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MODESTUM

Case Report **OPEN ACCESS**

Confluent cavitated nodules in invasive mucinous adenocarcinoma: A case report

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ABSTRACT

Invasive mucinous adenocarcinoma is a rare variant of lung adenocarcinoma. Here, we present the case of a patient with invasive mucinous adenocarcinoma with cavitary lung lesions. A 61-year-old painter-and-bricklayer. She was admitted due to mMRC2 dyspnea, a dry cough that during hospitalization mobilizes dense, abundant secretions, and becomes demanding. Differential diagnoses were made based on clinical symptoms and images, performing multiple laboratory tests ruling out immunosuppression, and two video-bronchofibroscopies finding the diagnosis in the transbronchial lung biopsy: Invasive mucinous adenocarcinoma that would explain the abundant bronchorrhea, advanced stage and poor prognosis led to ventilatory failure and death of the patient.

Keywords: lung cancer, lung cavity, invasive mucinous adenocarcinoma, prognosis, computed tomography, Peru

INTRODUCTION

Globally, there is an increase in cases of lung cancer, being the most important factor in cancer mortality [1]. Lung adenocarcinoma (ADC) is the most common subtype, accounting for approximately 40% of cases, and the invasive mucinous adenocarcinoma (IMA) variant has clinicopathological, radiological, and molecular features distinct from other ADCs [2, 3].

With an uncertain prognosis, IMA presents invasive patterns of columnar/goblet cells with abundant intracytoplasmic mucin [4, 5] and multiple consolidations or ground-glass opacities on computed tomography (CT) mimicking pneumonia. IAM with cavitary lesions has also been reported, although no cases of IMA with multiple lung cavities have been reported in Peru.

We report a case of IMA with confluent cavitary lesions in the lung following the guidelines of the CARE guide for case reports [6-9].

CASE REPORT

Case Presentation

An unseemliness, 61-year-old painter-and-bricklayer, with a history of recently diagnosed gonarthrosis, diabetes mellitus (treated with insulin R on a sliding scale), and smoker with IPY: 1.33, was referred to the emergency area of Hospital Nacional Guillermo Almenara (Peru) for not improving the previous management of mMRC2 dyspnea. Physical examination revealed clubbing of both hands, diffuse inspiratory and expiratory wheezing in bilateral lung fields, and bibasal crackles. General examination was unremarkable.

He has 25 years of work as a painter of electrical appliances with the use of personal protective equipment (PPE) and as a bricklayer without the use of PPE. One month ago, he had mMRC2 dyspnea, dry cough, and asthenia, and before admission, he went to a doctor for SatO2: 90% to FiO2: 21% and received treatment for infection with ceftriaxone and ciprofloxacin for seven days without clinical improvement. During his hospitalization, the cough was hyaline, and semi-dense, with hemoptoic features that became harsh, requiring supplemental low-flow oxygen using a 2L nasal cannula, Fio2: 28%, and maintaining Sato2: 97%.

Investigations

Antigen testing and PCR were performed for SARS-CoV-2 and were negative. He had multiple negative sputum smears, normal biometric parameters, viral hepatitis profile, HTLV-1/2, HIV, tumour markers, and rheumatology profile: negative. In addition, the CD4+/CD8+ count was normal, ruling out immunosuppression. The set of tests performed on the patient is shown in **Figure 1**.

Chest transmission electron microscopy (TEM) with contrast shows multiple dense nodules randomly distributed bilaterally,with a tendency to basal consolidation and cavitated (**Figure 2**).

