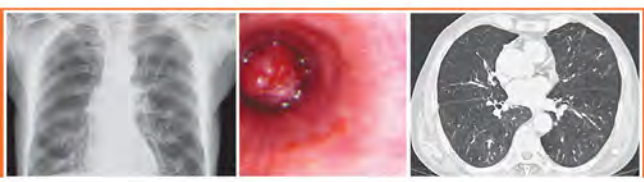
100 Cases in Pulmonary Medicine

Rajendra Prasad
Nikhil Gupta



Co-authors
Harsh Saxena

Foreword
Surinder K Jindal

JAYPEE

CONTENTS

CASE 16

Bilateral Chylothorax in a Young Female Secondary to Tuberculosis

CASE REPORT

A 15-year-old female was admitted to our department with the complaints of breathlessness, and off and on fever for 1 month. She came from a nonendemic zone of filariasis in Uttar Pradesh. The resting pulse rate was 92 beats/min and blood pressure was 112/74 mm Hg. There was no history of contact with a tuberculosis patient. Her physical examination revealed hard, mobile lymph nodes of size 2 × 2 cm approximately, present bilaterally in the cervical region, and pallor. On chest examination, there was stony dull note localized to the bilateral infrascapular, lower axilla. Rest of the systems were within normal limits.

Her blood examination revealed anemia and hypoalbuminemia but normal total and differential leukocyte counts. Her chest X-ray revealed bilateral pleural effusion (**Fig. 1**). The pleural fluid was aspirated (about 2.0 litre from left side and 1.2 litre from right side) on both

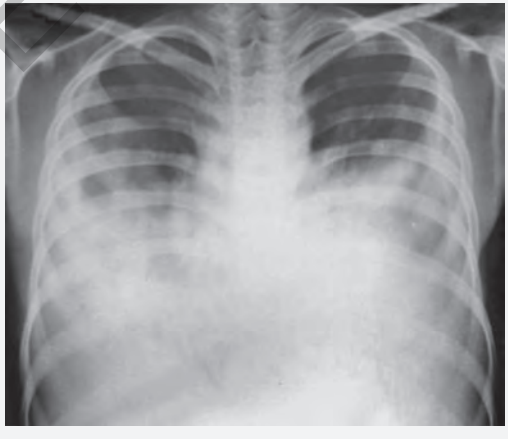


FIG. 1: Chest X-ray revealed bilateral pleural effusion.

sides that revealed a milky white fluid. Then we thought about the chylothorax. Clearing of the pleural fluid by adding ethyl-ether into it leads to the exclusion of pseudochylothorax. The pleural fluid of both sides was sent for examination that revealed, for the right side, protein 4.2 g%, sugar 44 mg%, and total leukocyte count 3,400 cells/mm^3; differential leukocyte count was neutrophils 20, lymphocytes 80, pleural fluid triglyceride 535 mg%, and pleural fluid cholesterol 24.8 mg%; pleural fluid of the left side revealed protein 4.0 g%, sugar 40 mg%, and total leukocyte count 3,280 cells/mm^3; differential leukocyte count was neutrophils 34, lymphocytes 66, pleural fluid triglyceride 188.0 mg%, and pleural fluid cholesterol 24.0 mg% (**Table 1**). Serum triglyceride and serum cholesterol was 72.7 mg% and 71.9 mg%, respectively. The Ziehl–Neelsen stain of the pleural fluid was negative but *Mycobacterium tuberculosis* was isolated on the Lowenstein–Jensen culture. The pleural fluid culture for pyogenic organisms was sterile in nature on both sides. PPD showed no indurations. Her biopsy of the cervical lymph node revealed caseating granuloma, and the Bactec culture for *M. tuberculosis* was also positive in the cervical lymph node biopsy specimen. Her abdomen serum triglyceride 72.7 mg%, serum cholesterol 71.9 mg%, Ziehl–Neelsen stain of the pleural fluid was negative but *M. tuberculosis* was isolated on the Lowenstein–Jensen culture. Biopsy of the cervical lymph node revealed caseating granuloma, and the Bactec culture for *M. tuberculosis* was also positive. Ultrasound also revealed multiple retroperitoneal and para-aortic lymphadenopathy. Thus, a diagnosis of bilateral chylothorax secondary to tuberculosis with cervical lymphadenopathy was established.

