CASE REPORT

Opportunistic infection mimicking COVID-19 in TB/HIV-coinfected patient: a case study

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Abstract

Coronavirus disease (COVID-19) and pulmonary tuberculosis (TB) can both emerge with similar clinical presentations. Despite their different nature of progressivity, both diseases may show similarity in an acute manner, especially in the presence of secondary infection, e.g., TB. This is particularly substantial in TB/HIV-coinfection, with a myriad of possible secondary infections and different clinical presentations. These includes diagnostically challenging *Pneumocystis carinii* pneumonia (PCP), one of the most common opportunistic infections in TB/HIV patients.

Here, we reported a case of an opportunistic infection mimicking COVID-19 in a TB/HIV-infected patient. A 22-year-old female presented to our hospital with decreased consciousness, shortness of breath, and cough. Lab results showed lymphocytopenia and thrombocytopenia as well as elevated C-reactive protein, D-dimer, and ferritin, which is classically suggestive of COVID-19 infection. She was diagnosed with probable COVID-19, but two subsequent consecutive RT-PCR tests for SARS-CoV-2 were negative. Both sputum GeneXpert MTB rapid molecular test and HIV immunoserology rapid test were positive, and chest X-ray showed bilateral miliary infiltrates. Therefore, the patient was diagnosed as TB/HIV with secondary opportunistic infection.

As both COVID-19 and TB/HIV with opportunistic infections can present similarly debilitating risk for the patient, we highlight the importance of accurate history-taking and rapid RT-PCR test for SARS-CoV-2 to ensure accurate and timely diagnosis.

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Key words: COVID-19, TB/ HIV, opportunistic infection.

Introduction

Coronavirus disease (COVID-19) is an infection caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Its spectrum ranges from asymptomatic to severe/critically ill condition [1, 2]. Although the most common presentation is pneumonia, there is no distinct feature that is reliable enough to differentiate COVID-19 from other causes of pneumonia, particularly pulmonary tuberculosis. Therefore, COVID-19 is considered as differential diagnosis in most patients presenting with respiratory symptoms [3].

Tuberculosis (TB) is a major public health threat, competing with human immunodeficiency virus (HIV) as the leading cause of death due to infectious diseases around the world [4]. Indonesia is the third country with the greatest

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number of TB cases globally and with high burden of TB/HIV. Opportunistic infection in TB/HIV patients is a severe infectious disease with high mortality [5].

Here, we reported a case of an opportunistic infection mimicking COVID-19 in a TB/HIV patient, in the context of decreasing prevalence of COVID-19.

Case report

A 22-year-old female presented to our hospital with gradual decrease of consciousness within 3 days. She also complaint of shortness of breath since 3 days and cough since 1 week without sputum production. The patient denied history of chronic cough, intermittent fever, and night sweating. Vital signs showed Glasgow Coma Scale (GCS) of E4M5V5, blood pressure of 100/65 mmHg, pulse rate of 120 ×/minute, respiratory rate of 28 ×/minute, and peripheral oxygen saturation of 98% on 8 liter/minute using non-rebreathing mask.

Laboratory examination revealed hemoglobin level of 9.7 g/dl, WBC: 3880/µl with lymphocytopenia (4.4%), platelet count: 98.000/µl, fibrinogen: 212.3 mg/dl (normal range, 154.3-397.9), ferritin: 24187 ng/ml (normal range, 13-150), D-dimer: 25.86 mg/l (normal range, ≤ 0.5), SGOT: 104 U/l, SGPT: 23 U/l, albumin: 2.72 g/dl, RBS: 124 mg/dl, urea: 20 mg/dl, creatinine: 0.64 mg/dl, C-reactive protein (CRP): 11.69 mg/dl, procalcitonin: 5.95 ng/ml, LDH: 1387 U/l (normal range, 135-214), and serum sodium, potassium, and chloride were 130, 3.4, and 96 mmol/l, respectively. Arterial blood gas analysis demonstrated pH of 7.51, PCO_2 of 23.2, PO_2 of 264.1, serum bicarbonate of 18.6, base excess of –4.7, and oxygen saturation of 98.1%. Immuno-serology HIV rapid test was reactive. Chest X-ray displayed bilateral miliary-sized infiltrates, which improved over time (Figure 1), while no intraparenchymal pathological process in the brain was shown on head CT.

