Reviewer #1:

Scientific Quality: Grade C (Good)

Language Quality: Grade A (Priority publishing)

Conclusion: Minor revision

Specific Comments to Authors: The manuscript presents a interesting and particular case. However, Introduction and discussion sections could be improved if some data are compared with those reported in previous works. Please consider the content of the following: - Trapnell, B. C., Nakata, K., Bonella, F., Campo, I., Griese, M., Hamilton, J., ... & McCarthy, C. (2019). Pulmonary alveolar proteinosis. Nature Reviews Disease Primers, 5(1), 16. - Salvaterra, E., & Campo, I. (2020). Pulmonary alveolar proteinosis: from classification to therapy. Breathe, 16(2). - Iftikhar, H., Nair, G. B., & Kumar, A. (2021). Update on diagnosis and treatment of adult pulmonary alveolar proteinosis. Therapeutics and Clinical Risk Management, 701-710. - Sakaue, S., Yamaguchi, E., Inoue, Y., Takahashi, M., Hirata, J., Suzuki, K., ... & Okada, Y. (2021). Genetic determinants of risk in autoimmune pulmonary alveolar proteinosis. Nature communications, 12(1), 1032. - Smith, B. B., Torres, N. E., Hyder, J. A., Barbara, D. W., Gillespie, S. M., Wylam, M. E., & Smith, M. M. (2019). Whole-lung lavage and pulmonary alveolar proteinosis: review of clinical and patient-centered outcomes. Journal of cardiothoracic and vascular anesthesia, 33(9), 2453-2461. The current strategies for studying patients with this condition should be highlighted. Also, you can add some therapeutic proposals from recent studies. Fig 3. Could be deleted.

1.**Discussion**

PAP is a syndrome characterized by the accumulation of alveolar surfactant and dysfunction of alveolar macrophages[3]. Management aims at improving symptoms and quality of life; whole-lung lavage effectively removes excessive surfactant[4]. Novel pathogenesis-based therapies are in development, targeting GM-CSF signalling, immune modulation and cholesterol homeostasis. PAP is a rare respiratory syndrome characterised by the accumulation of surfactant lipoproteins within the alveoli[5]. According to various pathogenetic mechanisms and aetiologies, PAP is classified as primary, secondary or congenital[5-7]. Primary PAP is led by GM-CSF signalling disruption; the autoimmune form is driven by the presence of anti GM-CSF autoantibodies and represents 90% of all the PAP cases; and the hereditary form is the result of mutations in genes encoding GM-CSF receptor. Secondary PAP is associated with various diseases causing a reduction in function and/or number of alveolar macrophages. Congenital PAP emerges as a consequence of corrupted surfactant production, due to mutations in surfactant proteins or lipid transporter, or mutations affecting lung developmen.PAP is more common in middle-aged and young adults and is approximately three times more common in men than in women. Dust exposure and particularly exposure to silica dust can cause PAP. It is believed that PAP may be a nonspecific response to certain stimuli, leading to the breakdown of alveolar macrophages and the production of PAS-positive proteins[6, 8-10]. PAP is one of the challenging diseases to diagnose clinically, and a definitive diagnosis is usually made by histopathology[11].The primary methods of obtaining tissue specimens include transbronchial forceps biopsy (TBFB), percutaneous biopsy, and surgical lung biopsy (SLB). TBFB has limited diagnostic efficacy due to the small size and poor quality of the specimens obtained, making it difficult to meet pathological requirements. Percutaneous lung puncture biopsy also faces the problem of small specimens often not meeting pathological needs and carries the risk of pneumothorax and hemopneumothorax.

SLB is less commonly used due to its high invasiveness, high cost, and limitations in patients with reduced cardiopulmonary function.

2. **Fig 3.and Fig 2. have been deleted.**

Reviewer #2:

Scientific Quality: Grade B (Very good)

Language Quality: Grade A (Priority publishing)

Conclusion: Accept (General priority)

Specific Comments to Authors: Thanks for submitting the finding.