

PATIENT WITH NEWLY DIAGNOSED HIV INFECTION IN AIDS STAGE WHO DEVELOPED OESOPHAGEAL CANDIDIASIS AND MILIARY TUBERCULOSIS: A CASE REPORT

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This clinical case is about a 50-year old woman, a refugee from Ukraine, without known epidemiological risk factors, with newly diagnosed human immunodeficiency virus-1 (HIV-1) infection in acquired immune deficiency syndrome (AIDS) stage. Oesophageal candidiasis as an indicator disease of HIV in this case was the first diagnosis that promoted further investigation, revealing both primary HIV infection and sequentially other opportunistic infections — cytomegalovirus, Epstein Barr virus, and miliary tuberculosis. Oesophageal candidiasis was visualised by the oesophagogastroduodenoscopy method, which was initially performed due to detected anaemia to rule out bleeding from the gastrointestinal tract. Deep immunosuppression was provided to this patient and this led to the development of miliary tuberculosis, worsening the prognosis. The patient was prescribed treatment for several opportunistic infections, also anti tuberculosis treatment, as well as combined antiretroviral treatment, which stabilised the situation. In this case our patient developed odynophagia and dysphagia, common complications of oesophageal candidiasis, so she had to receive nutrition through a nasogastric tube.

Keywords: Human immunodeficiency virus-1, candida infection, anaemia, EGDS.

INTRODUCTION

It is known that oesophageal candidiasis in human immunodeficiency virus (HIV) positive patients can be the first manifestation of the acquired immune deficiency syndrome (AIDS). The aim of this clinical case was to describe a case that when oesophageal candidiasis was detected and further additional examination was performed, both the primary HIV infection and sequentially other opportunistic infections — cytomegalovirus (CMV), Epstein Barr virus (EBV) and miliary tuberculosis were detected. Oesophageal candidiasis was visualised with oesophagogastroduodenoscopy (EGDS) method due to revealed anaemia to rule out causes of bleeding from the gastrointestinal tract. It is incredible how often HIV patients develop gastroenterological manifestations (especially often of fungal aetiology), which sometimes help to get to the correct diagnosis.

Mycobacterium tuberculosis DNA (MTB DNA) was found in patient's bronchial lavage, and later miliary tuberculosis was also diagnosed, which is known to worsen the progress of HIV infection. The patient was given antifungal, antibacterial, antiviral, antiretroviral (anti HIV), and anti tuberculosis treatment.

Further the patient developed odynophagia and dysphagia, which are the most common manifestations of oesophageal candidiasis, so further feeding was performed through a nasogastric tube.

CASE HISTORY

The patient entered the admissions department with complaints of increasing weakness during the month, which had become more pronounced in the last two weeks. Patient had

difficulties in performing daily activities and mostly slept during the day. Also, she had a fever for a week, used paracetamol, but it helped only for a while, and then the temperature reoccurred. In the admissions department patient had temperature 39.5° C, SARS-Cov-19, A and B influenza and RSV were not found.

An X-ray of the chest was performed, where no infiltrative changes were found in the lungs, but there was a small round shadow on the parahilar left side 1.2 cm in diameter. The USG of the abdomen found cloudy contents in the bladder, a small amount of free fluid in the ileocecal region. Next, the patient was hospitalised in the Gastroenterology Department for further examination and treatment. Due to the parahilar visible shadowing on the left side of the X-ray, a chest CT was performed, where foci or infiltrates in the lungs were not differentiated, and the parahilar visible shadowing on the left side was shown to be a formation on the skin — a wart that simulated the pathology on the X-ray.

A CT scan of the abdomen with intravenous bolus contrast revealed fluid in the pleural spaces bilaterally, lying on the back — 3.7 cm on the right side, and 4 cm on the left side (Fig. 1), also ascites and specific retroperitoneal lymphadenopathy. Therefore, to rule out suspicions of lymphoproliferative disease (lymphoma), it was recommended to take a lymph node biopsy from the lymph nodes in the abdominal cavity. Of note, lymphoma and Kaposi's sarcoma are the most common HIV related neoplastic lesions found in the oesophagus (Zaidi and Cervia, 2002; Olum *et al.*, 2020). It was decided to perform a lymph node biopsy, however, the biopsy was not taken due to the poor general condition of the patient.