Initially, we diagnosed the patient as a probable COVID-19 case, with moderate criteria for differential diagnosis of bacterial infection, lung mycosis, and *Pneumocystis carinii* pneumonia (PCP). The patient was also diagnosed with lung TB and newly diagnosed HIV. She was placed in our COVID isolation room, and RT-PCR for SARS-CoV-2 nasopharyngeal swab test was done. Since two consecutive RT-PCR test results were negative, she was admitted to a regular ward. Sputum XPert MTB/Rif results were MTB detected very low and Rif resistance indeterminate. The patient was treated with intravenous levofloxacin 750 mg OD, fluconazole 200 mg OD, and methylprednisolone 31.25 mg OD. Subcutaneous heparin 5000 IU BD and oral cotrimoxazole 1440 mg QD were administered. In addition, the patient was treated with modified liver regiment of antituberculosis drugs using intravenous streptomycin (S) 500 mg OD, intravenous levofloxacin (L) 750 mg OD, and oral ethambutol (E) 750 mg OD. To avoid complications associated with antituberculosis treatment, antiretroviral (ARV) therapy was postponed. After 14 days, the patient condition improved significantly, as she was able to communicate and was stable without oxygen supplementation.

Discussion

Our patient presented with arrays of non-specific symptoms suggestive of both COVID-19 and pulmonary TB. As diseases with many faces, diagnostic process of both COVID-19 and TB is challenging, with both the diseases being spread by similar methods, such as airdrops and close contacts.

Moreover, COVID-19 and TB show similar laboratory findings, which is typically lymphopenia with mild thrombocytopenia. However, these findings are less frequent in TB. Typical findings of TB are normocytic anemia, leukocytosis, thrombocytosis, and elevated liver enzymes due to inflammatory process [3]. Anemia in COVID-19 is rare, but can occur as a result of underlying comorbidities rather than the course of COVID-19 itself. Other inflammatory markers, such as erythrocyte sedimentation rate (ESR), CRP, and ferritin can show increased levels in both the diseases. In short, when these findings are considered in COVID-19 diagnosis, TB should also be considered as differential diagnosis in patients presenting with anemia and elevated ESR and CRP [6-8].

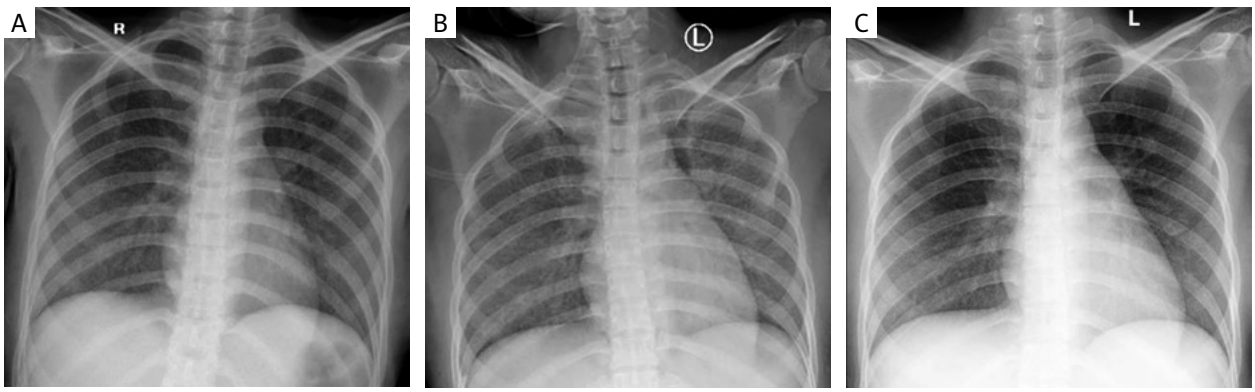

Figure 1. Progression of disease shown on chest X-ray (CXR) images. **A**) CXR taken on the day of admission showing miliary-sized infiltrates. **B**) CXR taken on day 5. **C**) CXR taken on day 14 after admission

A previous paper reported on SARS-CoV-2 infection mimicking pulmonary tuberculosis. This case shows that COVID-19 can present with an unique, atypical symptoms, and COVID-19 diagnosis should be considered first as it possess a potential risk for community and healthcare providers [9]. The World Health Organization's 2021 Global TB Report states that TB-related deaths have increased for the first time in over a decade, with much lower rate among TB-diagnosed individuals and treated using preventive therapy, as compared with 2019. This marks the impact of COVID-19 pandemic on TB, and troubling fact that COVID-19 may bring negative effect on global TB progress [10].

On the other hand, delayed diagnosis of other infections due to COVID-19 suspicion was also reported. A study presented a delayed diagnosis of rifampicin-induced pneumonitis due to a clinical suspicion to severe COVID-19-associated pneumonia, supported by consistent clinical, CXR, and thorax CT findings. As both the diseases, i.e., COVID-19 and TB, show similar findings, this paper stressed the importance of determining accurate patient history, as previous TB treatment was the key in determining the diagnosis in this case [11].

The most prevalent opportunistic infections found in HIV patients are oral candidiasis, tuberculosis, pneumonia, and PCP. Other infections include toxoplasmosis, chronic diarrhea, cytomegalovirus, meningitis TB, hepatitis C, amoebiasis, and cerebritis [5]. Tuberculosis and PCP are the most common opportunistic diseases, often serious in HIV-positive patients. Among them, PCP is the most dangerous for immunocompromised patients, particularly those infected with HIV [12].

