ASIAN JOURNAL OF PHARMACEUTICAL AND CLINICAL RESEARCH

INNOVARE ACADEMIC SCIENCES
Knowledge to Innovation

Vol 18, Issue 4, 2025

Online - 2455-3891
Print - 0974-2441

Case Report

TUBERCULOSIS BEYOND THE ORDINARY: A CASE SERIES INVESTIGATING PULMONARY AND EXTRA-PULMONARY PRESENTATIONS

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Received: 06 February 2025, Revised and Accepted: 18 March 2025

ABSTRACT

Objective: Despite advancements in diagnostic modalities and treatment strategies, diagnosing and managing atypical cases of pulmonary and EPTB, which can affect multiple organ systems, present unique challenges. We present a case series highlighting diverse presentations of TB, emphasizing diagnostic dilemmas and management approaches.

Methods: We conducted a retrospective analysis of patients diagnosed with tuberculosis at our tertiary care center over a specified period. Clinical presentations, diagnostic evaluations, treatment regimens, and outcomes were systematically reviewed.

Results: We present seven cases illustrating the heterogeneity of TB manifestations. These include tuberculosis of the pleura, tuberculoma, bone marrow tuberculosis, and metastasis. Each case presented distinct diagnostic challenges, such as differentiating TB from other pathologies and identifying suitable diagnostic modalities. Management strategies involved anti-tubercular therapy tailored to the site and extent of disease, often supplemented by adjunctive measures.

Conclusion: Our case series underscores the varied clinical presentations and diagnostic complexities of pulmonary and EPTB. Early recognition, a high index of suspicion, and multimodal diagnostic approaches are crucial for timely initiation of appropriate treatment. Further research is warranted to elucidate optimal diagnostic algorithms and therapeutic interventions for pulmonary and EPTB, particularly in resource-limited settings.

Keywords: Tuberculosis, Pleural effusion, Extrapulmonary tuberculosis, Diagnosis, Management, Case series.

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INTRODUCTION

Tuberculosis (TB), an airborne infection, caused by Mycobacterium tuberculosis that can affect any organ but usually affects the lungs. PTB is responsible for 63% of TB cases worldwide, but EPTB is responsible for at least 17% [1]. Although the lymph node is the most often infected location of EPTB, other sites of infection include the pleura, urogenital tract, bones and joints, meninges, central nervous system, gut and/or peritoneum, pericardium, and skin [2].

In 2022, an estimated 1.30 million persons (95% UI: 1.18–1.43 million) died from tuberculosis globally, including 167 000 HIV-positive individuals. Twenty-nine percent of these deaths occurred in India alone. When 24.2 lakh cases were reported in 2022, a record high that indicates an increase of more than 13% from 2021, India's TB surveillance efforts achieved a major milestone [1].

An efficient strategy for detecting, diagnosing, and treating pulmonary and EPTB is still difficult to develop despite the presence of important risk factors. The primary cause of this is the nature of EPTB, which might resemble other prevalent diseases [3-6]. In addition, different clinical manifestations of EPTB make it challenging to collect a pertinent test sample for diagnostic analysis [7]. Due to a high degree of clinical suspicion, the diagnosis of EPTB remains challenging. This is exacerbated by differences in clinical expertise across practitioners, restricted access to diagnostic resources, and a lack of management experience. The diagnosis and treatment of EPTB are significantly hampered by each of these issues.

CASE PRESENTATION

Case 1: Tuberculosis of the pleura

The first case presentation involves a 55-year-old male plumber who presented to the hospital with a 24-h history of hiccups. Any symptoms of a cough, fever, chest pain, hemoptysis, or night sweats were denied by the patient. In addition, he complained of headache and fatigue. He had no significant past medical or surgical history, was not taking any medications, and had no history of smoking, alcohol use, or illicit drug abuse.

On physical examination, the patient's temperature (37°C) was within the normal range. There was stone dullness on percussion in the right lower lobe of the lung, as well as decreased expansion of the chest wall on the right side. The right lower lobe's air entry was nonexistent, according to auscultation.

Subsequent analysis revealed higher PT, INR, and a modest increase in platelet count (Table 1). An X-ray of the chest showed a right-sided pleural effusion (Fig. 1). Although the pleural fluid analysis revealed an exudative effusion, the sputum ZN Stain and pleural fluid CBNAAT tests were negative for tuberculosis (Table 2). In addition, a substantial right side pleural effusion was seen on the USG abdomen. The procedure was pleurocentesis (Fig. 2). After 5 days of intravenous Augmentin 1.2 g (Amoxycillin 1000 mg and Clavulanic Acid 200 mg) twice a day, the patient did not improve.

