Review Article

Pulmonary alveolar microlithiasis

Surender Kashyap, Prasanta R. Mohapatra[1]

Department of Pulmonary Medicine, Kalpana Chawla Government Medical College, Karnal, Haryana, [1]Department of Pulmonary Medicine, Government Medical College and Hospital, Chandigarh, India

ABSTRACT

Pulmonary alveolar microlithiasis (PAM) is a rare, chronic lung disease with bilateral intra-alveolar calcium and phosphate deposition throughout the lung parenchyma with predominance to lower and midzone. Although, etiology and pathogenesis of PAM is not fully understood, the mutation in SLC34A2 gene that encodes a sodium–phosphate co-transporter in alveolar type II cells resulting in the accumulation and forming of microliths rich in calcium phosphate (due to impaired clearance) are considered to be the cause of the disease. Chest radiograph and high-resolution CT of thorax are nearly pathognomonic for diagnosing PAM. HRCT demonstrates diffuse micronodules showing slight perilobular predominance resulting in calcification of interlobular septa. Patients with PAM are asymptomatic till development of hypoxemia and cor-pulmonale. No therapy has been proven to be beneficial except lung transplantation.

KEY WORDS: Calcification, calculi, microliths, pulmonary alveolar microlithiasis

Address for correspondence: Dr. Surender Kashyap, Director, Kalpana Chawla Government Medical College, Karnal, Haryana, India.
E-mail: surenderkashyap@hotmail.com

INTRODUCTION

Pulmonary alveolar microlithiasis (PAM) is a rare disease, characterized by presence of diffuse innumerable minute calculi called microliths in the alveoli of the lungs. PAM was first reported by Norwegian Harbitz in 1918;[1] accordingly, it is also known as Harbitz' syndrome. It was named as Pulmonary Alveolar Microlithiasis by Ludwig Puhr of Hungary in 1933.

EPIDEMIOLOGY

Less than 800 cases have been reported worldwide in medical literature till date from all the continents of the globe without any particular geographic or racial distribution. Asia and Europe shared nearly one-third cases each. Most of the cases have been reported from Turkey, Japan and Italy. PAM occurs in both sexes with a slight predominance among males worldwide except in Italy where female predominance has been reported.

Access this article online

Quick Response Code:

Website: www.lungindia.com

DOI: 10.4103/0970-2113.110424

Although, PAM is seen in all age groups, it is most frequently diagnosed from birth to 40 years of age. The age at diagnosis spans a wide range, with predominance of younger age of nearly half of Japanese patients diagnosed in the pediatric age group, whereas one-fifth patients are found at over 40 years of age.[2] Two reviews on the subject have been reported with mean age of 27-30 years at the time of diagnosis.[3,4] Of the 576 patients analyzed together, 35.8% were below 20 years of age and 88.2% were under 50 years.[4] There was no sex difference as far as the rate of occurrence, age at diagnosis and the progression of the disease was concerned.[3-5] The youngest reported cases were premature twins and two newborn, while the most elderly was of 80 years of age.[5]

It was first reported from India by Viswanathan.[6] Not more than 30 cases are reported in literature from different regions of India till date.[7-11] We came across six cases of PAM during our chest practice at Shimla and all of them belonged to the hilly region of Himachal Pradesh.[12,13]

ETIOLOGY, PATHOGENESIS AND PATHOLOGY

Definite etiology and pathogenesis are not known. The most accepted etiology suggested that it is an inherited abnormality, limited to alveolar surface, involving the enzyme carbonic anhydrase, which promotes alkalinity of the alveolar surface, with consequent precipitation of calcareous salts.[14] The serum concentrations of surfactant

proteins A and D and other markers for interstitial lung disease are higher in patients with PAM.[15]