PCP is difficult to be diagnosed correctly, and can cause acute respiratory distress or respiratory failure with poor prognosis. PCP clinically presents with shortness of breath, cough, and fever. *Pneumocystis jirovecii*, the fungus responsible for PCP, cannot be cultured by typical viral media. Golden standard for PCP diagnosis is the presence of the organism in respiratory sample and cyst using different stains, such as Grocott-Gomori methenamine silver, Giemsa, and Diff-Quik [13, 14].

Serological diagnosis, i.e., β-D glucan levels, has been established to diagnose PCP, but is not specific [15]. The problem about PCP diagnosis is that such examination is not available in all centers. Thus, in cases where such diagnostic procedures are not available, PCP is widely identified clinically by using physician's clinical judgement, increase level of serum lactate dehydrogenase (LDH), and decrease of alveolar-arterial oxygen gradient ($AaDO_2$). However, LDH is a non-specific biomarker, and elevated serum of LDH in PCP patients is thought to be due to lung inflammation and tissue injury [16].

As both COVID-19 and TB/HIV with their opportunistic infections can present similar debilitating risk for the patient, we highlight the importance of accurate history-taking and rapid RT-PCR test for SARS-CoV-2, to ensure accurate and timely diagnosis.

Disclosures

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References

1. Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet 2020; 395: 497-506.
2. Chen N, Zhou M, Dong X, Qu J, Gong F, Han Y, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. Lancet 2020; 395: 507-513.
3. Uyaroğlu OA, Başaran NÇ, Özişik L, Inkaya AC, Güven GS. COVID-19 at first glance, pulmonary tuberculosis with a glance in depth. Journal of Emergency Medicine Case Reports 2020; 12: 22-24.
4. Sulis G, Roggi A, Matteelli A, Raviglione MC. Tuberculosis: epidemiology and control. Mediterr J Hematol Infect Dis 2014; 6: e2014070. DOI: 10.4084/MJHID.2014.070.
5. García JI, Mambuque E, Nguenha D, Vilanculo F, Sacoor C, Sequera VG, et al. Mortality and risk of tuberculosis among people living with HIV in whom TB was initially ruled out. Sci Rep 2020; 10: 15442. DOI: 10.1038/s41598-020-71784-3.
6. Oliva VM, Cezário GAG, Cocato RA, Marcondes-Machado J. Pulmonary tuberculosis: hematology, serum biochemistry and the relation with the disease duration. J Venom Anim Toxins Incl Trop Dis 2008; 14: 71-81.
7. Pourbagheri-Sigaroodi A, Bashash D, Fateh F, Abolghasemi H. Laboratory findings in COVID-19 diagnosis and prognosis. Clin Chim Acta 2020; 510: 475-482.
8. Mandal SK, Chavan L. Erythrocyte sedimentation rate values in cases of active tuberculosis without HIV co-infection. J Med Sci Clin Res 2016; 4: 13156-13159.
9. Akbar H, Kahloon R, Akbar S, Kahloon A. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection mimicking as pulmonary tuberculosis in an inmate. Cureus 2020; 12: e8464. DOI: https://doi.org/10.7759/cureus.8464
10. World Health Organization. Global tuberculosis report 2021. Available at: https://www.who.int/publications/i/item/9789240037021.
11. Ata F, Hussein MS, Mismar AY, Sharma R, Bozom IA, Ibrahim ZA, Ibrahim WH. Rifampicin-induced pneumonitis mimicking severe covid-19 pneumonia infection. Am J Case Rep 2020; 21: e927586. DOI: 10.12659/AJCR.927586.
12. Asmarawati TP, Putranti A, Rachman BE, Hadi U. Opportunistic infection manifestation of HIV-AIDS patients in Airlangga university hospital Surabaya. In: IOP Conference Series: Earth and Environmental Science 2018; 125: 012061. DOI: 10.1088/1755-1315/125/1/012061.
13. Sheikholeslami MF, Sadraei J, Farnia P, Moghadam MF, Kochak HE. Co-infection of Mycobacterium tuberculosis and Pneumocystis jirovecii in the Iranian patients with human immunodeficiency virus. Jundishapur Journal of Microbiology 2015; 8: e17254. DOI: 10.5812/jjm.17254.
14. Catherinot E, Lanternier F, Bougnoux ME, Lecuit M, Couderc LJ, Lortholary O. Pneumocystis jirovecii pneumonia. Infect Dis Clin 2010; 24: 107-138.
15. Tasaka S. Recent advances in the diagnosis and management of Pneumocystis pneumonia. Tuberc Respir Dis 2020; 83: 132-140.
16. Watanabe T, Yasuoka A, Tanuma J, Yazaki H, Honda H, Tsukada K, et al. Serum (1→ 3) β-d-glucan as a noninvasive adjunct marker for the diagnosis of Pneumocystis pneumonia in patients with AIDS. Clin Infect Dis 2009; 49: 1128-1131.