The patient was then put on regular anti-tuberculous medication after being diagnosed with pleural tuberculosis. A 2-month intensive phase and a 4-month maintenance phase comprised this regimen. The patient was given a combination of oral medications, including isoniazid, pyrazinamide, ethambutol, and rifampicin, throughout the intense phase. Rifampicin and isoniazid were used throughout the maintenance period. To avoid isoniazid-induced peripheral neuropathy, pyridoxine supplements were administered during the course of treatment. The patient was followed up closely throughout the treatment regimen and showed improvement and was treated.

Table 1: Blood investigation

Parameters	Values
Haemoglobin	11.7gm/dl
Total White blood cell	7250/cmm
platelet	557000/cmm
Sr. Creatinine	1
ALT (SGPT)	20
SGOT	21
ALP	73
HIV	Negative
HBsAg	Negative
HCV	Negative

Table 2: Pleural fluid analysis

Parameters	Values
Quantity	1ml
Colour	Reddish yellow
Sugar	79mg/dl
Protein	5.9gm/dl
Quantity	1ml
Colour	Reddish yellow
Transparency	Turbid
Total count	348/cmm
Polymorph	30%
Lymphocytes	70%
RBC	Numerous/hpf
ADA	65.45
Other cells	Occasional mesothelial cells noted in %

Case 2: Tuberculoma

A 49-year-old man arrived at the outpatient clinic complaining of deteriorating vision for the previous 15 days. He had never had a high-grade fever, hemoptysis, cough, or night sweats before, and he had never had surgery. However, he had a history of diabetes mellitus for the last 2 years and hypertension for the previous 5–6 years. He was only taking oral medicine for his hypertension, not his diabetes.

On physical examination of the lungs, bilateral air entry was present, and no dullness was noted on auscultation. Ophthalmological examination of the right eye revealed restriction in superior gaze on dilatation of pupils.

Further blood investigations (Table 3) showed a slight elevation in white blood cell count, high levels of blood urea, a slight elevation of C-reactive protein, and a high level of HbA1c. Sputum culture revealed no growth, and sputum smear results revealed no acid-fast bacilli. An X-ray of the chest (Fig. 3) showed a few small calcifications in both lung lobes. Mycobacterium tuberculosis was not detected by CSF examination (Table 4) for CBNAAT; nevertheless, chemical analysis indicated a small increase in glucose and more than double the value of microproteins. Furthermore, the 2D echocardiography showed a normal ejection fraction. But, it discovered cardiac anomalies, such as minor tricuspid regurgitation, moderate aortic stenosis, a calcific bicuspid aortic valve, and mild aortic regurgitation.

MRI of the brain (plain + contrast) (Fig. 3) revealed a ring-enhancing lesion involving the left paramedian thalamus, which appeared hypointense on T2W imaging with a surrounding hyperintense rim. Mild perifocal edema was observed on FLAIR imaging, and the lesion measured 9.2×4.8×7.8 mm. In addition, abdominal triple-phase CECT showed (Fig. 4a and b), (1) tiny subpleural nodules in the anterior segment of the right upper lobe, appearing nonspecific; (2) a few small calcified mediastinal lymph nodes, likely due to old infective etiology; (3) a small wall defect in the supraumbilical region with herniating preperitoneal fat, suggestive of a small supraumbilical hernia; and (4) a few small calcified lymph nodes in the mesentery in the supraumbilical region, also likely due to old infective etiology.

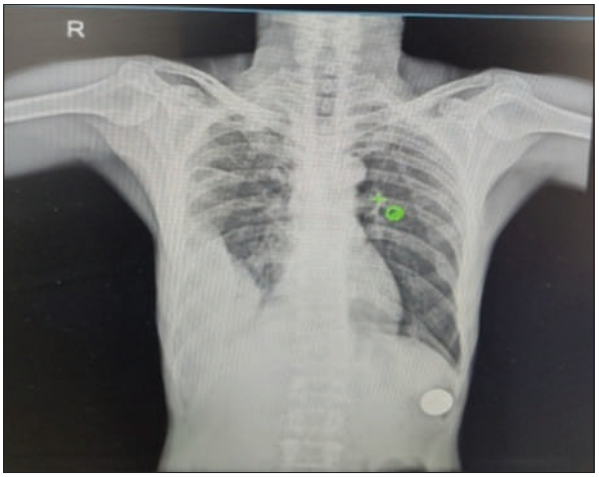

Fig. 1: Before Pleurocentesis (Right sided pleural effusion)