In 1957, Sosman showed a high incidence of familial links, signifying the presence of a hereditary factor in pathogenesis of PAM.[16] Further analyses of such familial cases indicate the characteristics of autosomal recessive disease. Recently, the gene responsible for PAM has been identified.[17,18] Familial occurrence has been observed in 50% of Japanese,[18] 48% of Turkish,[4,19] 43.7% of Italian patients, whereas 35.6% cases of PAM have familial presentation worldwide.[5] When multiple patients exist in a family, they are usually siblings. Interestingly, in Japan, 16 families had two patients each, six families had three patients each whereas, in another family four patients of PAM were confirmed.[18] A Turkish family having 6 patients from different generations has been reported.[15] There was history of consanguineous marriage in 30% of the parents of Japanese patients, who did not suffer from PAM.[18] Transmission is restricted to siblings; parental consanguinity was present in several siblings, supporting an autosomal recessive inheritance.[3,19-21] Both horizontal accumulation of the patients in a family and the presence of consanguineous marriages in the parents suggest that PAM is an autosomal recessive disease with a high penetrance. An inborn error of metabolism has been suggested due to the high rate of occurrence within families.

Composition of the calcifications is calcium and phosphate, in a ratio of 2:1.[22] The microliths are rounded, oval and lobular concentric laminated in appearance. Grossly the lungs are firm to hard in consistency. So far less than 60 autopsy proven cases of PAM are reported in the literature. Among the cases reported, the maximum weight of the lung was 4880 g followed by 4500 g.[1,5] There is presence of numerous tiny calculi (calcospherites), ranging from 0.01 to 3.0 mm, within the alveoli. During early stage, alveolar walls are normal but with the progression of the disease and at a later stage fibrosis, interstitial thickening and giant cells are noticed. Apical blebs and bullae may cause recurrent pneumothorax in these cases.

GENETICS OF PAM

One study group from Turkey used linkage analysis on a large affected family in Turkey[17,21] and the other group from Japan studied a modified homozygosity mapping method using three unrelated patients. Both the groups confirmed that the SLC34A2 gene encodes a type IIb sodium-dependent phosphate transporter.[18] The SLC34A2 gene is primarily expressed in alveolar type II cells,[17] and is known for sodium-dependent phosphate transporter expressed in the lungs. This protein transports the phosphorus ion from the alveolar space into the alveolar type II cells. Accordingly, the alveolar type II cells are unable to clean up the phosphorus ion from the alveolar space resulting in its accumulation and forming microliths rich in calcium phosphate.

PATHOLOGY

Lung histology shows intraalveolar calcospherites and slight fibrosis of alveolar wall. Further, the lung shows exclusively calcified material in the form of amorphous powdery particles chemically consisting the microliths that are rich in calcium and phosphate.[23-25] Interestingly, patients of PAM have normal serum calcium and phosphorus levels. Lung biopsy and autopsy specimens demonstrate the characteristic intraalveolar lamellar microliths [Figure 1]. In the autopsy cases of advanced PAM, the microliths are crowded in the subpleural space, interlobular septa and bronchovascular bundles, where fibrosis and ossification are often observed.[18,26] Scanning with electron microscopy has demonstrated microliths with a distinguishing concentric lamellar structure in the lung tissue.

CLINICAL COURSE

Little information is available on the clinical course, including the initial phase, evolution and stabilization of PAM. Most of the literature is in the form of case reports highlighting a particular aspect. Paucity of patients in particular locality in certain duration may be the reasons for it. In some cases, the illness remains static as regards to both symptoms and radiographic findings.[23-25] We followed a female patient for more than 15 years with very slow progression of the disease. While, in some other cases it has worsened over time in different rates, leading to pulmonary fibrosis, respiratory failure and chronic pulmonary heart disease (cor-pulmonale). Dyspnea on exertion occurs as the disease progresses and late symptoms of cor pulmonale appear as terminal manifestation. Cough may occur at any stage of disease and even the expectoration of microliths has been reported. Usually, the slow progression of the disease ultimately leads to fatal respiratory or cardiac failure. Pulmonary fibrosis, cor pulmonale and respiratory insufficiency may result with severe involvement. The diagnosis can be made before clinical manifestations by