To confirm the exact site of the thoracic duct tear, lymphangiography was planned but her parents refuse for further investigations.

She was put on standard 6-month antitubercular treatment: A combination of isoniazid, rifampicin, pyrazinamide, and ethambutol was started for 2 months followed by isoniazid and rifampicin for a further 4 months. Following this, she showed clinical as well as radiological improvement and chylothorax resolved after 2 months of treatment (**Fig. 2**), and on regular follow-up she had no further symptoms.

TABLE 1: Characteristics of the pleural fluid on both sides of the cavity.

Pleural fluid	Right pleural cavity	Left pleural cavity
Protein	4.2 g%	4.0 g%
Sugar	44 mg%	40 mg%
TLC	3,400 cells/mm^3	3,280 cells/mm^3
DLC	P20, L80	P34, L66
Pleural fluid	535 mg%	188 mg%
Pleural fluid cholesterol	24.8 mg%	24 mg%
Pleural fluid culture	Sterile	Sterile

(DLC: differential leukocyte count; TLC: total leukocyte count)

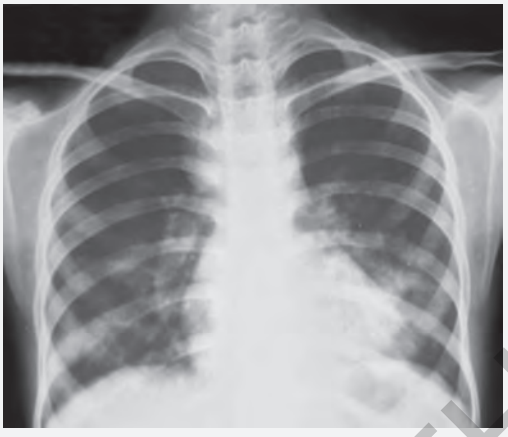


FIG. 2: Revealed radiological clearing of the bilateral chylothorax after 2 months of antitubercular treatment.

DISCUSSION

Chylothorax was recognized in the 17th century but it is still a rare enough entity to be viewed by most physicians as a clinical curiosity. In a historical review by Johsman, Bartolet is credited with the initial description of chylothorax in 1633, and Quincke reported the first case in 1875. Sassion et al. divided the causes of chylothorax after 2 months of antitubercular treatment into four major categories: Trauma, tumor, idiopathic, and miscellaneous. Trauma is the leading cause of chylothorax. This trauma is usually a cardiovascular, pulmonary, or esophageal surgical procedure. Another leading cause of chylothorax is malignancy. The most common malignancy to cause chylothorax is a lymphoma, followed by bronchogenic carcinoma, and rarely leukemia. Very few cases of chylothorax secondary to acute lymphoblastic leukemia (ALL) are reported. The third category of chylothorax is idiopathic including most cases of congenital chylothorax. Most cases of idiopathic chylothorax in adults are probably due to minor trauma such as coughing or hiccupping after the ingestion of fatty meals. The fourth category of chylothorax is the miscellaneous category and causes are thrombosis of superior vena cava or subclavian vein, cirrhosis, lymphangioleiomyomatosis Gorham's syndrome, Kaposi sarcoma, Castleman disease, filirasis and familian lymphedema, sarcoidosis, radiation-induced mediastinal fibrosis, and hypothyroidism.

Tuberculosis is described as a possible cause of chylous effusion, but only a single case, described by Brandt in 1917, appears to have been recorded. Cakir et al. reported the concurrence of chlothorax and endobronchial tuberculosis in a 4-month-old boy.

Grobbelaar et al. reported one case of bilateral and other case of unilateral chylous effusions associated with extensive mediastinal and hilar lymphadenopathy secondary to pulmonary tuberculosis, in children. Deniel et al. reported spontaneous bilateral chylothorax secondary to disseminated tuberculosis complicated by massive pulmonary embolism.