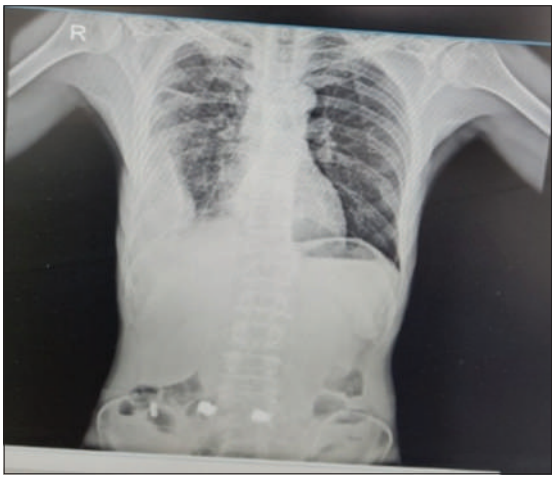

Fig. 2: After Pleurocentesis

Case 3: Bone marrow tuberculosis

A 33-year-old male patient arrived at the hospital complaining of shortness of breath for the past 2 weeks and fever persisting for 10 days. He had no chronic medical conditions or recent surgical history. The patient had a similar episode 2 weeks ago and received treatment at a primary health center. However, his condition worsened, prompting transfer to a district hospital where He was found to have malaria caused by Plasmodium vivax. He remained in the ICU for 2 weeks, receiving 10–12 L of O_2 support to maintain a saturation of 96%. Due to undisclosed reasons, against medical advice, the patient left and was subsequently admitted to a private hospital. On admission, his saturation of oxygen was 94% with 10 L of O_2 support, and he was placed in the ICU.On physical examination, the patient appeared mildly pale and non-icteric. He had a fever with a temperature of 39.4°C, and bilateral crackles/rales were noted on auscultation. A cardiovascular examination produced normal results, with blood pressure at 134/84 mmhg, pulse at 96/min and RBS at 139 mg/dl on admission. Abdominal examination showed a soft, non-tender abdomen with mild hepatosplenomegaly. Neurological examination was normal, and ophthalmic fundus examination was unremarkable.

A complete blood count (Table 5) revealed pancytopenia, and the C-reactive protein level was elevated. Blood and urine cultures showed no growth, and renal function and prothrombin time were normal. Liver enzymes were elevated (ALP, SGOT, and SGPT), but serum bilirubin levels were normal. CSF examination showed normal sugar and protein levels, with sterile culture. G-6-PD levels were normal, and the Mantoux test was negative. A 2D echocardiogram revealed an ejection fraction

Table 3: Blood investigation

Parameter	Values
Haemoglobin	14.7 g/dl
WBC	15140/cmm
Platelet	194000/cmm
Blood urea	25 mg/dl
Sr. Creatinine	1.5 mg/dl
CRP	1.5 mg/dl
HbA1C	11.3%

Table 4: CSF examination

Parameter	Values
ADA	1.12IU/L
Glucose	82mg/dl
Protein	96.30gm/dl

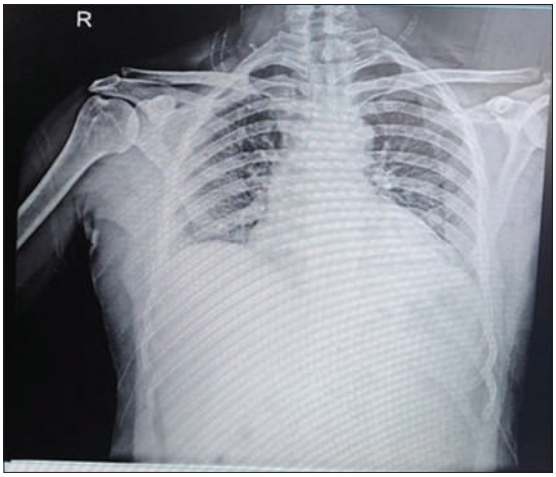

Fig. 3: Chest X Ray (few tiny calcifications in both lobes)

(LVEF) of 55% with mild mitral, aortic, and tricuspid regurgitation. Chest X-ray (Fig. 5) performed at time of admission show acute respiratory distress syndrome (B/L diffuse patchy alveolar opacities). Hepatitis profile and HIV serology were negative.

