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Case report

Pleural tuberculosis mimicking malignant mesothelioma

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A 67-year-old male presented to our clinic with suspicion for mesothelioma. He had an episode of pneumonia with right-sided parapneumonic effusion, treated with thoracentesis with 1.3 L of pleural fluid removed nine months prior to his presentation. On our evaluation he was asymptomatic and denied cough, fever, sweats or shortness of breath. Computer tomography (CT) of the chest revealed a complex appearing small right pleural effusion surrounded by thickened pleura with significant nodularity (Fig. 1A) suspicious for mesothelioma. Further investigation with positron emission tomography-computed tomography demonstrated increased fludeoxyglucose F 18 avidity along nodular right pleural thickening, including mediastinal pleural, typical for mesothelioma (Fig. 1B and Fig. 1C). Initially, patient underwent an ultrasound guided pleural biopsy that showed scant atypical cells with high nuclear to cytoplasmatic ratio. Immunostains performed on cell block, show the atypical cells to be positive for CD68 and negative for calretinin and pan-cytokeratin. The findings were suggestive of histiocyte origin of the cells and re-biopsy was recommended. Cultures for tuberculosis (TB) and fungus were negative. Due to suspicion of malignancy, anterior thoracotomy with pleural biopsies were done which revealed extremely thick and rock-hard pleura with atypical spindle cell proliferation admixed with lymphohistiocytic inflammatory reaction and dense fibrocollagenous tissue. Immunohistochemical studies show that the atypical cells were positive for CD163, weakly and focally positive for WT-1 and negative for pancytokeratin, calretinin, CK5/6, TTF-1, and p40. Overall, this immunophenotype favored a reactive process. Mediastinoscopy and lymph nodes biopsies in level IVR, IVL, VII revealed no tumor evidence of mesothelioma, besides non-necrotizing granulomatous inflammation. Patient was placed on surveillance and few months after, the PET showed persistent active right pleural thickening but now with new left pleural effusion, ascites, and FDG peritoneal nodularity (Fig. 2A). Repeat pleuroscopy on the left showed a lymphocytic predominant pleural effusion, with high protein (6.5 gm/dl) and cholesterol levels (102 mg/dl) while biopsy showed florid granulomatous inflammation (Fig. 2B and Fig. 2C). Final cultures were positive for mycobacterium tuberculosis (MTB).

Untreated pneumonic-related pleural effusion resulting in fibrothorax is the second most common cause of pleural effusions in the US with a reported incidence of 300,000 annually [1]. Many etiologies exist for pleural effusions such as CHF and infectious-related empyemas and their treatments vary accordingly [1]. Although most patients recover, for untreated or complicated cases the mortality rate is approximately 10% with 15–20% of patients needing surgical intervention such as drainage or decortication [2]. In patients with pleural inflammation caused by bacteria, mycobacterium, or autoimmune disease, patients present with fever, malaise, chest pain, nonproductive cough, and dyspnea [2]. Interestingly, the asymptomatic diffuse pleural thickening in our case showed intense FDG uptake and proved to be due to MTB and not malignancy. The pathogenesis of primary tuberculous pleurisy is a delayed-type hypersensitivity immunogenic reaction to a few mycobacterial antigens entering the pleural space rather than direct tissue destruction by uncontrolled mycobacterial proliferation [3]. This appears to be the reason for the low rate of positive results in acid-fast staining of pleural fluid and tissue, as seen in this case.

Conclusion

Active tuberculosis must be considered in the differential diagnosis of diffuse FDG avid pleural thickening even when there are no subjective symptoms. Patient was started on a four-drug regimen with ethambutol, isoniazid, pyrazinamide and rifampin and showed clinical improvement.

