The authors mention that both the cases were successfully managed with whole lung lavage. It is also mentioned that there was marked improvement in the condition of both the patients after one week of BAL. There is however, no follow up available for either of the patients. It is well known that patients of PAP may require repeat BAL after a certain interval of time which may vary from patient to patient. Some of the patients do not show any remission and rather worsen in severity. Hence, is it appropriate to comment on the success of BAL just on the basis of one week of observation? For the benefit of readers of the MJAFI, we illustrate serial CT scan findings of case 2 who was followed up with CT scan once after nearly six months and then exactly after 2 years (Fig. 1). The follow up images available with us, depict only partial resolution of lesions even after two years.

Overall, we find that the role of radiology has been understated by the authors in the diagnosis and management of both these cases. This is likely to give an erroneous message to the readers of the esteemed journal of the AFMS.

Reply

Dear Editor,

On behalf of all the authors, I thank the writers for their deep interest, thorough study and in depth imaging analysis and follow up of the case report titled "Primary alveolar proteinosis – A report of two cases".¹ I agree with the writers that HRCT scan chest is an important test in the diagnosis and follow up of a case of pulmonary alveolar proteinosis (PAP).

However, we disagree with the writers about listing HRCT scan chest as one of the key words in the present case report. Key words are listed by authors of scientific articles to help readers to search for the articles they are interested in reading or doing research. HRCT scan chest gives a clue to the diagnosis of PAP in an appropriate clinical set up. However, PAP is a rare disease. Hence, out of more than a few hundreds of HRCT scan chest done in a centre, only a few cases will be finally diagnosed as PAP. The readers have also given at least eight causes of HRCT scan chest findings similar to PAP. Hence, listing HRCT scan chest as one of the key words in the present case report is not justified as it is unlikely to help many readers searching for articles on PAP or whole lung lavage (WLL).

Both the cases were treated at peripheral hospitals as pulmonary tuberculosis. This has been mentioned in the case report. The patients were evaluated subsequently at the tertiary care centre with HRCT scan chest, bronchoscopy, bronchoalveolar lavage (BAL) and lung biopsy, which confirmed the diagnosis of PAP. Readers will agree that HRCT scan chest alone cannot confirm a diagnosis of PAP without BAL and/or histopathological findings.

Response to WLL is evaluated clinically (both symptoms and signs), and by objective physiological parameters (which

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include forced vital capacity (FVC), DLCO, PO₂ at rest and with exercise), along with X-ray and HRCT scan chest.² In the present case report, both patients showed improvement in clinical and physiological parameters following WLL. Similar responses were maintained when they came for follow up subsequently. But case 2 showed partial resolution in HRCT scan chest as mentioned by the readers. Similar observation has also been recorded in literature: "Some patients are asymptomatic despite significant radiographic abnormalities: others undergo spontaneous remission and do not require treatment".²

We agree with the writers that response of patients of PAP to WLL is variable; and the same has been mentioned in the case report in discussion.^{1,3}

There was no intention of the authors to understate the role of radiology in diagnosis and management of both these cases. Rather we very much appreciate the role of radiology in both diagnosis and management of PAP. Actually management of PAP is a multispecialty team approach with pulmonologists, radiologists, pathologists, and anaesthesiologists (for single lung ventilation during WLL).

On behalf of all the authors, I again thank the writers for their critical evaluation and discussion of both the cases and for publishing the serial HRCT scan chest findings of case 2.

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Is HAPO the exclusive diagnosis of pulmonary oedema at HA?

Dear Editor,

The article on extreme altitude pulmonary oedema (EAPO) in acclimatised soldiers published in MJAFI 2012; 68: 339–345 by Maj I. D. Khan suggests that the soldiers were expected to be “acclimatised” after 39 ± 13.79 days of stay at 19,000–23,000 ft. Lowlanders never acclimatise to extreme altitude and some changes that constitute some acclimatisation at this altitude occur over much longer periods of time.¹ The title of the article is thus misleading. HAPO is known to occur in acclimatised individuals and usually follows an event like gain in altitude or respiratory infection.^{2,3} Its *de novo* occurrence in 21 soldiers, resident at a given altitude for weeks, is intriguing. That 71% cases were native highlanders is interesting since native highlanders of Ladakh have minimal musculature in the pulmonary artery which mitigates acute pulmonary hypertension which is central to the pathophysiology of HAPO.⁴ In view of the atypical setting and the large number of cases reported, we request the author to clarify the following:

1. A number of conditions like bronchitis, acute MI and pulmonary embolism may mimic HAPO.⁵ How were these conditions ruled out?
2. What were the values of vital signs and SpO₂ in the patients and healthy soldiers at that altitude?
3. Did investigations at Base Camp/Siachen hospital confirm the diagnosis?
4. Over what time period did the 31 cases occur?
5. What is the validity of using SpO₂ <75% as a criterion for diagnosing HAPO?
6. Why were acetazolamide and dexamethasone used to treat HAPO instead of internationally accepted treatment modalities^{2,5}
7. Why and on what basis was Nifedipine administered to only four “select” patients?
8. How were the symptoms/signs of HAPO (predominantly pulmonary) confusing with those of AMS (predominantly neurological)?

We also wish to correct certain factual inaccuracies in the manuscript:

1. Hypertension and orthopnoea are not features of HAPO.
2. Stage I “Acclimatisation” begins at/above 9000 ft and not below 9000 ft as depicted in Table 2.

3. HAPO bag therapy does not reset the patients’ physiology to lower altitude. It improves oxygenation and residual benefits may last for a few hours outside.
4. The descent simulated by the HAPO bag has been overstated in the example of Mt Everest.
5. Compression increases the PO₂ and not the oxygen concentration (FiO₂) inside the bag.
6. Some HAPO bags are designed to be carried as stretchers (DRDO bags).

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