Initially, the patient received injectable Zostum 1.5 g (Cefoperazone 1000mg+Sulbactam 500 mg) and injectable steroid Dexamethasone (2 ml) once daily. However, his fever persisted, prompting a switch to injectable Meropenem (1 gm) thrice daily along with steroids. Despite treatment adjustments, his condition worsened, necessitating the transfusion of 3 units of blood due to low hemoglobin levels.

Pancytopenia and high-grade fever prompted a bone marrow biopsy, which showed many caseating granulomas. Mycobacterium tuberculosis assays using acid-fast staining and polymerase chain reaction yielded positive results, suggesting tuberculosis-associated fever.

The patient was initiated on standard anti-tuberculosis treatment (isoniazid, rifampicin, pyrazinamide, and ethambutol) with intravenous steroids. Rapid improvement in symptoms occurred within 2 days, with fever subsiding and oxygen saturation maintained without BiPAP or oxygen support. Blood parameters (Table 6) improved within 5–6 days, and liver enzyme levels normalized. Chest X-ray (Fig. 6) also shows that patient is recovering from ARDS (initial improvement of alveolar opacities in both upper zones of lung). ARDS and plasmodium vivax malaria were also treated for the patient during this period. A diagnosis of bone marrow tuberculosis was established.

The patient's health improved with anti-tuberculosis treatment, and he was referred to a chest physician for further care. He had no fever or other symptoms during a 5-month follow-up, and his total blood count was within normal limits. Following 9 months of antitubercular therapy, the patient resumed his regular daily activities and reverted to his premorbid state.

Case 4: Metastases

A 45-year-old male taxi driver arrived at the hospital after experiencing dyspnea and a persistent cough for 2 days. He denied having a fever, nocturnal sweats, or hemoptysis. He also suffered from easy fatigability

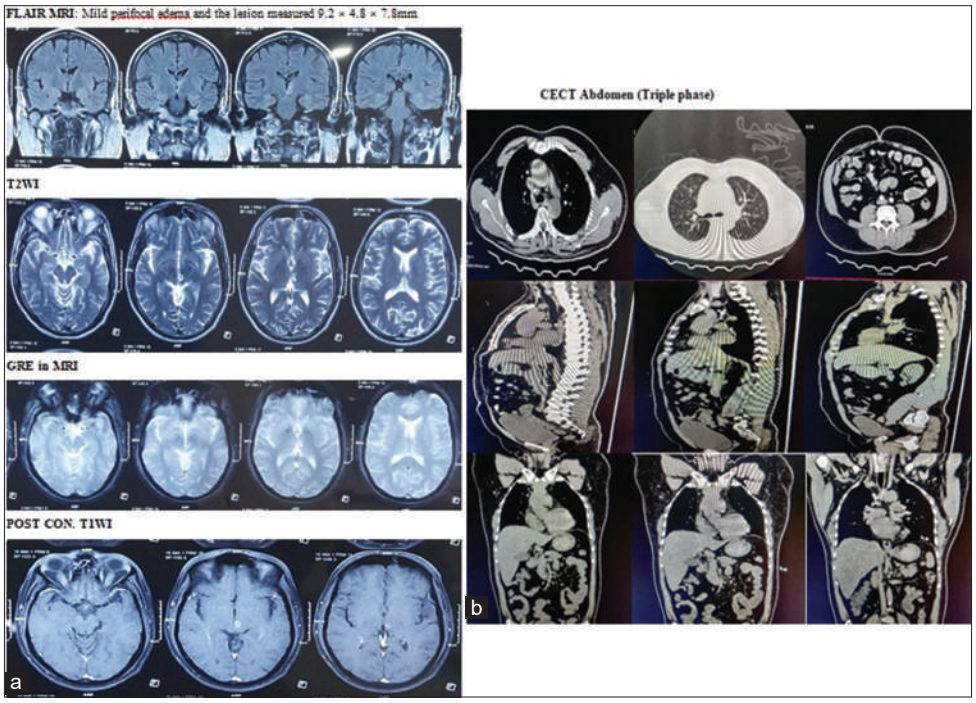

Fig. 4: (a) MRI brain (plain+contrast) (b) CECT abdomen (triple phase)

Table 5: Blood Investigation (At the time of admission)

Parameter	Value	Parameter	Value
Hb	7.7 g/dl	Bilirubin Total	3.37 mg/dl
WBC	1040/cmm	Bilirubin Direct	2.89 mg/dl
Platelet	14000/cmm	SGPT	77 U/L
PCV	25.90%	SGOT	92.29 U/L
MCV	104.4 fL	ALP	526.46 U/L
HIV 1/2	Negative	Dengue IgG/IgM	Negative
CRP	127.04 mg/dl	G6PD	15.87 IU/g
Amylase	205.65 U/L	D-Dimer	3292g/L
Lipase	184 U/L	PT	15.9 secs
Urea	22 mg/dl	aPTT	11.2 secs
Sr. Creatinine	0.67 mg/dl	INR	1.43
Malarial Parasite	Positive for Plasmodium vivax		