Tan et al. reported a patient with persistent chylothorax and generalized lymphadenopathy who was subsequently diagnosed to have concurrent tuberculosis and malignant lymphoma. Very few cases of bilateral chylothorax have being reported in the literature. Chylothorax has no predilection for age and sex. Symptoms of chylothorax mostly depend upon the amount of fluid in the pleural cavity.

The exact pathogenesis for the development of chylothorax secondary to tuberculosis remains controversial. Fraser et al. and Yunis et al. reported that the enlarged lumber and iliac group of lymph nodes produced obstruction of the cisterna chyli and thoracic duct, as a result of which there was dilatation of the lumbar channels; this was followed by the opening up of collateral anastomoses, many lymphaticovenous anastomosis existing between the thoracic duct system and the azygos, intercostal, and lumbar veins. The increased pressure in the system resulted in the transudation of chyle into the pleural space. Grobbelaar et al. reported that the possible explanation for the development of a chylothorax in our patients is the obstruction of the thoracic duct by tuberculous lymphadenopathy with subsequent increase in pressure in the surrounding lymphatic system and leaking of chyle into the pleural space.

Best way to establish the diagnosis of chylothorax is to determine the concentration of triglycerides in the pleural fluid. The triglyceride concentration >110 mg/dL (in our case it was 289.3 mg/dL), a ratio of pleural fluid to serum triglycerides of >1.0 (in our case it was 3.77), and a ratio of pleural fluid to serum cholesterol of >1.0 (in our case it was 0.344) usually confirm chylothorax. Chylothorax will be excluded if the pleural fluid triglyceride concentration is >50 mg/dL. However, in the case of levels from 50 to 110 mg/dL, a lipoprotein analysis of the pleural fluid should be performed, and the demonstration of chylomicrons in the fluid confirms the diagnosis of chylothorax (**Table 2**).

Primary treatment in the case of chylothorax should be directed toward the correction of malnutrition and compromised immunologic status which is due to repeated pleural fluid aspirations of chyle with its high levels of protein, fat, electrolytes, and lymphocytes. The defect in the thoracic duct often closes spontaneously in the case of traumatic injury. In the case of severe dyspnea, the placement of the pleuroperitoneal shunt or chest tube drainage is mandatory. If the chylothorax persists for >4 weeks, consideration should be given to surgical exploration with ligation of the thoracic duct.

In our case, diagnosis of chylothorax was established on typical pleural fluid color, high pleural fluid triglyceride level, high ratio of pleural fluid to serum triglyceride, and low ratio of pleural fluid to serum cholesterol. She responded well to antituberculous treatment.

TABLE 2: Differential points of chylothorax, pseudochylothorax, and empyema, and parental nutrition entering the pleural space via the subclavian line.

Parameter	Chylothorax	Pseudochylothorax	Empyema thoracis	Parental nutrition entering the pleural space via the subclavian line
Definition	Presence of chyle in the pleural cavity; chyle contain chylomicrons, triglycerides, and lymphocytes*	Caused by high-lipid levels (cholesterol/lecithin–globulin complexes) in the pleural fluid	Pleural effusion due to bacterial pneumonia	Presence of high triglyceride levels in the pleural cavity
Color	Milky white	Milky white	Milky white	Milky white
Odor	Odorless	Odorless	Foul smelling	Odorless
Clinical onset	Acute	Chronic	Acute	Acute
Most common cause			Bacterial pneumonia	
On centrifugation	Remain opalescent	Remain opalescent	Supernatant part clear	Remain opalescent
Diagnostic criteria • PF TG • PF Cho/serum Cho ratio • Pleural fluid culture • PCR of the pleural fluid • Addition of 1–2 mL of ethyl ether to the pleural fluid	 >110 mg/dL <1.0 Usually negative Usually negative Remain opalescent Ingestion of a fatty meal with lipophilic dye (drug and cosmetic green no. 6, a coal tar dye), followed by thoracocentesis 30–60 min later and if color changed to green fluid, then it also confirms chylothorax	 >1.0 Usually negative Usually negative Clears as cholesterol dissolved	 Usually negative, even in the case of frank pus in cavity Most specific to detect DNA of various bacterial populations	 >110 mg/dL Remain opalescent The presence of chylomicrons in the lipoprotein electrophoresis profile could be traced back to the lipofundin component of parental nutrition

(PF TG: pleural fluid triglyceride; PF Cho/serum Cho ratio: pleural fluid cholesterol/serum cholesterol ratio)

*In the case of levels 50–110 mg/dL, a demonstration of chylomicrons in lipoprotein analysis confirms chylothorax. Levels below 50 mg/dL virtually exclude chylothorax.

