# Extensive unilateral pulmonary tuberculosis with segmental atresia of principal bronchus

Sir,

Despite being a curable disease, tuberculosis remains a major cause of morbidity and mortality in developing countries.[1] In most people with immunity, the primary lesion undergoes calcification, thus successfully controlling the infection. In people with less effective immune systems, this develops into progressive primary tuberculosis.[2] In such cases, though granuloma formation is initiated, it is unsuccessful in containing the bacilli. The caseous areas liquefy due to effect of proteolytic enzymes released from dead neutrophils.[3]

We recently encountered a patient, who had to undergo left pneumonectomy for a totally destroyed lung that was entirely replaced by cheesy caseous material. This was associated with segmental atresia of principal bronchus. In an immunocompetant individual, extensive caseation, as seen in our case is rare. A literature search showed only a single case resembling ours in which there was extensive caseation involving a single lobe in an 11-year-old child.[4]

Our patient was a 38-year-old female, with complaints of cough with expectoration of 1 year duration. There was no history of chest pain, fever, or hemoptysis. One year back, she was treated for pulmonary tuberculosis for 6 months. Though she was sputum negative after treatment, her symptoms persisted. She was thin built and poorly nourished. Respiratory system examination showed shift of trachea to left, with absent air entry on the left side of chest. Other system examinations were within normal limits. Computed tomography scan of chest showed collapsed left lung, possibly destroyed lung [Figure 1].

Peroperatively, the left lung was fully collapsed and consolidated. Left principal bronchus was atretic, suction tube could be introduced only up to a distance of 0.5 cm. There was no pleural effusion. Postoperative period was uneventful. The pneumonectomy specimen measured 19 × 15 × 15 cm. The pleura was thickened, the hilum showed only blood vessels, and bronchial stump was not seen. Perpendicular serial sections at hilum for a depth of 1 cm were taken, which showed normal well-developed bronchi. The entire lung was replaced by a mass of cheesy caseous material [Figure 2]. No areas of cavitation or fibrosis were seen. Microscopic examination showed extensive areas of acellular necrosis with a few granulomas with Langhans giant cells [Figure 3a and b]. No normal lung tissue was seen. Though histochemical stains for acid fast bacilli were negative, tissue submitted for microbiological examination showed few acid fast bacilli in the concentrated sample, which was subsequently grown in MB/BACT system (BioMerieux). The growth was confirmed as Mycobacterium tuberculosis by polymerase chain reaction [Figure 3c].

Another interesting feature was that, even though the left lung was totally destroyed by the caseous necrosis, the patient had no evidence of disease process elsewhere.

Endobronchial tuberculosis is known to produce bronchial stenosis by destroying the bronchial cartilage and subsequent fibrosis.[5] Our patient had an atretic segment of left principal bronchus distal to which the whole of left lung was replaced by a mass of cheesy caseous material. The atretic segment of bronchus probably succeeded in confining the disease, at the expense of completely

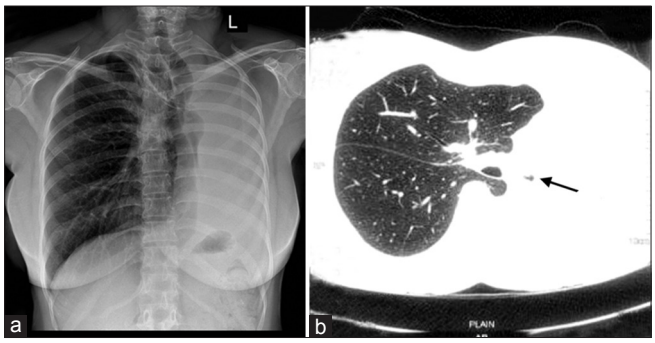


**Figure 1:** (a) X-ray chest showing collapsed left lung, (b) Computed tomography chest shows absent air shadow in left lung with atretic bronchus (arrow)