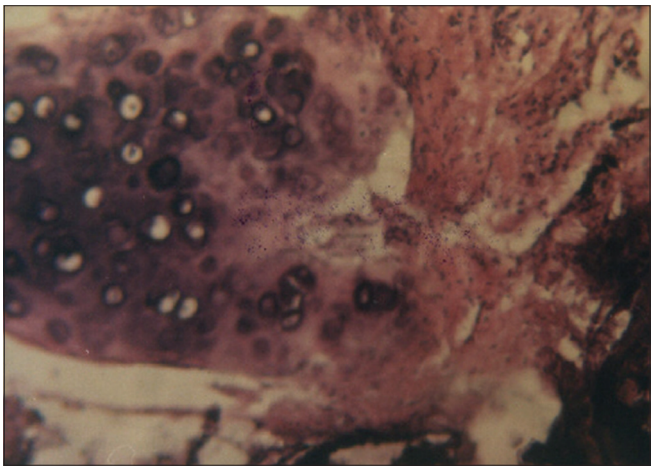

Figure 1: Histopathology section showing multiple calcospherites and fibrosis in the alveoli of lung parenchyma (H and E, ×100)

the screening of the family of the index patient, or by a mass chest radiograph examination in selected population.

RADIOLOGY

PAM is a typical example of clinical and radiological dissociation. The characteristic picture of PAM on the chest radiograph shows infiltrates as fine sand-like calcific micronodules also called 'sandstorm lung', diffusely involving both lungs, usually most marked in middle and lower zones (inferior and posterior predominance), which often obliterates the mediastinal and diaphragmatic outline. In advanced stages of PAM, the microliths are distributed within the perilobular interstitium. Characteristic chest CT findings at advanced stage are calcified thickening of interlobular septa, bronchovascular bundles, and pleura.[18,26-28] CT shows symmetrical abnormalities in the lungs, usually as marked calcifications. The calcifications are most prominent in peripheral, mediastinal and fissural subpleural regions and each lobe is surrounded by a fine dense outline, giving the overall appearance of a stony lung [Figures 2 and 3]. Areas with ground-glass opacity are commonly seen. Intervening consolidation often includes calcification and its calcification can be visualized at mediastinal window settings. Subpleural multiple small cysts are also seen.[18,26] The opacities may be so dense to make the lungs appear almost uniformly white. Radiological appearance of stannosis, talc granulomatosis and calcified miliary histoplasmosis may simulate microlithiasis but the lesions in these conditions are larger and have different distribution. The heart border and the diaphragm may be obliterated. Felson first reported a linear radiolucency in the area of the lateral pleura on chest radiographs and called it a black pleural line.[29] CT demonstrates the inferior and posterior predominance of the lesions with a high concentration of microliths in the subpleural parenchyma and along the bronchovascular bundles. Pleural calcification gives pencil thin sharp dense white lines along the costal surfaces and over the hemidiaphragm. High-resolution CT shows that the so-called black pleural line on chest radiographs is caused by thin-walled subpleural cysts, ranging from 5 to 10 mm in diameter. Hoshino, *et al*. reported for the first time the MRI findings in PAM as diffuse calcific micronodules, characterized by an increased signal intensity on T1-weighted images, predominantly in the posterior lower zones.[30]

Isotopic bone imaging tracer studies can trace the pulmonary uptake of ^{99m}Tc diphosphonate.[24] The diagnosis can be made with confidence from the classic radiographic pattern and the striking radiological and clinical dissociation. Microliths can be identified in sputum, BAL fluid and TBLB specimens as well. Recently 18F-FDG PET in pulmonary alveolar microlithiasis has shown deep tracer uptake in the lower zones of both the lungs suggesting inflammatory pathology.[31]

CO-MORBID ASSOCIATIONS

Cases of PAM have been commonly misdiagnosed and treated as miliary tuberculosis.[32] The case series by Ucan, *et al*. reported that initial diagnosis of miliary tuberculosis was made in 13 cases whereas the real association was noticed in only two cases.[3] In Saudi Arabia, differential diagnosis has also been made with desert lung syndrome and non-professional pneumoconiosis due to inhalation of the desert sand.[33] Association with pulmonary tuberculosis has been reported at least in five cases.[34,35] Other association are milk-alkali syndrome,[36] renal transplant recipient,[37] pericardiac cyst[5] and lymphocytic interstitial pneumonitis.[38]