Table 6: Blood Investigation (At the time of discharge)

Parameter	Value	Parameter	Value	Parameter	Value
Haemoglobin	11.4g/dl	ALP	128 U/L	D-Dimer	550g/L
WBC	1270/cmm	CRP	<25 mg/dl	PT	11.4 secs
Platelet count	32000/cmm	Amylase	84 U/L	aPTT	10.3 secs
MP	Negative	Lipase	55 U/L	INR	1.11
SGPT	16 U/L	Urea	18 mg/dl	Sr. Creat.	0.6 mg/dl
SGOT	22 U/L				

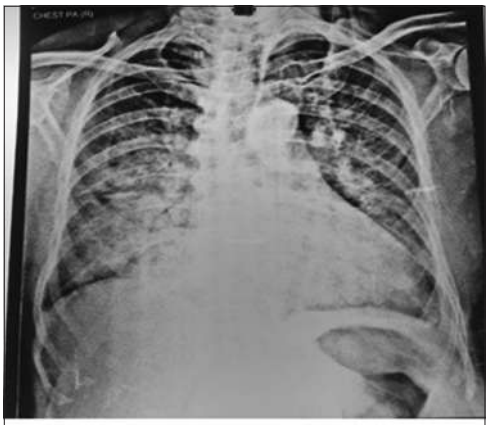

Fig. 5: Chest X-ray time of admission

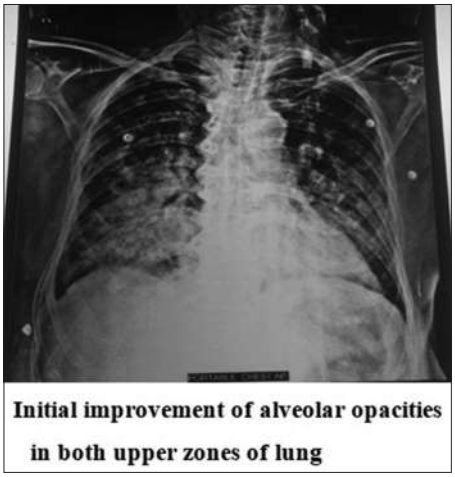

Fig. 6: Chest X-ray after treatment

and palpitations. The patient is not currently taking any medications and has no prior medical or surgical history. He has never smoked, abused alcohol, or used illegal drugs. Because he works as a driver, he interacts with a lot of individuals in various cities.

On physical examination, he was afebrile and lethargic. His oxygen saturation on admission was 92% on room air. There was a noticeable reduction in chest wall movement on the right side compared to the left. Percussion of the chest revealed a dull sound, and Auscultation revealed a reduction in breath sounds on the right lung.

A low hemoglobin level, high levels of white blood cells and platelets, and higher levels of erythrocyte sedimentation rate and C-reactive protein were found by additional testing (Table 7). The stool examination revealed weakly positive occult blood (Table 8). Cultures exhibited no growth, and sputum analysis confirmed negative results for acid-fast bacilli. An X-ray of the chest showed bilateral cannonball opacities (Fig. 7). Subsequent CT chest with contrast (Figs. 8 and 9) revealed multiple variously sized heterogeneously enhancing lesions in the left upper lobular bronchus, left pleura, paratracheal region of bilateral axilla, bilateral abdominal wall, and lytic lesions in the vertebrae and right scapula, all consistent with metastasis.

DISCUSSION

The diagnostic and treatment of extrapulmonary tuberculosis are complicated by the need for comprehensive clinical evaluation, pathoradiological methods, and microbiological cum molecular tests to identify Mtb in specimens from afflicted areas. These methods can occasionally be intrusive, which discourages patients. Though it also affects immunocompetent people (15–20%). The prevalence of EP-TB is more common in immunocompromised patients (40–50%), particularly in India [8]. When pulmonary Kochs are absent, diagnosing EP-TB and unusual presentation of tuberculosis is more difficult. Despite treatment, the mortality rate is higher in immunocompromised individuals [9,10]. In this series, four uncommon TB patients with unusual presentations are highlighted.