FURTHER READING

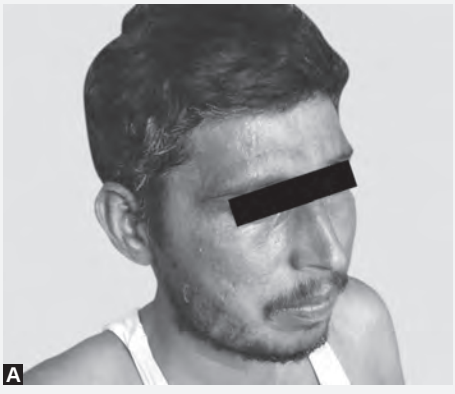
1. Kant S, Verma SK, Anand SC, Prasad R, Verma RK. Development of bilateral chylothorax in a younger female secondary to tuberculosis. Lung India. 2011;28:56-9.
2. Anton PA, Rubio J, Casan P, Franquet T. Chylothorax due to *Mycobacterium tuberculosis*. Thorax. 1995;50:1019.
3. Sassoon CS, Light RW. Chylothorax and pseudochylothorax. Clin Chest Med. 1985;6:163-71.
4. Valentine VG, Raffin TA. The management of chylothorax. Chest. 1992;102:586-91.
5. Menon BS, Juraida E, Mahfuzah M, Hishamshah I. Bilateral chylothorax in paediatric acute lymphoblastic leukaemia. Br J Haematol. 2006;132:253.
6. Kushwaha RAS, Prasad R, Verma SK, Mehra S. Bilateral chylothorax secondary to acute lymphoblastic leukaemia(ALL). Indian J Med Paediatr Oncol. 2007;28:42-3.
7. Kushwaha RAS , Verma SK, Mahajan V, Prasad R. Bilateral chylothorax in a child after mild trauma-a case report. Indian J Chest Dis Allied Sci. 2008;50:355-7.
8. Brandt M. Ein Beitrag zur Kasuistik der Ductus thoracicus-uberkulose und des Chylothorax. Inaug. Diss. Heidelberg: Universitats-Buchdruckerei von J Horning; 1917.
9. Cakir E, Gocmen B, Uyan ZS, Oktem S, Kiyan G, Karakoc F, et al. An unusual case of chylothorax complicating childhood tuberculosis. Pediatr Pulmonol. 2008;43:611-4.
10. Grobbelaar M, Andronikou S, Goussard P, Theron S, Mapukata A, George R. Chylothorax as a complication of pulmonary tuberculosis in children. Pediatr Radiol. 2008;38:224-6.
11. Browse NL, Allen DR, Wilson NM. Management of chylothorax. Br J Surg. 1997;84:1711-6.

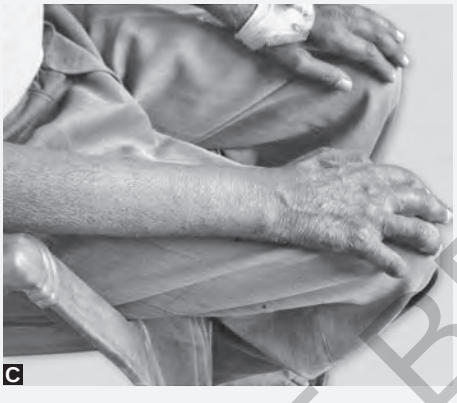
CASE 17

Concomitant Pulmonary Tuberculosis and Borderline Leprosy with Type-II Lepra Reaction in Single Patient