CALCIFICATION IN EXTRAPULMONARY SITES

Calcification due to PAM has been reported involving extrapulmonary sites. Coetzee reported histological documentation of the presence of microliths in the lung and sympathetic ganglia and radiological evidence of

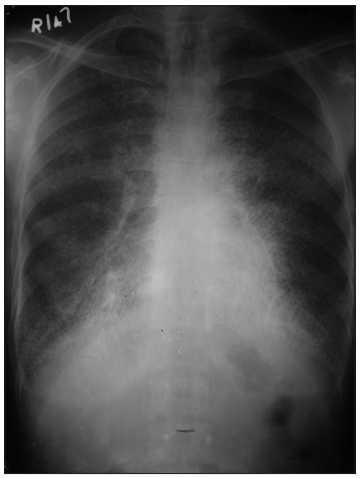

Figure 2: Chest radiograph showing bilateral diffuse dense micronodular opacities

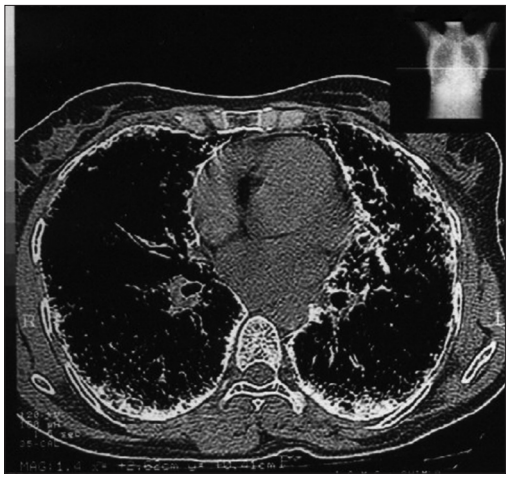

Figure 3: HRCT thorax showing diffuse bilateral calcified fine nodular pattern along with pericardial and pleural calcification

microliths in gonads.[39] Pericardial calcification and nephrolithiasis has also been reported.[40] We described the pleural calcification in one of the cases, which is uncommon in this rare disease and probably the second case reported from India.[12] Calcific deposit of prostate was reported by Castellana[41] while calcification of seminal vesicles by Arslan, *et al*.[42]

DIAGNOSIS

The first diagnostic clue is the characteristic chest radiograph findings. The characteristic chest radiograph and CT findings suggest the diagnosis of PAM. Lung biopsy (trans-bronchial or open) confirm the diagnosis. The characteristic intraalveolar lamellar microliths in the lung tissue establish the diagnosis. The demonstration of microliths in the bronchoalveolar lavage (BAL) fluid is possible. Some young patients lack clinical symptoms and may have normal pulmonary functions, or a slightly decreased diffusing capacity. It may take many years for the progression of the disease to an advanced stage.[4,15,18,24,26] However, extrapulmonary manifestation and complications are uncommon.[4,18]

Routine blood biochemistry including serum calcium concentration, hepatic, renal and parathyroid functions are usually normal. The deterioration of the pulmonary function may be useful to monitor disease activity and disease progression. The identification of the SLC34A2 gene mutation (where available) also suggests the diagnosis.[15,43] Serum concentrations of the surfactant proteins A and D are elevated in the patients with PAM, and can be the markers to monitor the activity and progression of the disease.

DIAGNOSTIC PROCEDURES

Chest radiograph remains the most important tool for diagnosis of PAM. The other diagnostic procedures used to diagnose PAM are open lung biopsy, needle biopsy, transbronchial biopsy and bronchoalveolar biopsy under CT scan guidance. Lung biopsy remains the most definitive procedure for confirmation of PAM. Open lung biopsy may not be feasible in all cases; therefore, transbronchial biopsy remains the most acceptable technique for obtaining tissue biopsy. Four of our cases were confirmed by transbronchial biopsy.