The first one was a case Pleural TB who presented with hiccup and exudative pleural effusion. Due to a lack of bacilli and nonspecific

Table 7: Blood investigation

Parameter	Value
Hemoglobin	7.2 gm/dl
White blood cell count	14540/cmm
Polymorphs	79.8%
Lymphocytes	8.8%
Platelet count	996000/cmm
Erythrocyte sedimentation rate	80 mm/h
TSH	4.453 mIU/L
HBA1C	6.4%
Iron	15 ug/dl
Ferritin	18.7 ng/ml
C-reactive protein	8.5 mg/dl
SGPT	27 U/L
SGOT	17 U/L
ALP	139 U/L
Total protein (TP)	6.2 g/dl
Albumin (ALB)	2.7 g/dl
Globulin	3.5 g/dl
A/G Ratio	0.77
Total bilirubin	0.2 mg/dl
Direct bilirubin	0.1 mg/dl
Indirect bilirubin	0.1 mg/dl
GAMMA GT	136 U/L
Blood urea	22 mg/dl
Sr. Creatinine	0.7 mg/dl

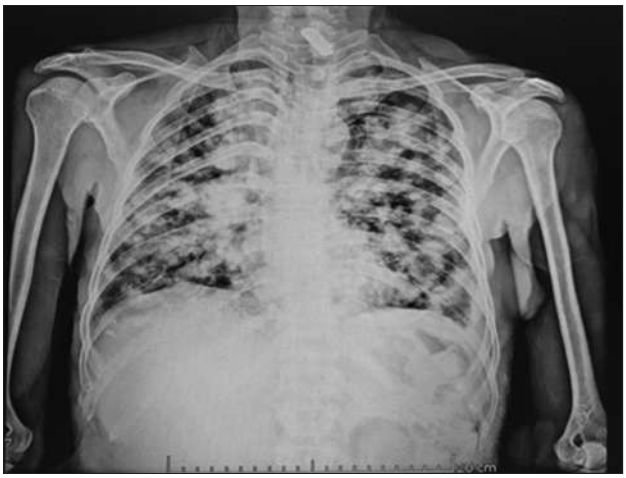

Fig. 7: Chest X Ray (B/L Canon ball opacity)

pleural fluid features, pleural tuberculosis is difficult to diagnose. When evaluating pleural TB, the conventional diagnostic techniques of ZN staining, pleural fluid sample culture, and tuberculin testing for pulmonary TB have low sensitivity [11,12]. The culture of the pleural biopsy samples is the gold standard for pleural tuberculosis diagnosis. Testing for Adenosine deaminase (ADA) and interferon-gamma (IFN-γ) levels in pleural fluid have continuously shown great sensitivity and specificity for identifying pleural TB cases [13].

The main objectives of the various suggested treatment plans for pleural tuberculosis are to stop active pulmonary tuberculosis from developing, lessen pleural fluid re-accumulation, and stop long-term consequences from developing. Han *et al*. showed that 49% of patients with substantial pleural effusions at presentation had disappeared by the end of 6 months of treatment in a trial of 85 pleural TB patients. Furthermore, even after taking anti-TB medicine for 6 months, those with residual disease showed improvement [14]. A rare symptom of TB is intractable hiccups. Infected lung and pleural tissue, as well as swollen and infected lymph nodes in the anterior mediastinum, can activate the phrenic nerve [15].

The second case was cerebral tuberculoma decreasing vision over the past 15 days and restriction in superior gaze on dilatation of pupils. Blood borne spread of Mycobacterium tuberculosis (MT), which is mostly linked to TB meningitis, causes cerebral tuberculomas, a rare and dangerous form of tuberculosis (TB). Non-specific radiologic findings and symptoms might occasionally result in a misdiagnosis. There have also been reports of paradoxical TB growth or development after antituberculous treatment, which may have an immunological foundation [16-18]. High doses of steroids and ongoing antituberculous medication are used as treatment, frequently for an extended period of time. In rare instances, surgery has been used [19,20].

Even when a patient is on medication, tuberculomas should be taken into consideration if they have focal neurology or elevated intracranial pressure. It is probably wise to continue treatment until the tuberculomas completely resolve, even though it is unclear whether contrast enhanced lesions indicate active lesions or simply inflammation [21].

The third case was TB bone marrow presented with shortness of breath, fever, and pancytopenia.

Pancytopenia is one of the hematological symptoms that extrapulmonary tuberculosis can present with [22,23]. Another potential explanation of pancytopenia in tuberculosis is the infiltration of caseating or non-caseating granulomas into the bone marrow, which results in reversible or irreversible fibrosis [24]. In an endemic area, patients who present with peripheral cytopenia and pyrexia of known origin must warn the doctor about the probable diagnosis of disseminated TB. Bone marrow tests must be performed as soon as possible because delayed diagnosis carries a high morbidity and mortality rate.