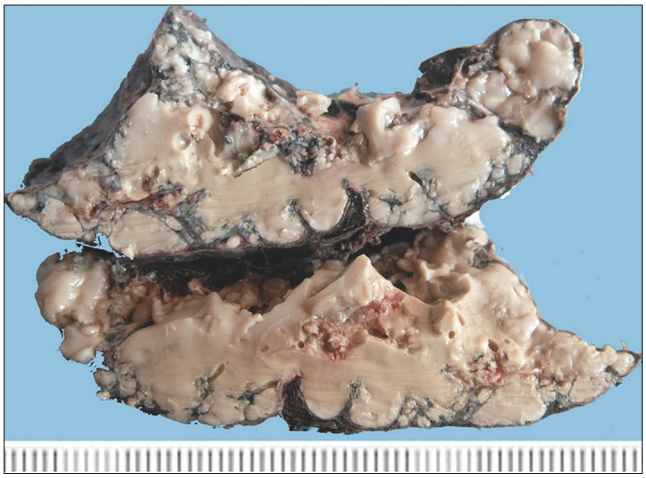

**Figure 2:** Pneumonectomy specimen shows total replacement of lung by cheesy caseous material

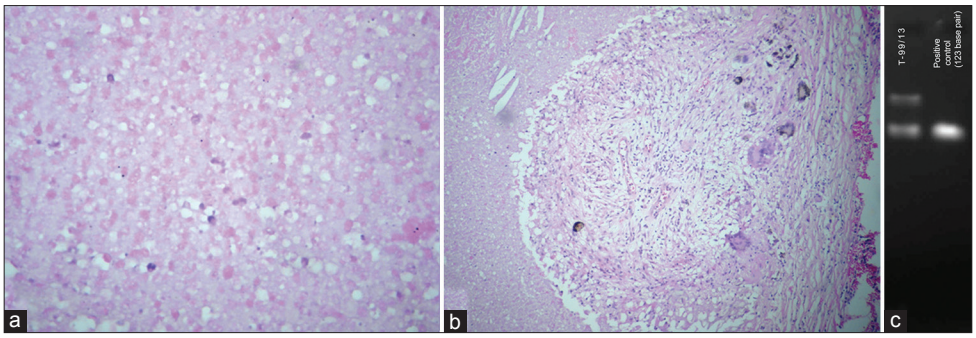


**Figure 3:** (a) Acellular necrotic material, (b) Granuloma with giant cells (both H and E, ×200), (c) Polymerase chain reaction photograph showing positive band for Mycobacterium tuberculosis

destroying it.

Postoperatively, the patient was put on antitubercular treatment for 6 months and is doing well 10 months after her surgery. There is no evidence of the disease process elsewhere.

## ACKNOWLEDGEMENT

We thank Dr. Molly Anthony; Department of Microbiology, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Thiruvananthapuram; for her professional assistance with microbiological evaluation of specimens.

*R. Amita, S. Sandhyamani, M. Unnikrishnan[1]*

*Departments of Pathology, and [1]Cardiovascular and Thoracic Surgery, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Thiruvananthapuram, Kerala, India*
*E-mail: amita.rnair@sctimst.ac.in*

## REFERENCES

1. Knechel NA. Tuberculosis: Pathophysiology, clinical features, and diagnosis. Crit Care Nurse 2009;29:34-43.
2. Ghai OP. Tuberculosis in childhood. In: Ghai OP, Gupta P, Paul VK, editors. Essential Pediatrics, 5th ed. Delhi: Mehta Publishers; 2001. p. 199-205.
3. Maniar BM. Cavitating pulmonary tuberculosis below age of 2 years. Indian Pediatr 1994;31:181-90.
4. Chadha R, Tripati RK, Singh D, Choudhury SR. Extensive unilobar primary pulmonary tuberculosis in an infant: A diagnostic dilemma. J Indian Assoc Pediatr Surg 2005;10:52-4.
5. Sihoe AD, Shiraishi Y, Yew WW. The current role of thoracic surgery in tuberculosis management. Respirology 2009;14:954-68.

**Access this article online**

Quick Response Code:

Website: www.lungindia.com

DOI: 10.4103/0970-2113.126012