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The Medicine Forum

Volume 25 Article 23

2024

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Recommended Citation

Perez, MD, Edwin; Patel, MD, Avish; Awan, MD, Taha; Bhasin, MD, Amman; and George, MD, Gautam (2024) "A Case of Reactivation Tuberculosis," *The Medicine Forum*: Vol. 25, Article 23.
DOI: https://doi.org/10.29046/TMF.025.1.022
Available at: https://jdc.jefferson.edu/tmf/vol25/iss1/23

A Case of Reactivation Tuberculosis

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INTRODUCTION

Approximately 22 percent of the world population is estimated to be infected with *Mycobacterium tuberculosis*. The largest number of cases are seen in Sub-Saharan Africa, India, and the islands of Southeast Asia, while North America and Western Europe are "low incidence" regions.[1] In the US, most cases are seen in foreign-born individuals who emigrate from high-incidence countries. Despite the low incidence in the US, prevention and control of Tuberculosis (TB) remain an important component of Public Health.[2] Here we present a case of a patient of Vietnamese origin with reactivation TB and rare manifestations of TB.

CASE PRESENTATION

Subjective

A 52-year-old man from Vietnam and a past medical history of tobacco use disorder, alcohol use disorder, chronic cough, and thyroiditis presented as a direct admission from his pulmonologist's office due to concern for active TB. The patient reported odynophagia, dysphagia, and a chronic cough that started 3-4 months prior to admission (PTA). The cough was described as mostly dry with occasional brown sputum and scant hemoptysis. He described odynophagia mostly with solid foods, leading to a transition in his diet to mostly soft foods and liquids. He reported a few fevers at night and a 46-pound weight loss since his symptoms started. He denied any exposure to TB or sick contacts.

His symptoms prompted him to see an outpatient pulmonologist. Given the concern for TB, computed tomography (CT) of the chest, interferon-γ release assay (IGRA), and sputum samples were ordered. The CT Chest was remarkable for upper lobe predominant cavitary lesions with nodules in both lungs in a bronchogenic distribution consistent with endobronchial spread rather than hematogenous spread. IGRA was positive and sputum cultures had yet to result. Overall, his findings were suspicious for TB and inpatient evaluation was recommended.

Further social history was elucidated upon meeting him. He moved to the US from Vietnam in 1991 and last visited Vietnam in 2018 for 2 months, with no other travel outside the country. He reported a 40+ year smoking history and a long-standing alcohol history of drinking 6-7 beers a day but quit both 3 months PTA once his symptoms started. He reported working as a machinist in a computer factory.

Objective

On presentation to the ED, he was afebrile, hypotensive with initial blood pressure of 90/60 mmm hg, tachycardic to the 130s, and oxygen saturation of 98% on room air. On physical exam, he was a thin, cachectic-appearing man in no respiratory distress, with no palpable cervical or axillary lymphadenopathy, and with scattered rhonchi bilaterally on auscultation of lungs. Notably, he was found to have white plaques on both sides of his tongue that did not scrape off along with poor dentition.

Lab work was significant for hyponatremia (125), leukocytosis (16), mild anemia (hgb 8), and thrombocytosis (607). Lactate was within normal limits.

A portable chest X-ray showed bilateral multifocal airspace opacities concerning for multifocal infectious process and a right upper lobe (RUL) cavitary lesion (**figure 1**). A CT angiogram of the chest was also ordered due to concern for a pulmonary embolism (PE) given the patient's reported history of hemoptysis. The CT angiogram was negative for PE but did show cavitary lesions in the RUL similar to his outpatient imaging (**figures 2 and 3**). Specifically, it was read as centrilobular/tree-in-bud nodularity with a thick-walled cavitary lesion in the right lung apex, measuring 5.9 x 5.9 x 5.6cm, consistent with reactivation TB, and mediastinal and right hilar adenopathy. CT abdomen & pelvis showed abdominopelvic ascites with peritoneal enhancement, concerning for peritonitis from TB.