Tuberculosis accounts for 6–48% of the cases of bone marrow granulomas. Granulomas (33–100%) are typically seen on bone marrow biopsy in patients of miliary tuberculosis; caseation is infrequent [29%]; and acid-fast bacilli detected by Ziehl-Neelsen staining are rarely found [25]. The vague symptoms of disseminated TB continue to make diagnosis difficult. The primary cause of the bad prognosis in cases of disseminated tuberculosis is delayed diagnosis due to a lack of distinct clinical characteristics. In contrast to only performing a mycobacterial blood culture, Wang *et al*. reported that the parallel culture and histological eevaluation of bone marrow is more sensitive in identifying disseminated tuberculosis [26]. Our patient's positive prognosis was most likely brought about by an early diagnosis and antituberculosis treatment.

The fourth one was a case of metastatic TB presenting with shortness of breath and CT chest with contrast revealed multiple variously sized heterogeneously enhancing lesions. Many asymptomatic TB cases are discovered by chance on chest radiography, and individuals with active TB frequently test negative for AFB stains, culture, and PCR. In these situations, the radiologic findings may mimic several different illnesses and are not typical of tuberculosis. Furthermore, descriptions of pulmonary tuberculosis (TB) infrequently manifesting as many distinct nodules that resemble multiple lung metastases have been scattered throughout the English literature [27-29]. Multiple sharply or poorly defined micronodules measuring 1–4 mm bilaterally are a symptom of miliary tuberculosis, which is caused by the hematogenous dissemination of tubercle bacilli [27-29].

Hematogenous bacilli dissemination is seen in the uncommon appearance of metastatic tuberculosis. The combination of the host's immune level, the infection route, and the aggressiveness of the bacilli causes metastatic TB, which is prone to hematogenous dissemination with internal organ damage and cutaneous abscesses. It usually affects the thorax and lower limbs. Focused areas of necrosis and abscess formation with a large number of acid-fast resistant bacilli are among the histologic findings [30].