CASE REPORT

A 34-year-old, nonsmoker male was admitted, in September 2007, to the Department of Pulmonary Medicine, as a follow through case of borderline lepromatous leprosy. He had complaints of breathlessness, loss of appetite with cough with expectoration for last two and half months and skin lesions over face, forearm, and dorsum of hands with exfoliative skin lesions over forearm for last 2 months and recurrent hemoptysis for last 25 days. He was on medication, tablet dapsone 100 mg daily, tablet clofazimine 500 mg daily and capsule rifampicin 600 mg, once a month, for the last 9 months. He had also taken oral prednisolone for >3 months duration about 6 months back for neurological complications. There was no past history of tuberculosis as per information provided by the patient. Clinical examination revealed multiple hypopigmented skin lesions varying in size from 2 to 4 cm over the trunk with thickened left ulnar nerve and nodular lesions over face, forearm and dorsum of hands (**Figs. 1A** and **B**) and exfoliative skin lesions over forearm (**Fig. 1C**). His resting pulse rate was 102 beats/min and blood pressure was 112/74 mm Hg and his respiratory rate was 26 breaths/min. His general examination revealed no significant abnormality. His respiratory system examination revealed coarse crepts localized to left infraclavicular and axillary areas. Initially he was put on symptomatic treatment for hemoptysis. Subsequently, his chest X-ray showed a large cavity with a mass filled opacity confined to left upper zone and fibrotic changes in right upper zone (**Fig. 2**). Computed tomography of thorax revealed fibro-consolidation with cavitation in the anterior segment of left upper lobe, fibrotic nodule in both anterior and posterior segments of right the upper lobe (**Fig. 3**). Thus, a possibility of aspergilloma with other possibilities was raised. The clinical examination of the rest of the system revealed no abnormality. His blood biochemistry revealed total leukocyte count: 10,200/mm^3, differential leukocyte count: P 66%, L 34%. His purified protein derivative (PPD) was 2,219 mm induration. His sputum for acid fast bacilli (AFB) on three consecutive days was positive. Thus, a diagnosis of pulmonary tuberculosis was confirmed. For leprosy, patient consulted the skin department, was diagnosed with borderline lepromatous leprosy (on the basis of slit skin smear) with type-II lepra reaction (biopsy of nodular lesion revealed diffuse sheets of foamy macrophages centered around adnexal structures with heavy infiltration of lepra bacilli suggestive of erythema nodosum leprosum). He was advised to stop dapsone and





FIGS. 1A TO C: (A and B) Revealed skin lesions over face, forearm, and dorsum of hands due to type-II lepra reaction. (C) Revealed exfoliative skin lesions over forearm due to type-II lepra reaction. ***(For color version, see Plate 2)***

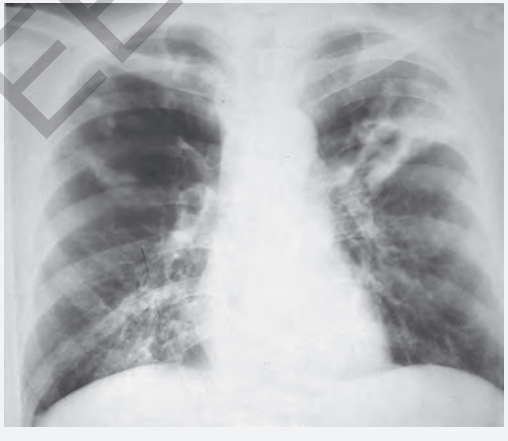


FIG. 2: Chest X-ray reveals a large cavity with a mass filled opacity confined to left upper zone and fibrotic changes in right upper zone.

started oral prednisolone, thalidomide, and clofazimine. Thus, a final diagnosis of borderline leprosy with type-II lepra reaction with concomitant pulmonary tuberculosis was made. The patient was referred to DOTS clinic and started on Category I treatment. The oral prednisolone was subsequently tapered and stopped while the other antileprotic drugs continued. The patient's general condition improved and was on regular follow-up.