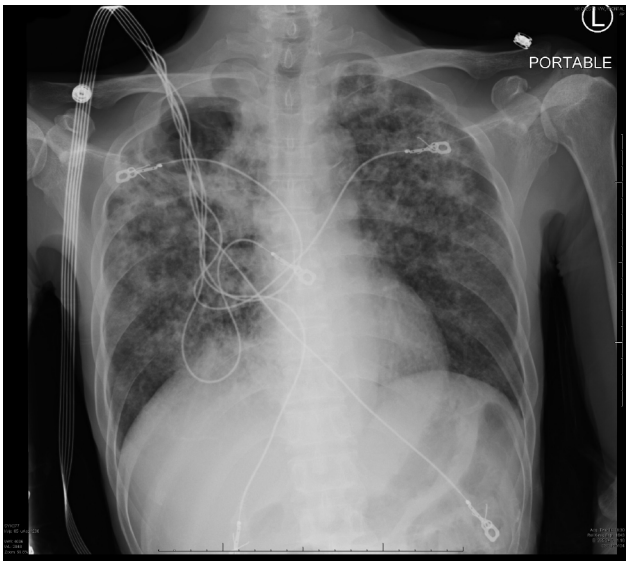

Figure 1

Investigations, management, and hospital course

The patient was initiated on broad-spectrum antibiotics and IV fluids. Given the patient was a direct admission for potential TB with a physical exam significant for leukoplakia, there was concern for immunosuppression. As part of an immunosuppression workup, HIV, EBV, CMV, HSV, Syphilis, and Hepatitis C labs were all collected and resulted as negative. Hepatitis B serologies were consistent with previous but resolved infection.

Infectious disease was consulted. Sputum cultures for acid-fast bacilli (AFB), mycobacterium TB (MTB) PCR, and rifampin resistance were ordered. All three sputum cultures were positive, MTB PCR was reactive and rifampin resistance was negative. The patient was subsequently started on RIPE therapy and micafungin (due to concern for esophageal candidiasis in the setting of immunosuppression and leukoplakia).

Gastroenterology was consulted and noted that the white plaque of the tongue and odynophagia might have been related to chronic fungal infection versus oral tuberculosis or a manifestation of undiagnosed malignancy. An endoscopy was deferred due to the patient being actively treated for TB.

A few days into his hospital stay, the patient had continued odynophagia and dysphagia that radiated to his jaw. An X-ray of the mandible was obtained to rule out osteomyelitis from TB, but it only showed an edentulous mandible. His course was further complicated by new onset heart failure with reduced