Blood tests showed haemoglobin level of 8.4 g/dl, moderate normochromic normocytic anaemia. Due to anaemia of unspecified origin, EGDS was performed to rule out possible bleeding site from the gastrointestinal tract, where incidentally oesophageal candidiasis (Fig. 2) and erythematous gastropathy were detected. Treatment with fluconazole was started and 3 erythrocyte masses were transfused.

Since the patient was diagnosed with oesophageal candidiasis, which is one of the opportunistic infections, testing for HIV test was also performed, which showed a positive result for HIV-1 infection for the first time in the patient's life, right at the AIDS stage (CIII) with very low clusters of differentiation 4 (CD4) and clusters of differentiation 8 (CD8) cell count. HIV viral load was 1.28 E⁶ copies/ml. Immunophenotyping was performed and the immunoregulatory index CD4/CD8 was 0.057 (Table 1). Antiretroviral therapy was added relatively early because of a high risk of developing the immune reconstitution syndrome immune reconstitution syndrome (IRIS). Also, treatment with antibiotics was started.

Examinations were performed for opportunistic diseases — CMV DNA and EBV DNA, which were found positive and antiviral treatment was initiated.

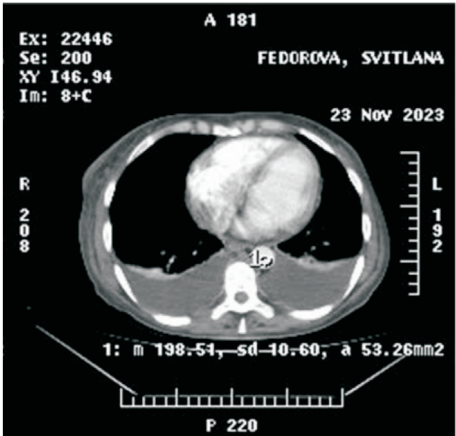


Fig. 1. Hydrothorax shown on abdominal CT scan (sindx).

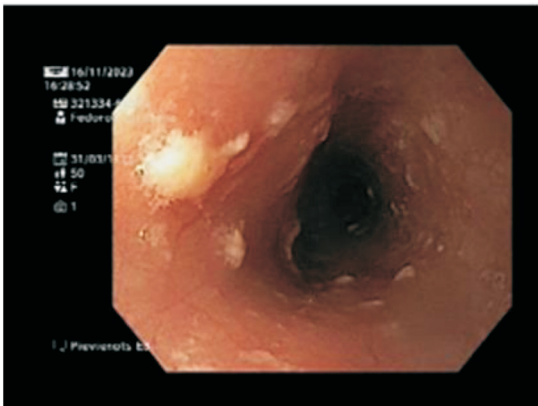


Fig. 2. Oesophageal candidiasis found on EGDS.

Table 1. Lymphocyte immunophenotypes

Shift	Investigation	Result	Measuring unit	Reference range
Lymphocyte immunophenotypes, FACSlyric				
<	T-lymphocytes (CD3+)	51.64	%	(60.00 – 85.00)
<<	T-lymphocytes (absol.)	0.196	×10 ⁹ /l	(0.700 – 1.900)
<<	T-helpers-inductors (CD3+CD4+)	2.52	%	(35.00 – 55.00)
<<	T-helpers-inductors (absol.)	0.010	×10 ⁹ /l	(0.400 – 1.300)
>>	T-supresors/cytotoxic (CD3+CD8+)	44.34	%	(20.00 – 32.00)
<	T-supresors/cytotoxic (absol.)	0.168	×10 ⁹ /l	(0.200 – 0.700)
<	Immunoregulatory index (CD4+/CD8+)	0.057		(1.20 – 2.50)

Hepatitis B surface antigen (HBsAg), hepatitis C antibodies, *Pneumocystis jirovecii* (carinii) DNA and antibodies against Treponema Pallidum immunoglobulin G + immunoglobulin M and DNA of bacterial pneumonia agents were negative.

Next, due to the appearance of catarrhal symptoms and the decrease in saturation when SpO₂ showed 87%, X-ray of the lungs was repeated, where bilateral hydrothorax (sin > dx) with compressive infiltrations was detected. CTA was

also performed in suspicion of pulmonary artery thromboly (PATE), but it showed no data on PATE. However, there were also diffuse small nodular changes in the lungs that were not visible on the previous CT of lungs; these changes appeared to be likely infectious changes, suspected of fungal infection, miliary tuberculosis or CMV pneumonia. Therefore, further bronchoscopy was performed, bronchial lavage was taken for *M. tuberculosis* and MTB DNA was found positive. Next, the patient was transferred to the Centre of Tuberculosis and Lung Diseases for further treatment with anti tuberculosis therapy. It should be noted that the patient developed intolerance to isoniazid during tuberculosis treatment, as the level of bilirubin in the blood increased from time to time, so there were made changes in therapy. The *M. tuberculosis* complex was also found in blood, faeces, and urine (miliary tuberculosis).