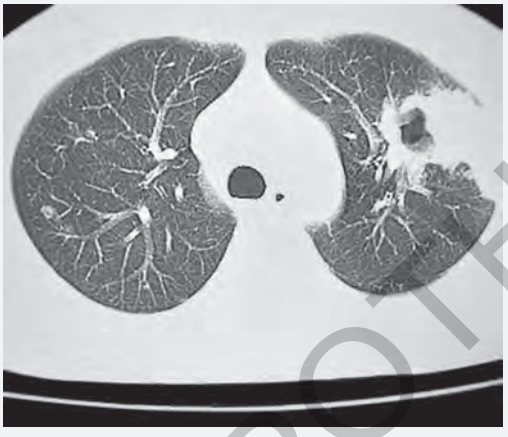


FIG. 3: Computed tomography of thorax revealed fibro-consolidation with cavitation in anterior segment of left upper lobe with fibrotic nodule in anterior and posterior segment of right upper lobe.

DISCUSSION

Leprosy and tuberculosis continue to be prevalent in our country. Prevalence of tuberculosis is estimated to be 4.0 and 16.0/1,000 for bacteriologically and radiologically active tuberculosis cases respectively, while the national prevalence rate of leprosy in India is 0.88/10,000. The great attention about leprosy and tuberculosis coinfection was carried out by Chaussinand in 1948, and concluded that the prevalence of leprosy was inversely related with the prevalence of tuberculosis. In some leprosy communities, however, tuberculosis appears to be quite common. This may be the result of a small group of people unable to defend against either organism. Leprosy has failed to return in areas where tuberculosis has been controlled, but this may be because other conditions have changed (e.g., because of the introduction of Bacilli Calmette–Guérin). The principal means of transmission of both leprosy and tuberculosis is by aerosol spread. The incubation period in leprosy varies from 6 months to 40 years or longer, while in case of tuberculosis it is only 4 weeks. Review of literature suggest that the occurrence of leprosy and tuberculosis coinfection first time reported by

Relvich AL et al. in 1954 and strongly argued that association of tuberculoid form of leprosy with tuberculosis was uncommon. Gajwani et al. in 1968 and Gupta et al. in 1971, reported the association of tuberculoid type of leprosy with tuberculosis. This was further supported by Agnihotri MS et al. in 1974, who documented three cases of tuberculoid leprosy with tuberculosis and Nigam P et al. (1979), who documented two cases of tuberculoid leprosy in association with tuberculosis. On the other hand, most of the cases of tuberculosis were associated with lepromatous leprosy followed by borderline lepromatous leprosy, as observed in present case also. The duration of gap between the development of leprosy and tuberculosis varied between two months to 10–15 years, the study with largest data showed gap duration of about 10–15 years, where duration of tuberculosis in most of the cases was within 6 months (while in present case it was 11 months). Only two cases of tuberculosis were found to occur earlier than leprosy whereas one study concluded that tuberculosis can occur during full spectrum of leprosy. It is well known that tuberculosis infection can develop with certain risk factors such as HIV infection, low socioeconomic status, silicosis, diabetes mellitus, gastrectomy, renal failure, organ transplant; these have been incorporated in the risk stratification in tuberculosis guideline (no such type of risk factor present in present case). There are also other purporated risk factors such as smoking, rheumatic disorders, and use of low dose immunosuppressive agents or glucocorticosteroids but substantive epidemiological data about these risk factors of tuberculosis are scarce. In case of leprosy, corticosteroids are used primarily in the treatment of type I (reversal) reactions and type II reactions and silent neuropathy. Chandrashekhar et al. (2000) reported development of pulmonary tuberculosis after corticosteroid intake in two cases of leprosy. Agarwal et al. (2000), reported a case of leprosy and tuberculosis coinfection in a patient of renal transplant recipient and who had taken prednisolone, azathioprine, and cyclosporine for >9 years (while in the present case, he received corticosteroid for >3 months). In leprosy, majority of the cases reported were pulmonary tuberculosis while in two cases of extrapulmonary tuberculosis (tuberculosis of larynx and cutaneous tuberculosis) were reported. Diagnosis of leprosy was established in majority by slit skin smear but diagnosis by nasal smear and histopathological examination was also reported (while in present case by slit skin smear and biopsy also). Most common findings on chest radiographs were bilateral infiltrates (as in the present case). Sputum smear for AFB was positive in majority of the cases with available data (as in present case also). Available data among three cases of leprosy with tuberculosis had lepra reaction including one of the author, in which one of them had type-II lepra reaction (ENL) while one was type-I reversal reaction (while in present case it was type-II lepra reaction). Management of tuberculosis in leprosy coinfection does not change; with the same WHO treatment categorization, i.e., Cat I, Cat II or Cat III. The detailed features of all the cases of tuberculosis and leprosy coinfection and their comparison with present case are summarized in **Tables 1** and **2**.