CLINICAL COURSE

There are a few reports with long-term follow-up data for these patients. In a series, 5 patients who were 13-30 years old at the time of their diagnosis, died after a follow-up period of 5-41 years.[25] In another group of 7 patients who were followed for 5-35 years (mean 12.5 years) remained stable.[24,27] Long term (30-45 years) follow-up data of family members, who were 27-44 years old at the time of diagnosis, clearly suggested that the patients of PAM can have a long life after the diagnosis. As noted above, the first daughter of the family died of respiratory failure at the age of 75 and her younger brother who was 27 years old at diagnosis remained stable even at the age of 71.[44] Most of the patients of PAM die of progressive respiratory insufficiency.

TREATMENT

No definite treatment is available. Home oxygen therapy is necessary for the patients with respiratory insufficiency. Systemic corticosteroid and bronchoalveolar lavage have been shown to be ineffective. In general, no therapy has proved beneficial including whole lung lavage,[23,45] although one case report indicated improved oxygenation using nasal CPAP ventilation.[46] Disodium etidronate, which is known to inhibit the micro crystal growth of hydroxyapatite has been used in the dose of 10 mg/kg per day orally for as long as one year with considerable regression of the calcific densities.[19] The therapeutic bronchoalveolar lavage (BAL) is not helpful in PAM unlike pulmonary alveolar proteinosis (PAP). In PAM, there is deposition of calcium-embedded particle in lung interstitium, which does not get dislodged during BAL; however, in PAP the proteinaceous material from the alveolar surface can be collected by BAL. Lung transplantation has been performed in a few patients. Some patients have undergone bilateral sequential lung transplantation or unilateral lung transplantations,[47-49] but their long-term survival is yet to be proved.

CONCLUSION

Pulmonary alveolar microlithiasis is a rare lung disease with an autosomal recessive trait with mutation of the SLC34A2 gene. Characteristic chest radiograph and CT findings along with lung biopsy confirms the diagnosis. There is no effective medical therapy. The long-term prognosis is poor and respiratory failure is the usual cause of death. Need of lung transplantation is warranted at some stage of progression of the disease.

REFERENCES

1. Harbitz E. Extensive calcification of the lungs as a distinct diseases. Arch Intern Med 1918;21:139-46.
2. Tachibana T, Hagiwara K, Johkoh T. Pulmonary alveolar microlithiasis: Review and management. Curr Opin Pulm Med 2009;15:486-90.
3. Ucan ES, Keyf AI, Aydilek R, Yalcin Z, Sebit S, Kudu M, *et al*. Pulmonary alveolar microlithiasis: Review of Turkish reports. Thorax 1993;48:171-3.
4. Mariotta S, Ricci A, Papale M, De Clementi F, Sposato B, Guidi L, *et al*. Pulmonary alveolar microlithiasis: Report on 576 cases published in the literature. Sarcoidosis Vasc Diffuse Lung Dis 2004;21:173-81.
5. Castellana G, Lamorgese V. Pulmonary alveolar microlithiasis. World cases and review of the literature. Respiration 2003;70:549-55.
6. Viswanathan R. Pulmonary alveolar microlithiasis. Thorax 1962;17:251-6.
7. Gayathri Devi HJ, Mohan Rao KN, Prathima KM, Das JK. Pulmonary alveolar microlithiasis. Lung India 2011;28:139-41.
8. Chandra S, Mohan A, Guleria R, Das P, Sarkar C. Bilateral stony lung: Pulmonary alveolar microlithiasis. BMJ Case Rep 2009; 1012,(Abstract) 2009.
9. Hira HS, Singh T, Chowdhary V. Pulmonary alveolar microlithiasis: Role