Despite the applied therapy, the patient's general condition did not improve. Subfebrile-febrile body temperature constantly alternated with normal body temperature.

Later, the patient developed Covid-19 infection and eye pain — suspicion of CMV retinitis or meningitis was not confirmed.

The patient received a protein diet, but ate less than 25%, she was unable to eat normally through the mouth because she developed dysphagia and odynophagia as common oesophageal candidiasis complications. This led to malnutrition and hypoalbuminemia, so the feeding through nasogastric tube was started, and the patient also started receiving medical supplementary nutrition.

DISCUSSION

The clinical presentation of primary HIV-1 infection can range from asymptomatic to severe. Symptoms usually occur within 2–4 weeks and may include fatigue, sudden onset of fever, sore throat, heartburn, diarrhea, rash, night sweats, weight loss, mouth ulcers, genital ulcers, lymphadenopathy, and meningitis (Mindel and Tenant-Flowers, 2001; Mohamed *et al.*, 2019; Dockrell *et al.*, 2019; Symptoms of HIV, 2022).

In this clinical case the patient had sudden onset of fever and increasing weakness. Here also normochromic normocytic anaemia was present, her vitamin B12 level was decreased (ferritin and folic acid levels were increased). Vitamin B12 deficiency can be present because of gastric pathology caused by infections that affect the gastric mucosa in HIV-infected patients (Volberding *et al.*, 2004). Anaemia is the most common (it affects 60–80% of patients) haematologic abnormality associated with HIV infection, occurring in late stage HIV disease (Meidani *et al.*, 2012). Due to anaemia we performed EGDS and found oesophageal candidiasis and erythematous gastropathy. Candidiasis in oesophagus is called oesophageal candidiasis or *Candida* oesophagitis (*candida* infections of the mouth, throat, and oesophagus, 2021). It should be noted that oeso-

phageal candidiasis is one of the diagnostic criteria for AIDS (Diagnostic criteria for AIDS-defining conditions, 1992; Pena *et al.*, 2018; Vaezi *et al.*, 2018). Also, oesophageal candidiasis in HIV-positive patients may be the first manifestation of AIDS (Vazquez, 2010). *Candida* oesophagitis usually causes dysphagia (difficulty swallowing, one of the commonest symptoms of oesophagitis), odynophagia (pain when swallowing), and retrosternal pain (Vazquez, 2010; Ching *et al.*, 2021). Dysphagia can develop due to oesophageal stricture (a rare complication), which may lead to food bolus obstruction (Ching *et al.*, 2021). In addition to strictures also Barrett oesophagus and adenocarcinoma of oesophagus are serious gastrointestinal complications of oesophagitis (Ching *et al.*, 2021; Kingsley-Godwin and Kingsley-Godwin, 2021). There are also known cases of oesophagotracheal, tracheobronchial and aortoesophageal fistulas, haemorrhage and perforation of oesophagus (Nishimura *et al.*, 2013; Vaezi *et al.*, 2018; Mohamed *et al.*, 2019; Nishimura *et al.*, 2013; Khan *et al.*, 2022). Oesophageal candidiasis usually presents as superficial oesophagitis, however, transmural necrotising candidiasis cases also may be present (Mohamed *et al.*, 2019).

In general population, the frequency of oesophageal infections is not high, it is only 0.32–5.2% and indicates immunodeficiency or underlying disease (Manfredi *et al.*, 2007; Mohamed *et al.*, 2019).

