having a "foamy appearance" or a "feather appearance."

1.7 Treatment

Treatment with systemic antifungals is always necessary. An endoscopic examination should be performed after a diagnostic trial of antifungal medication (strong recommendation; high-quality evidence).

Oral Fluconazole, 200 mg - 400 mg (3-6 mg/kg) daily, for 14-21 days is recommended (strong recommendation; high-quality evidence). For patients who cannot tolerate oral therapy, intravenous fluconazole, 400 mg (6 mg/kg) daily, OR an Echinocandin (micafungin, 150 mg daily, caspofungin, 70 mg loading dose, then 50 mg daily, or anidulafungin, 200 mg daily) is recommended (strong recommendation; high-quality evidence).

A less preferred alternative for those who cannot tolerate oral therapy is Amphotericin B deoxycholate, 0.3 mg/kg - 0.7 mg/kg daily (strong recommendation; moderate-quality evidence).

Consider de-escalating to oral therapy with Fluconazole 200 mg - 400 mg (3 mg/kg - 6 mg/kg) daily once the patient is able to tolerate oral intake (strong recommendation; moderate-quality evidence).

For fluconazole-refractory disease, itraconazole solution, 200 mg daily, OR voriconazole, 200 mg (3 mg/kg) twice daily either intravenous or oral, for 14-21 days is recommended (strong recommendation; high-quality evidence).

Alternatives for fluconazole-refractory disease include an echinocandin (micafungin: 150 mg daily; caspofungin: 70 mg loading dose, then 50 mg daily; or anidulafungin: 200 mg daily) for 14–21 days, OR Amphotericin B deoxycholate, 0.3 mg/kg - 0.7 mg/kg daily, for 21 days (strong recommendation; high quality evidence).

Posaconazole suspension, 400 mg twice daily, or extended-release tablets, 300 mg once daily, could be considered for fluconazole-refractory disease (weak recommendation; low quality evidence). For patients who have recurrent esophagitis, chronic suppressive therapy with fluconazole, 100 mg - 200 mg 3 times weekly, is recommended (strong recommendation; high-quality evidence) [5-10].

2. Case Study

A male patient of 86 years visited Prathima hospital. He complained that he has pain in left hip since yesterday and a history of trauma i.e., history of accidental slip and fall and black stools and black vomiting of 3 episodes, fever 15 days back, cough with mucus sputum for 5 days. His past history shows that he has gone through DHS (Dynamic hip screw) to left ITH 10 years back and involuntary movements of both upper limbs. The patient is an occasional alcoholic. On examination the patient is conscious and coherent with BP 120/70 mm of hg and PR 106 b/m and GRB's 199 mg/dl. The provisional diagnosis was made to be Gastritis upper Gastro intestinal bleed Melena decreased evaluation.

The patient was immediately given normal saline 500 ml and DNS 500 ml with nebulization budesonide and mucomix along with Inj Piptaz 4 gm IV twice daily, Inj Zofen 4 mg IV thrice daily, Inj Pan 8 cc/hr, Syp Ascoryl 10 ml PO thrice daily, Metroglol 100 ml IV thrice daily. Inj Fluconazole 200 mg IV twice daily, Syp Sucralofate 15 ml thrice daily, Tab Taxim 200 mg. Feeds were given with head end elevation. The patient was advised to undergo laboratory tests such as CBP, LFT, Urine analysis, USG abd, HRCT, Upper GI endoscopy, 2D Echo, Culture and sensitivity test, CRP.

USG abd: bilateral grade 1 renal parenchymal changes.

UGI: esophageal candidiasis, mucosal erosions.

X-ray: proximal femoral tubule with multiple screws displaced.

On day 2 the patient was conscious and coherent with vitals were 120/70 mm/hg, PR 82 b/m. the patient complained fever

spike Inj PCM 1 gm IV stat was given and stools pass with I/O 1500/900 ml and oral fluids were given and upper GI endoscopy was advised and the report shows Grade B Esophagitis, small hiatus hernia, Esophageal Candidiasis, Esophageal ulcers, infective (shown in FIG. 1-3) and 2D Echo is normal. The bilirubin conjugate level is slightly elevated being 0.5 mg/dl and total protein being 6 mg/dl (slightly elevated). Hematology reports show erythropenia showing microcytic hypochromic picture with ovalocytes.



FIG. 1

FIG. 2

FIG. 3

On day 3 the patient was conscious and coherent with vitals 140/70 mm/hg, PR 98 b/m GRB's 172 mg/dl. Patient complained fever spike since yesterday and hiccups with cough and stools passed(black). Tab Cardivas 3.125 mg PO BD, Inj. Lasix 20 mg IV stat, Tab Clopitab 75 mg PO OD. The TLC count decreased from 20k to 19k. Culture sensitivity test was advised and the report indicates growth of Micrococci isolated after 2nd day of aerobic incubation. HRCT test reports show consolidation involving anterior and posterior segments of right upper limb.

F/S/O infective etiology, fibrotic bands involving bilateral apical segments bilateral mild pleural effusion. The CRP value is 270, INR is 1.40, PT is 18.8, urine analysis reports show presence of protein and puss cells (4-6) and UGI: esophageal candidiasis, mucosal erosions.

On day 4 the patient was conscious and coherent with vitals BP 130/70 mmHg and PR 84b/m. the chief complaints include loose stools(loose) with no melena (2 times) with GRB's 136 mg/dl. Tab Sporlac DS PO 2 tab were added along with Inj Doxy 100 mg IV BD. Decrease in Sr Protein level was noted to 3.40 L.