of transbronchial lung biopsy. J Assoc Physicians India 2000;48:832-3.
10. Malhotra B, Sabharwal R, Singh M, Singh A. Pulmonary alveolar microlithiasis with calcified pleural plaques. Lung India 2010;27:250-2.
11. Singh NK, Gupta A. Pulmonary alveolar microlithiasis. J Assoc Physicians India 1995;43:647-8.
12. Kashyap S, Negi RS, Mohapatra PR. Pulmonary alveolar microlithiasis: A report of five cases from north western Himalayan region of India. Chest 2007;132:587-8.
13. Kashyap S, Kumar M, Pal LS. Pulmonary alveolar microlithiasis in a Himachali woman. J Assoc Physicians India 1996;44:56-7.
14. Lopez-Areal L, Zumarraga R, Gil Turner C, Martín Granizo IF, Vara Cuadrado F, Duque Fraile J. Infantile familial pulmonary alveolar microlithiasis. Description of 5 cases in 2 families. Rev Clin Esp 1965;97:389-95.
15. Takahashi H, Chiba H, Shiratori M, Tachibana T, Abe S. Elevated serum surfactant protein A and D in pulmonary alveolar microlithiasis. Respirology 2006;11:330-3.
16. Sosman MC, Dodd GD, Jones WD, Pillmore GU. The familial occurrence of pulmonary alveolar microlithiasis. Am J Roentgenol Radium Ther Nucl Med 1957;77:947-1012.
17. Corut A, Senyigit A, Ugur SA, Altin S, Ozcelik U, Calisir H, *et al*. Mutations in SLC34A2 cause pulmonary alveolar microlithiasis and are possibly associated with testicular microlithiasis. Am J Hum Genet 2006;79:650-6.
18. Huqun, Izumi S, Miyazawa H, Ishii K, Uchiyama B, Ishida T, *et al*. Mutations in the SLC34A2 gene are associated with pulmonary alveolar microlithiasis. Am J Respir Crit Care Med 2007;175:263-8.
19. Mariotta S, Guidi L, Papale M, Ricci A, Bisetti A. Pulmonary alveolar microlithiasis: Review of Italian reports. Eur J Epidemiol 1997;13:587-90.
20. Volle E, Kaufmann HJ. Pulmonary alveolar microlithiasis in pediatric patients: Review of the world literature and two new observations. Pediatr Radiol 1987;17:439-42.
21. Senyigit A, Yaramis A, Gurkan F, Kirbaş G, Büyükbayram H, Nazaroğlu H, *et al*. Pulmonary alveolar microlithiasis: A rare familial inheritance with report of six cases in a family. Contribution of six new cases to the number of case reports in Turkey. Respiration 2001;68:204-9.
22. Pracyk JB, Simonson SG, Young SL, Ghio AJ, Roggli VL, Piantadosi CA. Composition of lung lavage in pulmonary alveolar microlithiasis. Respiration 1996;63:254-60.
23. Mascie-Taylor BH, Wardman AG, Madden CA, Page RL. A case of alveolar microlithiasis: Observation over 22 years and recovery of material by lavage. Thorax 1985;40:952-3.
24. Prakash UB, Barham SS, Rosenow EC 3rd, Brown ML, Payne WS. Pulmonary alveolar microlithiasis. A review including ultrastructural and pulmonary function studies. Mayo Clin Proc 1983;58:290-300.
25. Moran CA, Hochholzer L, Hasleton PS, Johnson FB, Koss MN. Pulmonary alveolar microlithiasis. A clinicopathologic and chemical analysis of seven cases. Arch Pathol Lab Med 1997;121:607-11.
26. Sumikawa H, Johkoh T, Tomiyama N, Hamada S, Koyama M, Tsubamoto M, *et al*. Pulmonary alveolar microlithiasis: CT and pathologic findings in 10 patients. Monaldi Arch Chest Dis 2005;63:59-64.
27. Prakash UB. Pulmonary alveolar microlithiasis. Semin Respir Crit Care Med 2002;23:103-13.
28. Deniz O, Ors F, Tozkoparan E, Ozcan A, Gumus S, Bozlar U, *et al*. High resolution computed tomographic features of pulmonary alveolar microlithiasis. Eur J Radiol 2005;55:452-60.
29. Felson B. Thoracic calcifications. Dis Chest 1969;56:330-43.
30. Hoshino H, Koba H, Inomata S, Kurokawa K, Morita Y, Yoshida K, *et al*. Pulmonary alveolar microlithiasis: High-resolution CT and MR findings. J Comput Assist Tomogr 1998;22:245-8.