In all immunocompromised patients, the most commonly identified oesophageal pathogens are *Candida* spp., herpes simplex, and cytomegalovirus (Manfredi *et al.*, 2007; Olum *et al.*, 2020). Bacterial aetiologies cannot be excluded (Manfredi *et al.*, 2007). It is *Candida albicans* that is the main opportunistic yeast pathogen (Vazquez, 2010). From non-*albicans Candida* species more common are *Candida glabrata*, *Candida parapsilosis*, *Candida tropicalis*, *Candida dubliniensis* and *Candida krusei* (Zaidi and Cervia, 2002; Vazquez, 2010). Such non *Candida* fungi as *Histoplasma capsulatum*, *Exophiala jeanselmei* and *Penicillium chrysogenum* rarely colonise the oesophagus (Zaidi and Cervia, 2002; Vaezi *et al.*, 2018). As very rare causes of oesophagitis can be protozoa — *Cryptosporidium parvum*, *Pneumocystis carinii*, and *Leishmania* (Zaidi and Cervia, 2002).

In the gastrointestinal tract, the oropharynx is the most susceptible to candida infection, followed by the oesophagus; this means that 85–90% of HIV-infected patients will develop oropharyngeal candidiasis, but only 10–15% will develop oesophageal candidiasis (Mohamed *et al.*, 2019).

If defence mechanisms of the immune system are disturbed, it allows *Candida* fungi to proliferate in the lining of the oesophagus and form adhesive yellow-white plaques. These plaques can be seen on oesophagogastroduodenoscopy and cannot be removed by water irrigation. Plaques can be localised in the upper, middle or distal third of the oesophagus or diffusely throughout the oesophagus (Mohamed *et al.*, 2019). Possible predisposing factors for oesophageal

candidiasis should be reduced — chemotherapy agents, corticosteroids, and antimicrobial agents (Vazquez, 2010).

CD4 positive cells play an important role in protection against mucosal fungal infection. Immunological changes occur during or immediately after an acute episode of primary HIV infection, with an inverted ratio of CD4 positive to CD8 positive cells, often associated with an increased number of CD8 positive cells, which was also demonstrated in this clinical case (Podzamczar, 1988). It should be noted that the immune response of the digestive tract to *Candida* spp. invasion is highly dependent directly on Th17 helpers (Dockrell *et al.*, 2019).

Differential diagnoses of oesophageal candidiasis include viral oesophagitis (of cytomegalovirus or herpes simplex virus aetiology), idiopathic HIV ulcers, and gastroesophageal reflux disease, as well as eosinophilic oesophagitis (Vazquez, 2010; Kaufmann and Vazquez, 2024). Our patient was CMV positive, and it is known that CMV induced oesophagitis (with idiopathic oesophageal ulceration) is the most common cause of oesophageal ulceration in HIV-infected patients (idiopathic oesophageal ulcer usually occurs in severe immunodeficiency). In the oesophagus CMV may coexist with candida and/or HSV infections, however, the presence of CMV alone does not equate with clinical CMV oesophagitis (Zaidi and Cervia, 2002).

All patients with oesophageal candidiasis should be screened for HIV, hepatitis B and C viruses, and syphilis infection (Takahashi *et al.*, 2015).

As it was already mentioned, the patient received antibacterial, antifungal, antiviral, antiretroviral, and anti tuberculosis treatment. In our case treatment with the antibiotics bisepol at 160 mg/800 mg once daily perorally and 500 mg clarythromycin twice daily perorally was started.

First-line treatment of *Candida oesophagitis* is based on receiving fluconazole perorally at a dose of 200–400 mg (3–6 mg/kg) for 14–21 days daily. Intravenous fluconazole is reserved for patients who cannot tolerate oral treatment (Vaezi *et al.*, 2018). In our case antifungal therapy was applied with fluconazole of 150 mg twice daily perorally. It should be noted that empirical antifungal therapy is recommended before endoscopic examination. Oesophageal candidiasis usually responds well to antifungal therapy and treatment of oesophageal candidiasis must be systemic and not topical. The most commonly used drugs in treatment of oesophageal candidiasis are systemic antifungal agents and fluconazole is the first choice (Zaidi and Cervia, 2002; Vazquez, 2010; Vaezi *et al.*, 2018; Mohamed *et al.*, 2019; Zeng *et al.*, 2021). If there is resistance to fluconazole, then itraconazole can be used as an alternative (Vaezi *et al.*, 2018). It is important to know that fluconazole must be administered with caution to patients with liver dysfunction. If during fluconazole therapy abnormal liver tests are present, then the patient should be monitored for the development of more serious hepatic injury. However, hepatotoxicity of fluconazole is usually reversible on discontinuation of

therapy. Also, anaphylaxis of fluconazole has been reported (but in very rare cases). As from cardiac complications, fluconazole may cause prolonged QT interval dysfunction (Fluconazole description. Pfizer, 2022).