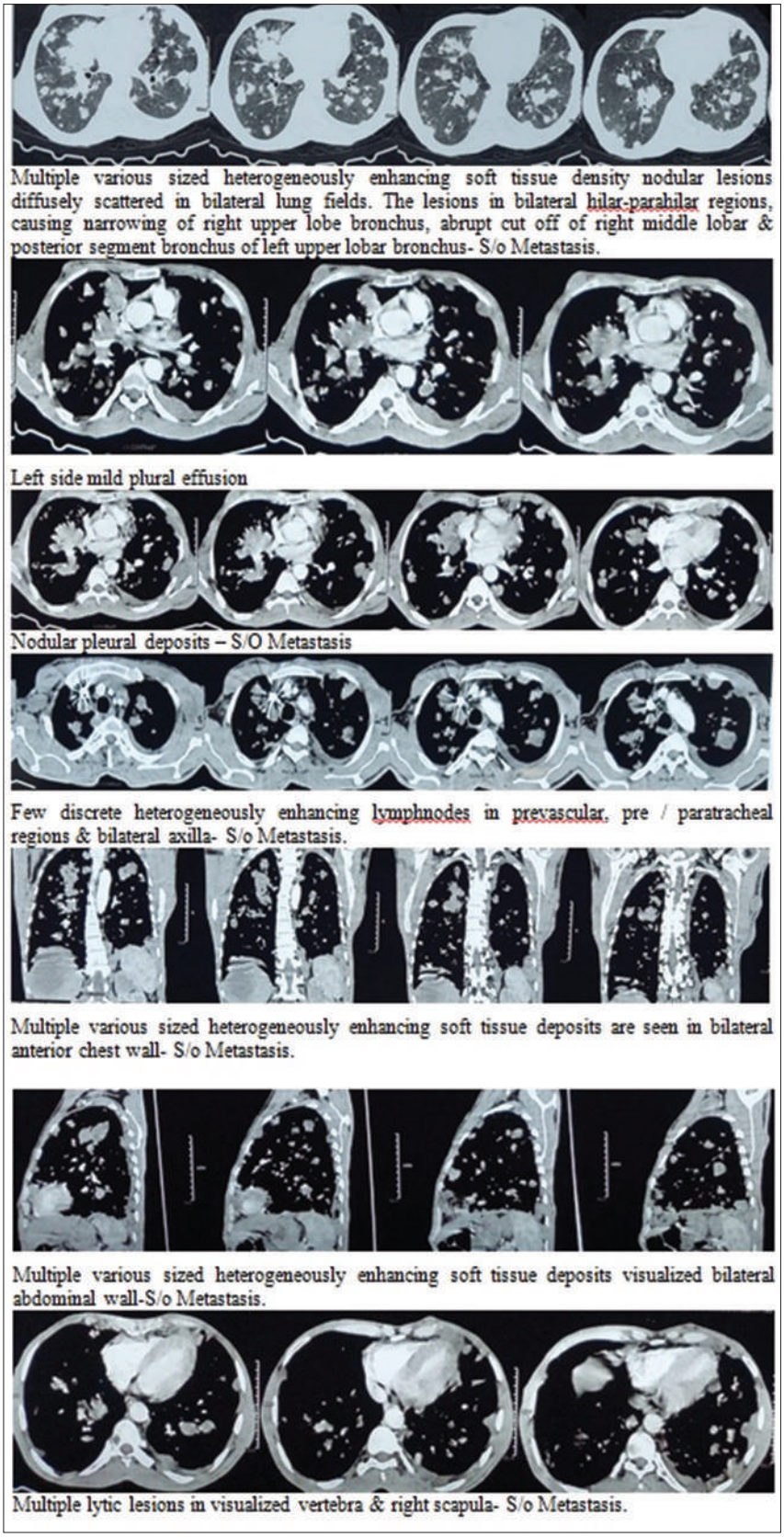

Fig. 8: CECT thorax

When a nodule surrounded by centrilobular or satellite micronodules is found, pulmonary tuberculosis should be considered in the differential diagnosis, even if there are no distinguishing features such as cavitation or the tree-in-bud sign. This is because metastases may be indicated by bilateral numerous well-circumscribed lung nodules [31].

Table 8: Stool occult blood examination

Stool occult blood (OB)	Weak positive

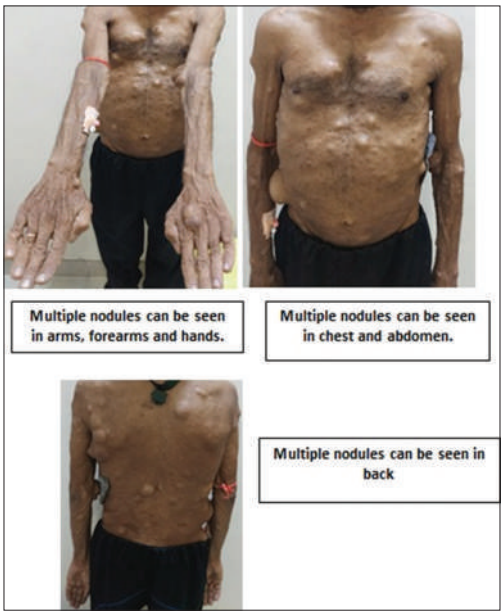

Fig. 9: Image of a patient with a multiple nodule

CONCLUSION

This case series highlights the diverse clinical manifestations and challenges in diagnosing and managing both pulmonary and extrapulmonary tuberculosis (EPTB). The cases presented emphasize the variability in TB presentations, from pleural effusions to complex cases involving lung and bone marrow, each with its own diagnostic hurdles. Despite advances in diagnostic tools, the identification of EPTB remains difficult due to its ability to mimic other diseases and the lack of specific symptoms, often leading to delayed treatment. Early recognition of TB, particularly EPTB, requires a high index of suspicion, especially in patients with atypical presentations or in those from high-risk populations. Multimodal diagnostic approaches, including imaging, laboratory tests, and pleural fluid analysis, are critical in confirming the diagnosis. The case series also underscores the importance of tailored anti-tuberculous therapy based on the site and extent of the disease, often requiring prolonged and intensive treatment regimens.

Further research is needed to optimize diagnostic algorithms and therapeutic interventions for EPTB, particularly in resource-limited settings where access to advanced diagnostic modalities may be restricted. This study reinforces the need for heightened awareness and clinical vigilance in managing TB, given its significant global health burden. This conclusion synthesizes the key findings from your case study, emphasizing the complexity and importance of timely diagnosis and tailored treatment in managing TB, especially in its extrapulmonary forms.

FUNDING

Nil.

CONFLICTS OF INTEREST

Nil.

AUTHORSHIP CONTRIBUTIONS

All the three authors equally contributed to the manuscript.

ETHICAL APPROVAL

Written informed consent was obtained from the patients for the publication of these case series and accompanying images.

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