On day 5 the patient was conscious, coherent with vitals BP 140/70 mmHg and PR 90 b/m and GRB's 229mg/dl. Complaints include loose stools (4-5 episodes). Tab V Bact PO OD and Tab clopitab 75/150 mg PO BD were added. Stop Metrogyl, Sporlac and Lasix. The TLC count elevated from to 19k-21k-24k.

On day 6 the patient was conscious, coherent and cooperative with vitals BP 160/80 mmHg, PR 88b/m, GRB's 143 mg/dl. Complaints include loose stools diffuse edema. Add Tab Redotol PO/TID, Tab Rifagut 550mg PO BD, Tab Chymorol forte PO TID. TLC is 19-21-24-25k, SrCl⁺ is around 96 L, SrCr 0.60 L, hematology showing erythropenia (9.9-9.8-8.1-8.0).

On day 7 the patient was conscious and coherent with vitals BP 100/70 mmHg, PR 99 b/min, GRB's 171 mg/dl. Complaints include dryness of mouth. TLC count decreased to 11.5.

On day 8 the patient was conscious, coherent and cooperative with BP 150/70 mmHg, PR 86 bpm, GRB's 125 mg/dl. No fresh complaints were noted, stools passed (1 episode). Stop Syp Ascoryl and Cap Redotol, change in dose Tab. Fluconazole 150 mg BD 7days.

Plan for discharge and the discharge medication include T. Amoxioclav 625 mg PO OD, T. Tab Fluconazole 150 mg PO BD, T. Pan 40 mg PO OD, T. Clopitab A 75/150 PO OD, T Cardivas 3.125 mg PO BD, Syp Sucralfate O 15 ml PO TID, T. Chymorol forte PO/TID, with regular physiotherapy and ensure protein powder 2 scoops in glass water. The patient was advised to use air bed.

3. Conclusion

Clinical gastroenterology is becoming progressively concerned with Candida esophagitis. Candida infections in this area of the gastrointestinal system are frequently discovered as a result of the extensive use of corticosteroids, immunosuppressive medications, and cancer treatment, as well as the regular use of endoscopy to assess esophageal symptoms. Although gastrointestinal bleeding may occasionally be the only presenting symptom, odynophagia and dysphagia are the two main clinical characteristics of Candida esophagitis. Improving medical disorders that can lead to immunosuppression is one method of reducing the risk of esophageal candidiasis because candida is a typical oral flora that grows in impaired states of health. The risk of esophageal candidiasis can also be reduced by reducing the use of systemic steroids, antibiotics, and inhaled steroids. Patients with recurrent infections may require prophylactic fluconazole.

REFERENCES

1. Rosołowski M, Kierzkiewicz M. Etiology, diagnosis and treatment of infectious esophagitis. *Prz Gastroenterol*. 2013;8(6):333-7.
2. Chocarro Martínez A, Galindo Tobal F, Ruiz-Irastorza G, et al. Risk factors for esophageal candidiasis. *Eur J Clin Microbiol Infect Dis*. 2000;19(2):96-100.
3. Robertson KD, Nagra N, Mehta D. Esophageal candidiasis. Treasure Island (FL): StatPearls Publishing; 2025.
4. Mohamed AA, Lu XL, Mounmin FA. Diagnosis and Treatment of Esophageal Candidiasis: Current Updates. *Can J Gastroenterol Hepatol*. 2019;2019:3585136.
5. Pappas PG, Kauffman CA, Andes DR, et al. Clinical Practice Guideline for the Management of Candidiasis: 2016 Update by the Infectious Diseases Society of America. *Clin Infect Dis*. 2016;62(4):e1-50.
6. Cameron BA, Xue AZ, Kiran A, et al. Esophageal Candidiasis Is Strongly Associated with Treatment Response to Topical Steroids in Eosinophilic Esophagitis and Could Be a Marker of Adherence. *Gastro Hep Adv*. 2024;3(5):612-4.
7. Mohamed AA, Lu XL, Mounmin FA. Diagnosis and Treatment of Esophageal Candidiasis: Current Updates. *Can J Gastroenterol Hepatol*. 2019;2019:3585136.
8. Abdelfattah AH, Mahgoub AM. Severe Esophageal Stricture Caused by Esophageal Candidiasis in a Non-HIV Patient. *Cureus*. 2023;15(10):e46641.
9. Chen YH, Jao TM, Shiue YL, et al. Prevalence and risk factors for Candida esophagitis among human immunodeficiency virus-negative individuals. *World J Clin Cases*. 2022;10(30):10896-905.

10. Long-Term Trends in Esophageal Candidiasis Prevalence and Associated Risk Factors with or without HIV Infection: Lessons from an Endoscopic Study of 80,219 Patients Yuta Takahashi, Naoyoshi Nagata, Takuro Shimbo, Takeshi Nishijima , Koji Watanabe, Tomonori Aoki, Katsunori Sekine , Hidetaka Okubo 1, Kazuhiro Watanabe, Toshiyuki Sakurai , Chizu Yokoi , Masao Kobayakawa , Hirohisa Yazaki, Katsuji Teruya , Hiroyuki Gatanaga , Yoshimi Kikuchi , Sohtaro Mine , Toru Igari , Yuko Takahashi , Akio Mimori , Shinichi Oka , Junichi Akiyama , Naomi Uemura.