Next, treatment with antiviral valganciclovir of 450 mg once daily perorally was started (valganciclovir was given against CMV and EBV).

Antiretroviral therapy has significantly reduced the prevalence of oropharyngeal and oesophageal candidiasis in HIV infected patients (Vazquez, 2010). Our patient received combination of first line drugs for treatment of HIV infection — 600 mg efavirenz, 200 mg emtricitabine, and 245 mg tenofovir perorally once daily according to international guidelines. This triple therapy was first approved by FDA in 2006, but there are also other drug regimens available approved later than in 2006 (Cutrel and Bedimo, 2016). Precautions are required for patients with severe psychiatric disease (or suicidality) and for patients with new or worsening renal impairment (Cutrel and Bedimo, 2016). In our case, based on a very low CD4 positive cell count, antiretroviral therapy was added relatively early because of a high risk of developing immune reconstitution syndrome IRIS.

For tuberculosis treatment the patient received 500 mg isoniazid, 1000 mg ethambutol and 150 mg rifabutin once daily intravenously. Because there was a side effect of bilirubin levels rise in blood, therapy for tuberculosis with isoniazid was adjusted (isoniazid was taken out from therapy). It should be noted that interactions between antiretroviral and anti tuberculosis drugs require attention. Adverse side effects of anti tuberculosis drugs (especially drug-induced liver injury) are more common in HIV infected patients, so in case of HIV-tuberculosis coinfection drug interactions and adverse drug reactions must be monitored (Qiaoli *et al.*, 2022).

CONCLUSION

We conclude that oesophageal candidiasis in HIV infected patients may be the first manifestation of HIV infection. In this case, oesophageal candidiasis was discovered as an incidental finding during EGDS, which was performed to find possible causes of anaemia. Next, we found that the patient was also positive for HIV-1 infection. Also, due to immunosuppression, various opportunistic infections such as miliary tuberculosis were found, which can complicate the further gait of HIV infection.

Even by starting treatment at late stage of HIV, simultaneously providing a combination of drugs necessary for the treatment of several diseases, it is possible to save the patient from an apparently hopeless situation. Also, possible predisposing factors for oesophageal candidiasis should be reduced like chemotherapy, corticosteroids, and antimicrobial agents.

It should be noted that in this case the patient developed common complications of oesophageal candidiasis —

odynophagia and dysphagia, so she had to receive nutrition through a nasogastric tube.

CONFLICT OF INTEREST

There are no conflicts of interest.

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ETHICS

Ethical approval is not required at our institution to publish an anonymous case report.

Written informed consent was obtained from the patient for publication of this case report.

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PACIENTE AR PIRMREIZĒJI DIAGNOSTICĒTU HIV INFEKCIJU AIDS STADIJĀ, KURAI ATTĪSTĪJĀS BARĪBAS VADA KANDIDOZE UN MILIĀRĀ TUBERKULOZE: KLĪNISKAIS GADĪJUMS

Šis klīniskais gadījums ir par 50 gadus vecu sievieti, bēgļi no Ukrainas bez zināmiem epidemioloģiskiem riska faktoriem, ar pirmreizēji diagnosticētu HIV-1 infekciju AIDS stadijā. Barības vada kandidoze kā HIV indikatorslimība šajā gadījumā bija pirmā diagnoze, kas veicināja turpmāku izmeklējumu veikšanu, atklājot gan primāro HIV infekciju, gan arī secīgi citas oportūnistiskas infekcijas — citomegalovīrusu, Epšteina-Barra vīrusu un miliāro tuberkulozi. Barības vada kandidoze tika vizualizēta ar ezofagogastroduodenoskopijas metodi, kas tika veikta anēmijas dēļ, lai izslēgtu asiņošanu no gastrointestinālā trakta. Pacientei tika pierādīta dziļa imūnsupresija, kas veicināja miliāras tuberkulozes pievienošanos, pasliktinot prognozi. Pacientei tika nozīmēta vairāku oportūnistisku infekciju, prettuberkulozes un kombinēta antiretrovirālā terapija, kas stabilizēja situāciju. Šajā gadījumā pacientei attīstījās odinofāģija un disfāģija, kas ir bieži sastopamas barības vada kandidozes komplikācijas, tāpēc viņai tika uzsākta barošana caur nazogastrālo zondi.