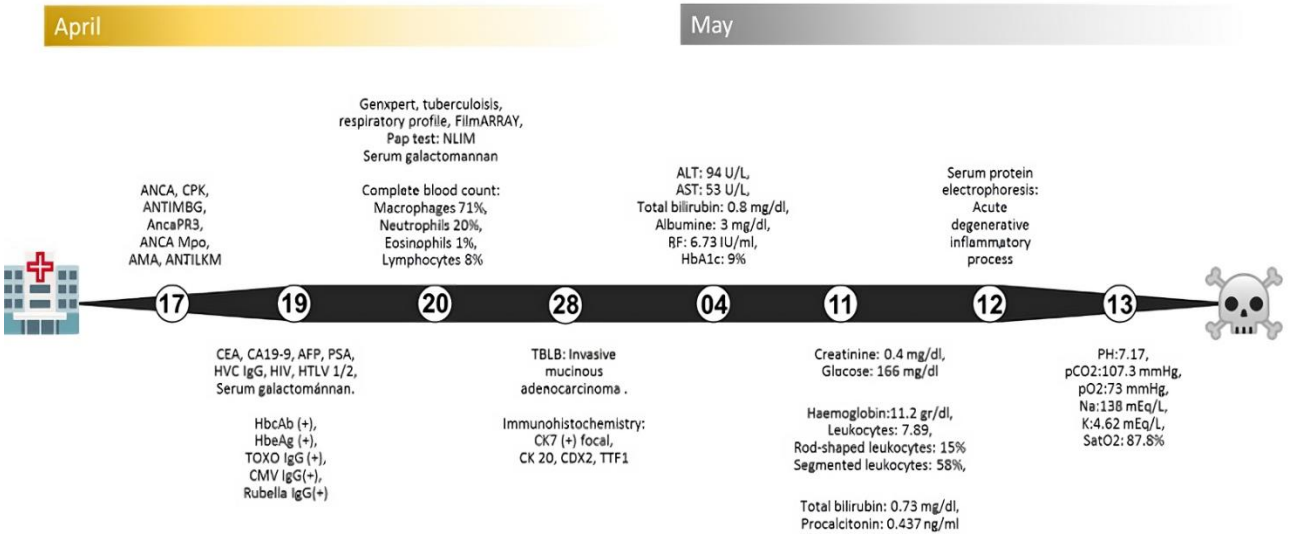

Figure 1. Roadmap of patient with invasive mucinous adenocarcinoma (RF: Rheumatoid factor; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; HbA1c: Hemoglobin A1c; TBLB: Transbronchial lung biopsy; HbcAb: Hepatitis B virus core antibody; HbeAg: Hepatitis B antigen; TOXO: Toxoplasma; CMV: Cytomegalovirus; ANCA: Antineutrophil cytoplasmic auto-antibodies; ANTILKM: Anti-liver kidney microsomal antibody type 1; AMA: Anti-mitochondrial autoantibodies; AncaPR3: Differentiate proteinase 3-ANCA; ANCA Mpo: Myeloperoxidase-ANCA; ANTIMBG: Anti-glomerular basement membrane disease; CPK: Creatine phosphokinase; CA19-9: Carbohydrate antigen 19-9; CEA: Serum carcinoembryonic antigen; AFP: Alpha fetoprotein; PSA: Prostate specific antigen; Pap-test: Papanicolaou-test, NLIM: Negative for intraepithelial lesion/malignancy; CK7: Cytokeratin 7; CK: Cytokeratin 20; CDX2: Homeobox protein CDX-2; TTF1: Thyroid transcription factor 1; & SatO2: Oxygen saturation) (Source: Own creation ©Jeel Moya-Salazar)