TABLE 1: Comparative analysis of leprosy-tuberculosis coinfection reported by various authors.

Details	Authors						
	Gupta MC (1971)	Vinik LA (1971)	Agnihotri MS et al. (1974)	Bhargava NC (1976)	Nigam P et al. (1979)	Gatner EM (1980)	Kumar B et al. (1982)
No. of cases	NA	NA	3	NA	20	NA	9
Gap between development of leprosy and dev. of tuberculosis	NA	NA	NA	NA	10–15 years duration of tuberculosis in most of them was within 6 months	NA	NA
Types of leprosy	Tuberculoid leprosy	NA	Tuberculoid leprosy	NA	15 were of lepromatous. 3 of dimorphous and 2 of tuberculoid leprosy	NA	NA
Is tuberculosis developed first	NA	NA	Only in one case	NA	NO	NA	Tuberculosis was found to occur throughout leprosy spectrum
Is leprosy developed first	NA	NA	NA	NA	YES	NA	NA
Family history of tuberculosis	NA	NA	NO	NA	NA	NA	NA
Any risk factors like use of corticosteroids/diabetes mellitus/ smoking/HIV/others were present	NA	NA	NO	NA	NA	NA	NA
TB: Pulmonary/extrapulmonary	Pulmonary	NA	Pulmonary	Pulmonary	Pulmonary	NA	Radiological evidence of tuberculosis found
Descriptions: Lesions of tuberculosis on chest radiographs	NA	NA	Bilateral infiltrates	NA	Bilateral extensive pulmonary lessons were seen in 14 cases	NA	NA
Sputum for AFB	NA	NA	Positive in all of cases	NA	Positive in 80% of cases	NA	NA
Diagnosis of leprosy	NA	NA	NS	NA	NA	NA	NA
Lepra reaction	NA	NA	NS	NA	NA	NA	NS

(NA: data not available; NS: not seen; TB: tuberculosis)

TABLE 2: Comparative analysis of leprosy-tuberculosis coinfection reported by various authors.

Details	Authors						
	Flanagan PM et al. (1993)	Agarwal DK et al. (2000)	Srilakshmi MA et al. (2003)	Lee HN et al. (2003)	Chandrashekhar T et al. (2007)		Present author
No. of cases	NA	1	1	1	2		1
					Case-I	Case-II	
Gap between development of leprosy and development of tuberculosis	NA	2 months	10 year	NA	3 months	2 year	11 months
Types of leprosy	LL	LL	LL	BL	BL	LL	BL
Is tuberculosis developed first	NA	YES	NO	NA	NO	NO	NO
Is leprosy developed first	NA	NO	YES	NA	YES	YES	YES
Family history of tuberculosis	NA	NO	NO	NA	NA	NA	NA
Any risk factors like use of corticosteroids/diabetes mellitus/smoking/HIV/others were present	NA	Renal transplant recipient and had taken prednisolone, azathioprine, and cyclosporin	NO	NA	Corticosteroids	Corticosteroids	Corticosteroids
TB: Pulmonary/extrapulmonary	TB of larynx	Pulmonary	Pulmonary	NA	Pulmonary	Pulmonary	Pulmonary
Descriptions lesions of tuberculosis on chest radiographs	NA	NA	Bilateral infiltrates	NA	Bilateral infiltrates	Bilateral infiltrates	Bilateral infiltrates
Sputum for AFB	NA	+ve and culture +ve	+ve	NA	+ve	+ve	+ve
Diagnosis of leprosy	NS	Biopsy	Slit skin smear				Slit skin smear
Lepra reaction	NA	Seen	NO	Type-I	NO	Type-II	Type-II