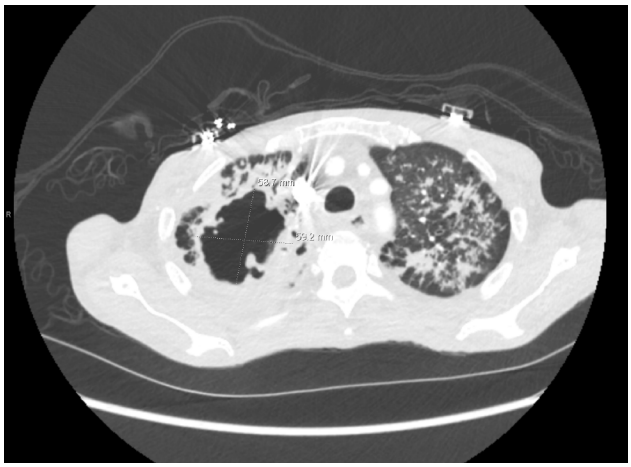

Figure 2

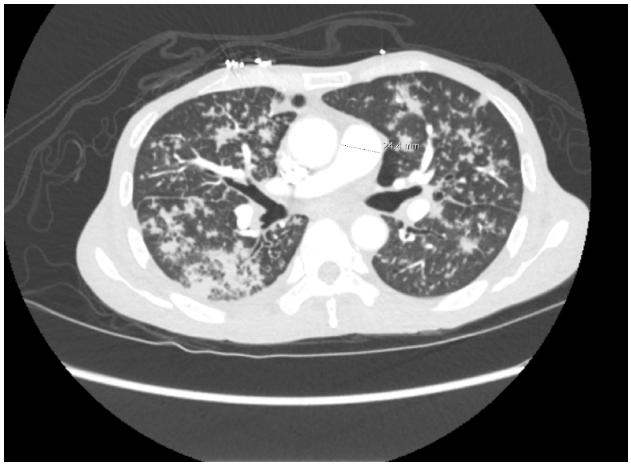

Figure 3

ejection fraction for which cardiology was consulted. The echocardiogram was consistent with reverse takotsubo cardiomyopathy.

Ultimately, he completed 2 weeks of inpatient RIPE therapy for TB (required time for active TB according to the Department of Health) and was treated empirically for candidal esophagitis with micafungin. At the time of discharge, his AFB smear was still positive. He was enrolled in the directly observed therapy (DOT) program and deemed safe for discharge with appropriate isolation precautions from immuno-compromised individuals and children under 5. He was set for close follow-up with pulmonology, gastro-enterology (for an EGD), and cardiology (for the reverse takotsubo). Unfortunately, at the time of this report, the patient has not presented to his follow-up appointments.

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DISCUSSION

Here we present a case of classic pulmonary reactivation tuberculosis, which is important to distinguish from primary TB. In primary TB, a previously MTB naïve host is exposed and unable to mount an effective cell-mediated response leading to acute infection.[3] Patients most commonly present with fever, about 1/3 of patients present with pulmonary symptoms (pleuritic chest pain, pleural effusion, enlarged bronchial lymph nodes), and general malaise is less common. Radiographically, the most common finding is hilar lymphadenopathy.[4] In reactivation TB, the host can mount a successful cell-mediated response, there is a period of latency, and then the disease reactivates (usually from immunosuppressive conditions). Patients typically have a more prolonged period of sickness with weeks to months of cough, malaise, fevers, and night sweats. Radiographically, the disease most commonly presents in the upper lobes of the lungs (apical and posterior in 80-90% of patients) with little lymph node involvement.[5] Disseminated disease is much less likely unless severely immunocompromised.

Based on this information, the patient presented here fits into the reactivation TB category; weeks to months of fevers, weight loss, fatigue, and a right upper lobe cavitary lesion. Interestingly, however, one of his initial symptoms, months PTA, was odynophagia. While the etiology of the odynophagia remained unclear during his hospital stay, it is worth considering whether this was an oral manifestation of TB.

Oral manifestations are rare, reported in less than 0.5-1% of all TB patients.[6] The lesions can be primary (single painless ulcer) or secondary (associated with pulmonary disease) and manifest in a wide variety of ways including ulcers, nodules, patches, and soft tissue lesions anywhere in the oral cavity including the jaw and tongue. Without treatment in reactivation TB, patients can present with painful ulcers anywhere in the GI tract with the mechanism being prolonged expectoration and swallowing of the infectious secretions.[7]

In our case, the patient presented with months of odynophagia, white plaques that were not scrapable on either side of his mouth, and overall poor dental health. His immunosuppressive tests all came back negative and an EGD was ultimately deferred to the outpatient setting as he improved with RIPE therapy. He also had an X-ray of the mandible and a CT of the neck that were both largely unremarkable except for multiple dental caries and periapical lucencies. In retrospect, his oral exam was like those seen in patients presented in other case reports of oral manifestations of TB. One report describes a patient with a single 2x3cm "necrotic and whitish" ulcerative lesion on the tongue[8] and another reports ulcerative lesions of the tongue that were grayish with a granular surface[9]. Of note, oral lesions often occur in association with pulmonary disease, with active pulmonary disease seen in 14/15 patients with oral lesions in one study.[10] Unfortunately, we did not take pictures of our patient's oral lesions on presentation, nor did we perform biopsies.