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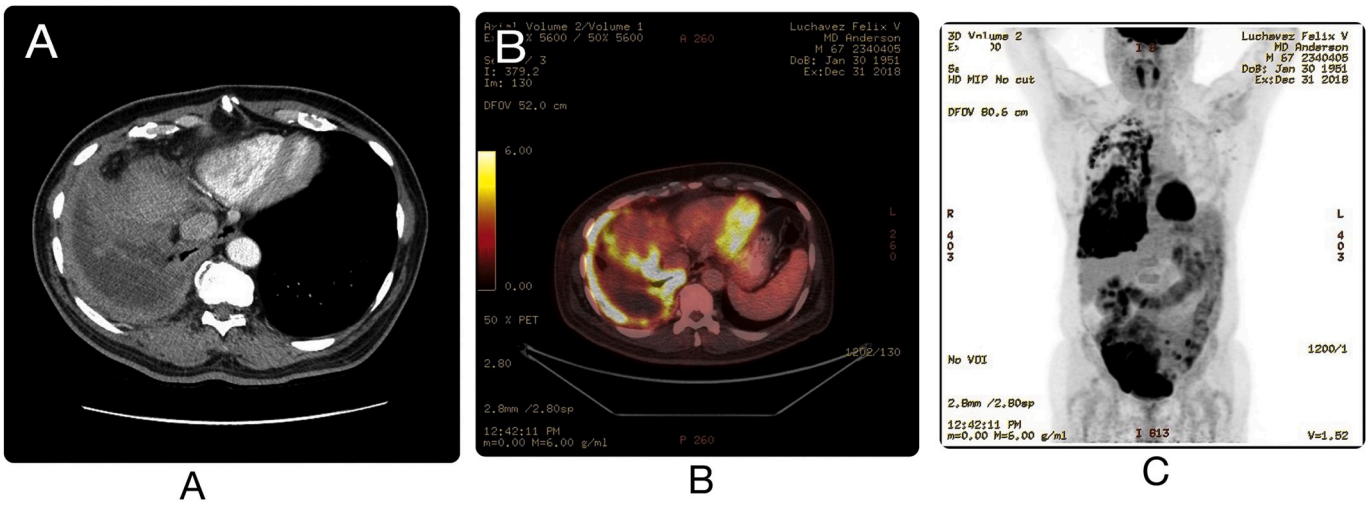

Fig. 1. A Computer tomography (CT) of the chest showing a complex appearing small right pleural effusion surrounded by thickened pleura with significant nodularity. B PET-CT showing increased FDG avidity along nodular right pleural thickening, including mediastinal pleural. C PET-CT(coronal view) showing increased FDG avidity along nodular right pleural thickening, including mediastinal pleural.

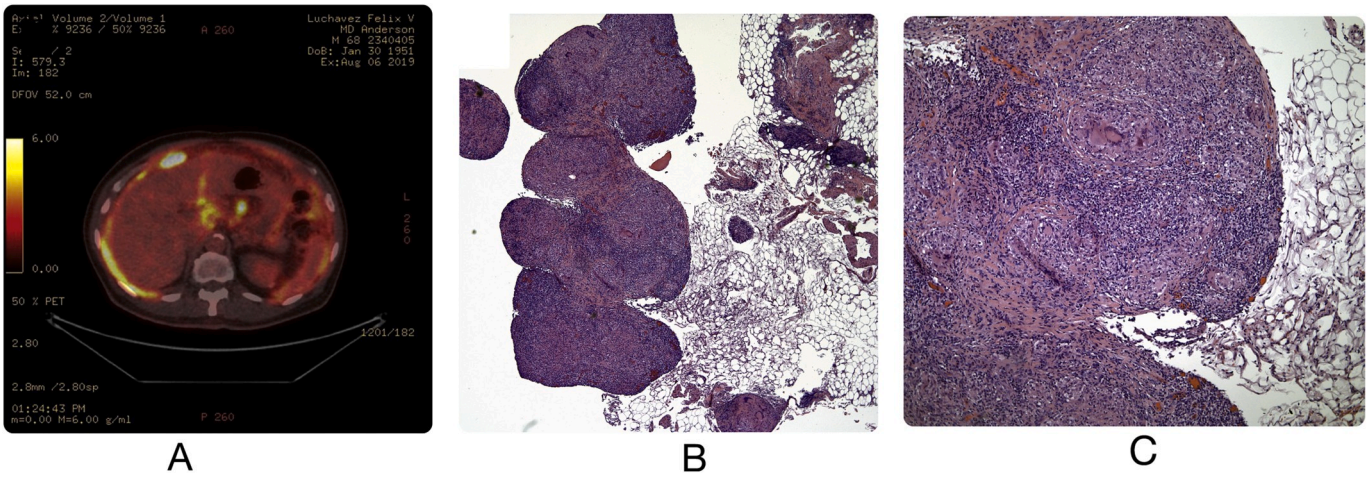

Fig. 2. A PET-CT showing increased FDG avidity along nodular right pleural thickening, including mediastinal pleural and new FDG avidity in the peritoneum. B: Pleural biopsy showing florid granulomatous inflammation(low-power field) C: Pleural biopsy showing florid granulomatous inflammation(high-power filed).

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Declaration of competing interest

The authors have no conflicts of interest to disclose.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.rmcr.2019.100964.

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