(NA: data not available; NS: not seen; TB: tuberculosis)

100 Cases in Pulmonary Medicine

Salient Features

- Compilation of interesting cases in Pulmonary Medicine in day-to-day clinical practice which are challenging and educative.
- Each case follows a step-by-step approach including history, salient findings on physical examination, and relevant investigations for diagnosis.
- Each case has lot a of radiological images, which makes reading extremely interesting.
- Plethora of cases including interesting tuberculosis, multidrug resistant (MDR), and extensively drug-resistant (XDR) cases. Diffuse parenchymal lung diseases, thoracic malignancies, and allergic bronchopulmonary aspergillosis.
- Rare cases such as pulmonary sequestration, alveolar microlithiasis, endobronchial polyp, carcinoid syndrome, Chilaiditi syndrome, and lymphotracheal fistula.
- The book is for resourceful both undergraduate and postgraduate medical students, general physicians, and consultants in pulmonary medicine and allied branches interested in diagnosis and treatment of pulmonary diseases.

Rajendra Prasad MD DTCD FAMS FCCP (USA) FRCP (GLASGOW) FNCCP FICS FCAI FIAB FIMSA FCCS DSc (Honoris Causa) is currently working as the Director of Medical Education and Head, Department of Pulmonary Medicine, Era's Lucknow Medical College and Hospital, Era University, Lucknow, Uttar Pradesh, India. He is the Former Director, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi, India; Former Professor and Head, Department of Pulmonary Medicine, King George's Medical University, Lucknow, Uttar Pradesh, India; and Former Director, UP Rural Institute of Medical Sciences and Research, Saifai, Etawah, Uttar Pradesh. He did his MBBS in 1974 and MD in 1979 from King George's Medical College, Lucknow, Uttar Pradesh, India. He has been an International Governor of American College of Chest Physicians, USA. He has a unique distinction of being President of all major scientific bodies in the field of pulmonary medicine in India. He was awarded Fellowship of the National Academy of Medical Sciences, India, American College of Chest Physicians, USA, and Royal College of Physicians and Surgeons, Glasgow, UK. He has supervised about 180 researches, and published 340 original articles, reviews, and book chapters. He also authored 8 books including 4 books on tuberculosis and an Atlas of Fiber Optic Bronchoscopy based exclusively on Indian patients. He has presented over 1,600 guest lectures and scientific papers at various national and international meetings. Apart from being a clinician par excellence, he is also a very popular medical teacher in pulmonary medicine. He is recipient of Dr. B.C. Roy National Award for devolving and popularizing Pulmonary Medicine in India. He has been mentor of many students who are now assuming important positions in pulmonary medicine.

Nikhil Gupta MBBS MD (Medicine) Diploma in Allergy and Asthma (CMC Vellore), Distance Fellowship in Diabetes (CMC Vellore), FCCS did his MBBS in 2002 with honors in biochemistry and MD Internal Medicine in 2006. He served as Assistant Professor, Department of Medicine, Era's Lucknow Medical College and Hospital, Lucknow, Uttar Pradesh, India from 2012 to 2017 and is currently working as an Assistant Professor, Department of General Medicine, Dr Ram Manohar Lohia Institute of Medical Sciences, Lucknow, Uttar Pradesh, India. He has presented more than 30 guest lectures and scientific papers. He has supervised 15 researches and published 35 original articles, review articles, case reports, and book chapters. He has also co-authored 3 books on tuberculosis.

Available at all medical bookstores
or buy online at www.jaypeebrothers.com

JAYPEE BROTHERS
Medical Publishers (P) Ltd.
www.jaypeebrothers.com

Join us on facebook.com/JaypeeMedicalPublishers

Shelving Recommendations
MEDICINE

ISBN 978-93-90020-09-6