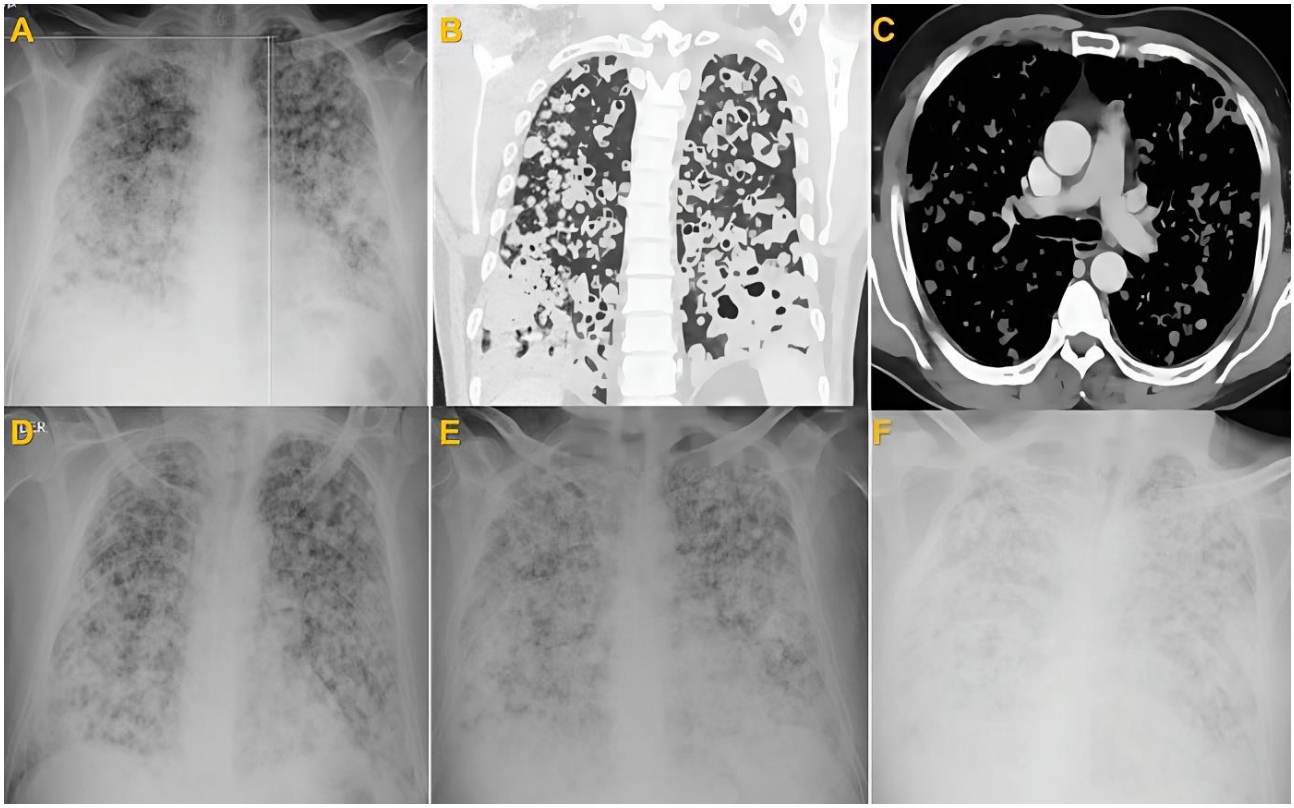

Figure 2. Follow-up of radiological alterations of patient with cavitated nodules & IMA: (A) Emergency admission (X-ray); (B & C). Hospitalization in pulmonology, day 2 TEM chest c/c; (D) Post first flexible bronchoscopy (X-ray); (E) Post second flexible bronchoscopy (X-ray); & (F) Day of death (Source: Own creation © Madaleine López-Hinostroza)

For this reason, acute pneumonic process and nicotic aetiology were considered. The first bronchoalveolar lavage (BAL) video bronchoscopy was performed in W5 of right lung with results of cell count (macrophages: 71%, neutrophils 20%, eosinophils 1%, lymphocytes 8%) PAP (papanicolaou test) negative for neoplastic cells, culture, fungi film array, sputum smear test, galactomano, and genxpert respiratory panel: negative. The procedure was suspended due to acute respiratory failure (Sato2<80%), recovering with oxygen supplemental.

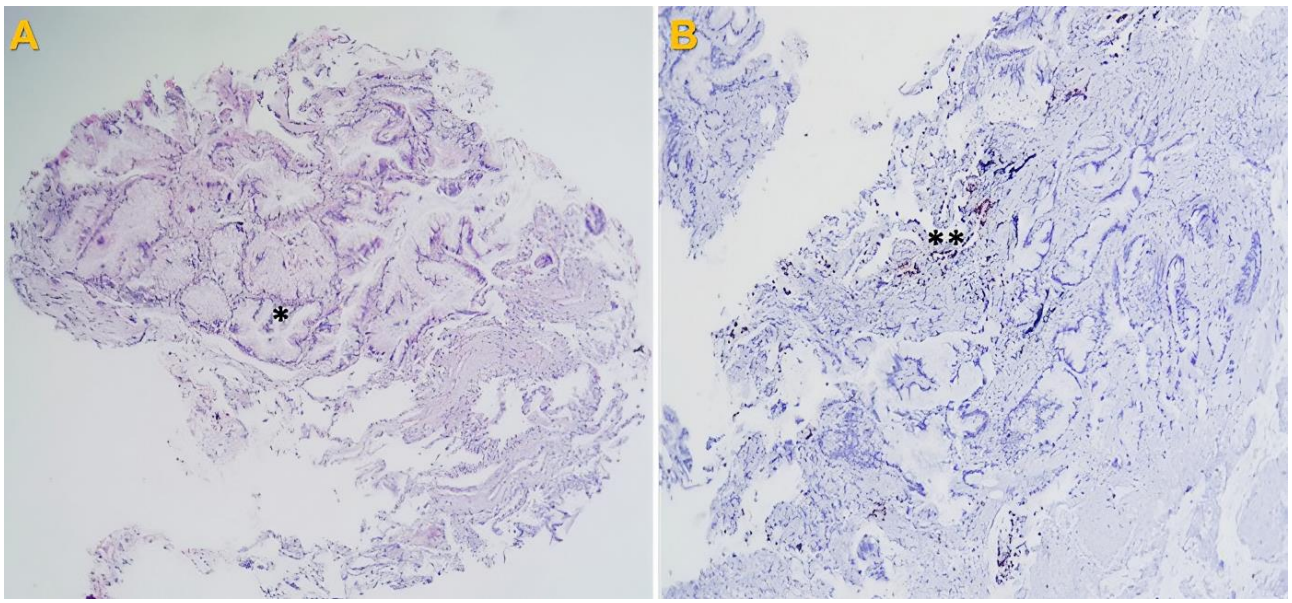

Figure 3. Histopathological results: (A) Lung biopsy by bronchoscopy in H&E (100X)–Invasive mucinous adenocarcinoma at the level of the lung parenchyma (*) & (B) TTF1 presence of nuclear staining at the level of the alveoli and negativity at the level of the neoplasm (**) (Source: Own creation © Angélica Y Asencio)

The day after the procedure, he presented an episode of fever, cough with dense yellow-greenish fetid expectoration, polypneic, and increased need for supplemental oxygen, starting treatment with piperacillin/tazobactam, classifying as hospital-acquired pneumonia (clinical pulmonary infection score scores two points), culminating treatment for 10 days. On the same day, the result of BAL and sputum culture was obtained: *Klebsiella pneumoniae spp ozaenae pansensitive*.