In another study of 19 patients with esophageal TB, 84% of them had dysphagia and 42% of them had odynophagia. Endoscopy with mucosal biopsy was diagnostic in these patients.[11] Interestingly, mediastinal lymphadenopathy was present in all patients in this study, similar to the patient we present in this report. Similarly, another study of laryngeal TB, shows the most common symptom was odynophagia (35.3% of patients) and hoarseness (64.7% of patients).[12]

In summary, it's possible that our patient with confirmed cavitary pulmonary TB, with odynophagia, and findings concerning for possible leukoplakia on his tongue were all consistent with a rare manifestation of TB such as oral, esophageal, or laryngeal TB. Ultimately, he would have needed a biopsy of the tongue lesions and/or an EGD/laryngoscopy to confirm his odynophagia was from TB, which was not done. However, without other etiologies for his odynophagia and improvement of his symptoms after receiving RIPE therapy, this remains a likely possibility.

CONCLUSION

While the US is considered a country with a low incidence of TB, the public health implications are significant. The goal is to identify patients with active TB, treat them until they are not infectious, and initiate screening for any contacts to rule out TB. Being able to identify symptoms that may represent TB (outside of the classic presentations) can help providers get closer to accomplishing better infection control of TB.

REFERENCES

1. Houben RMGJ, Dodd PJ. The global burden of latent tuberculosis infection: A re-estimation using mathematical modelling. *PLoS Med.* 2016;13(10):e1002152. doi: 10.1371/journal.pmed.1002152.
2. Cole B, Nilsen DM, Will L, Etkind SC, Burgos M, Chorba T. Essential components of a public health tuberculosis prevention, control, and elimination program: Recommendations of the advisory council for the elimination of tuberculosis and the national tuberculosis controllers association. . 2020:1-27. http://dx.doi.org/10.15585/mmwr.rr6907a1external icon.
3. de Martino M, Lodi L, Galli L, Chiappini E. Immune response to mycobacterium tuberculosis: A narrative review. *Front.Pediatr.* 2019;7:350. doi: 10.3389/fped.2019.00350.
4. POULSEN A. Some clinical features of tuberculosis. *Acta Tuberc.Scand.* 1957;33(1-2):37-92; concl.
5. Barnes PF, Verdegem TD, Vachon LA, Leedom JM, Overturf GD. Chest roentgenogram in pulmonary tuberculosis. new data on an old test. . 1988;94(2):316-320. doi: 10.1378/chest.94.2.316.
6. Jain P, Jain I. Oral manifestations of tuberculosis: Step towards early diagnosis. *J.Clin.Diagn.Res.* 2014;8(12):ZE18-21. doi: 10.7860/JCDR/2014/10080.5281.
7. Pozniak A. Pulmonary tuberculosis: Clinical manifestations and complications. https://www.uptodate.com/contents/pulmonary-tuberculosis-clinical-manifestations-and-complications.
8. Yiğit O, Cinar U, Uslu Coşkun B, Başak T. Tuberculous ulcer of the tongue: A case report. *Kulak Burun Bogaz Ihtis Derg.* 2004;13(3-4):98-101.
9. Fujibayashi T, Takahashi Y, Yoneda T, Tagami Y, Kusama M. Tuberculosis of the tongue. A case report with immunologic study. *Oral Surg.Oral Med. Oral Pathol.* 1979;47(5):427-435. doi: 10.1016/0030-4220(79)90124-5.
10. Eng H, Lu S, Yang C, Chen W, Jaconson J, Van Dis M. Oral tuberculosis. 1996;81(4):415-420. https://www.sciencedirect.com/science/article/pii/S1079210496800163. doi: 10.1016/S1079-2104(96)80016-3.1
11. Dahale AS, Kumar A, Srivastava S, Varakanahalli S, Sachdeva S, Puri AS. Esophageal tuberculosis: Uncommon of common. *JGH Open.* 2018;2(2):34-38. doi: 10.1002/jgh3.12043.
12. Lou ZC, Li X. Leukoplakia or LPR: The misdiagnosis of laryngeal tuberculosis. *Ear Nose Throat J.* 2021;100(5_suppl):549S-553S. doi: 10.1177/0145561319891264.