31. Basu S, Shah M, Joshi JM, Lad S. Imaging calcific concretions of pulmonary alveolar microlithiasis with PET: Insight into disease pathophysiology. Clin Nucl Med 2012;37:707-8.
32. Emri S, Coplu L, Selcuk ZT, Sahin AA, Baris YI. Hypertrophic pulmonary osteoarthropathy in a patient with pulmonary alveolar microlithiasis. Thorax 1991;46:145-6.
33. Nouh MS. Is the desert lung syndrome (nonoccupational dust pneumoconiosis) a variant of pulmonary alveolar microlithiasis? Report of 4 cases with review of the literature. Respiration 1989;55:122-6.
34. Petit MA, Hamladji O, Vedrenne C, Haddam T, Cernay J. A special case of pulmonary alveolar microlithiasis with added tuberculous infection. Rev Tuberc Pneumol (Paris) 1968;32:438-47.
35. Lee YC, Milne D, Rea HH. Pulmonary alveolar microlithiasis: An alternative diagnosis to miliary tuberculosis. Aust N Z J Med 1997;27:336-7.
36. Portnoy LM, Amadeo B, Hennigar GR. Pulmonary alveolar microlithiasis. An unusual case (associated with milk-alkali syndrome). Am J Clin Pathol 1964;41:194-201.
37. Richardson J, Slovis B, Miller G, Dummer S. Development of pulmonary alveolar microlithiasis in a renal transplant recipient. Transplantation 1995;59:1056-7.
38. Ratjen FA, Schoenfeld B, Wiesemann HG. Pulmonary alveolar microlithiasis and lymphocytic interstitial pneumonitis in a ten year old girl. Eur Respir J 1992;5:1283-5.
39. Coetzee T. Pulmonary alveolar microlithiasis with involvement of the sympathetic nervous system and gonads. Thorax 1970;25:637-42.
40. Pant K, Shah A, Mathur RK, Chhabra SK, Jain SK. Pulmonary alveolar microlithiasis with pleural calcification and nephrolithiasis. Chest 1990;98:245-6.
41. Castellana G. Pulmonary alveolar microlithiasis. Case reports and clinico-radiological corelations. Arch Med Int 1989;41:33-41.
42. Arslan A, Yalin T, Akan H, Belet U. Pulmonary alveolar microlithiasis associated with calcifications in the seminal vesicles. J Belge Radiol 1996;79:118-9.
43. Ishihara Y, Hagiwara K, Zen K, Huqun, Hosokawa Y, Natsuhara A. A case of pulmonary alveolar microlithiasis with an intragenetic deletion in SLC34A2 detected by a genome-wide SNP study. Thorax 2009;64:365-7.
44. Castellana G, Gentile M, Castellana R, Fiorente P, Lamorgese V. Pulmonary alveolar microlithiasis: Clinical features, evolution of the phenotype, and review of the literature. Am J Med Genet 2002;111:220-4.
45. Palombini BC, da Silva Porto N, Wallau CU, Camargo JJ. Bronchopulmonary lavage in alveolar microlithiasis. Chest 1981;80:242-3.
46. Freiberg DB, Young IH, Laks L, Regnis JA, Lehrhaft B, Sullivan CE. Improvement in gas exchange with nasal continuous positive airway pressure in pulmonary alveolar microlithiasis. Am Rev Respir Dis 1992;145:1215-6.
47. Stamatis G, Zerkowski HR, Doetsch N, Greschuchna D, Konietzko N, Reidemeister JC. Sequential bilateral lung transplantation for pulmonary alveolar microlithiasis. Ann Thorac Surg 1993;56:972-5.
48. Edelman JD, Bavaria J, Kaiser LR, Litzky LA, Palevsky HI, Kotloff RM. Bilateral sequential lung transplantation for pulmonary alveolar microlithiasis. Chest 1997;112:1140-4.
49. Jackson KB, Modry DL, Halenar J, L'abbe J, Winton TL, Lien DC. Single lung transplantation for pulmonary alveolar microlithiasis. J Heart Lung Transplant 2001;20:226.

How to cite this article: Kashyap S, Mohapatra PR. Pulmonary alveolar microlithiasis. Lung India 2013;30:143-7.

Source of Support: Nil, **Conflict of Interest:** None declared.