Seven days later, a second video bronchoscopy was performed, effecting transbronchial lung biopsy (TBLB) in LB10b of left lung, obtaining seven samples for histopathological study. The results of the TBLB diagnosed an IMA, with immunohistochemistry suggesting a primary pulmonary location with cytokeratin (CK) 7: focal positive, CK 20: negative, CDX2: negative, Thyroid transcription factor 1 (TTF1): negative (**Figure 3**).

Treatment

For NIH treatment, piperacillin/tazobactan 4.5 g every six hours for 10 days was used. In addition, codeine 20 mg every eight hours IV, enoxaparin 40 mg every 24 hours, SC acetylcysteine 200 mg every eight hours per os (P.O.) and salbutamol 2 puffs every four hours × were administered. The development of respiratory complications such as abundant bronchorrhea defined the additional use of tranexamic acid 1 g every eight hours IV, salmeterol/fluticasone 50 ug/250 ug 2 puffs every 12 hours × AEC, and after evaluation by oncology, morphine 30 mg diluted in Nacl 0.9% was used, and infused at 5 cc/h.

Outcome and Follow-Up

Seven days later, severe pulmonary compromise was evidenced, which progressed with respiratory deterioration, poor ventilatory pattern, inspiratory and expiratory wheezing in PCA, multiple shots, cough with increased mobilization of dense hyaline secretions, greater requirement for oxygen therapy with a Venturi mask at FiO2: 50% with SatO2: 97%, and arterial blood gas test results showed respiratory acidosis type II.

The patient was evaluated by oncology, which suggested support and quality of life measures, for which analgesia was started with morphine infusion and inhalation therapy, presenting agonal breathing, with death the following day.

DISCUSSION

IMA was defined as a rare variant of ADC because it occurs in 5% to 10% of cases according to a consensus of three lung cancer and respiratory disease research organizations [10]. As a rare entity, its diagnosis is usually dismissed as part of the differential diagnosis of lung cavities; therefore, clinical case reports in Peru are uncommon, as these carcinomas accounted for only 3.8% of cases [11].

Although the clinical presentation may show non-cavitary alterations, cases with cavitary lesions tend to have a poor prognosis due to complications and the rapid deterioration of patients, as we reported in this case, coinciding with other previous reports [7, 12]. Upon patient admission, an infectious process associated with tuberculosis or pneumonia was considered due to the radiological characteristics, as has been reported in IMA with *mycobacterium fortuitum* infection [13]. It is important to consider IMA with cavitary radiologic features as part of the differential diagnosis.

The differential diagnosis of cavitated lung lesions is mainly of infectious aetiology and from the neoplastic point of view, squamous cell carcinoma is the most com-mon (part A in **Figure 3**). Additionally, the multicentric presentation gives a suspicion of a metastatic lesion rather than a primary one; however, this could be explained by the aerogenic dissemination of the mucinous adenocarcinoma that causes multilobar and bilateral lesions (part B in **Figure 3**). Microscopically, it is characterized by cells with abundant intracytoplasmic mucin and small basal nuclei. His immunohistochemical profile was positive for CK7 and CK20 and negative for TTF1 and napsin A.

CONCLUSION

We report one of the first cases in Peru of IMA's lung cavitated lesions with tissue positivity for CK7 and CK20. IMA is a rare variant of adenocarcinoma that does not always present with lung cavity lesions. Multiple cavity lesions in the lung can be caused by a variety of conditions, such as tuberculosis and primary lung cancer, therefore, IAM should be considered as part of the differential diagnosis. Invasive mucinous adenocarcinoma with cavitary lesions has a poor prognosis due to complications and rapid patient deterioration.

Author contributions: ML-H & JM-S: provided study concept & design, data acquisition, data management, & wrote manuscript; **JD:** provided study concept & design, data acquisition, & data management; & **AYA:** data acquisition, data management, & wrote manuscript. All authors have agreed with the results and conclusions.

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Declaration of interest: No conflict of interest is declared by authors.

Data sharing statement: Data supporting the findings and conclusions are available upon request from the corresponding author.

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