FALSE-POSITIVE ACID-FAST BACILLI MICROSCOPY AS A CAUSE OF TUBERCULOSIS OVERDIAGNOSIS IN A PATIENT WITH SARCOIDOSIS: A CASE REPORT AND ANALYSIS OF DIAGNOSTIC ERRORS

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Abstract

Background. The differential diagnosis of sarcoidosis and disseminated pulmonary tuberculosis remains a challenging clinical problem owing to the radiological similarity of the two conditions and the possibility of detecting acid-fast bacilli (AFB) in the absence of active tuberculous disease.

Objective. To present a case of tuberculosis overdiagnosis in a patient with sarcoidosis attributable to a false-positive microscopy result, and to analyze the factors contributing to the diagnostic error, with a detailed account of the investigative methods employed.

Materials and Methods. A retrospective analysis was performed of clinical data from patient N., a 34-year-old male admitted to a tuberculosis dispensary with suspected disseminated pulmonary tuberculosis. The following methods are described in detail: laboratory investigations (complete blood count; biochemical blood analysis including serum angiotensin-converting enzyme [ACE] measured by enzyme-linked immunosorbent assay, ionized calcium, and 24-hour urinary calcium); instrumental investigations (computed tomography [CT] of the chest performed on a 16-slice scanner with a slice thickness of 1 mm, pulmonary function testing, and electrocardiography); and morphological investigations (video-assisted thoracoscopic lung biopsy with histological examination and Ziehl–Neelsen staining, followed by expert-centre slide review).

Results. The initial suspicion of tuberculosis was raised on the basis of a CT pattern of pulmonary dissemination and the detection of isolated AFB in the surgical specimen. The absence of clinical and radiological improvement after two months of anti-tuberculosis therapy prompted diagnostic reassessment. The definitive verification of sarcoidosis was established through: expert-centre review of histological slides (non-caseating granulomas with asteroid bodies; AFB staining negative), markedly elevated serum ACE (187 U/L), and hypercalciuria (8.3 mmol/24 h). The article includes tables presenting the dynamics of laboratory parameters and a chronology of diagnostic events.

Conclusion. The detection of isolated AFB on microscopy should not constitute the sole basis for a diagnosis of tuberculosis in the absence of a clinical and radiological response to specific therapy. Measurement of serum ACE

and urinary calcium represents a valuable non-invasive approach to the diagnosis of sarcoidosis. Expert-centre review of histological material is an obligatory step in the verification of complex granulomatous processes. Detailed reporting of methods and the inclusion of tabular data enhance the reproducibility and evidential quality of clinical case observations.

Keywords: sarcoidosis; tuberculosis; differential diagnosis; diagnostic error; acid-fast bacilli; angiotensin-converting enzyme; case report; tables; investigative methods

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Introduction

Pulmonary sarcoidosis is a multisystem granulomatous disease of unknown etiology characterized by the formation of epithelioid cell granulomas in the absence of caseous necrosis [1]. The incidence of the disease ranges from 1 to 40 per 100,000 population across different regions, with the intrathoracic lymph nodes and lungs being the most commonly affected sites [1, 2].

The differential diagnosis of sarcoidosis and disseminated pulmonary tuberculosis poses considerable difficulties for clinicians owing to the similarity of their clinical and radiological presentations [3]. According to published data, the rate of diagnostic errors in granulomatous lung diseases may reach 20–30%, with false-positive diagnoses of tuberculosis reported in 4.6% of cases [4, 5]. A particular diagnostic challenge arises when isolated acid-fast bacilli (AFB) are detected on microscopy of biopsy specimens in the absence of active tuberculous disease; as reported by Lepikhina et al. [4], such artifacts are encountered in 6% of observations.

The pathogenesis of sarcoidosis is driven by the accumulation of activated T-lymphocytes (predominantly CD4+) and macrophages at target organs, resulting in granuloma formation and a shift of the immune response toward a Th1 profile [6]. This accounts for the diagnostic significance of serum angiotensin-converting enzyme (ACE), which is produced by the epithelioid cells of granulomas: elevated ACE demonstrates a sensitivity of approximately 60% and a specificity of up to 90% for active sarcoidosis [7].

The gold standard for diagnosis remains morphological verification through the identification of non-caseating epithelioid cell granulomas and the absence of AFB on Ziehl–Neelsen staining [8]. However, as emphasized in international clinical guidelines [9], the interpretation of histological findings requires considerable pathological expertise, and in complex cases, referral to expert reference centres for review is warranted [11, 12].

The aim of this study was to present a case of tuberculosis overdiagnosis in a patient with sarcoidosis attributable to a false-positive microscopy result, and to analyze the factors contributing to the diagnostic error, with a detailed account of the investigative methods employed to ensure the reproducibility and evidential quality of the observation.

Materials and Methods

This case report was prepared in accordance with the international CARE (Consensus-based Clinical Case Reporting) guidelines. A retrospective analysis of the medical records of a patient who underwent examination and treatment at the Tambov Regional Clinical Tuberculosis Dispensary and the National Medical Research Centre for Phthisiopulmonology and Infectious Diseases (NMRC FPI) of the Ministry of Health of the Russian Federation in 2024–2025 was performed.

Inclusion Criteria

A patient with a morphologically verified diagnosis of sarcoidosis in whom an erroneous assumption of tuberculous etiology had been made at the initial diagnostic stage.

Laboratory Methods

Blood samples were collected in the morning in a fasting state by venepuncture of the cubital vein into vacuum tubes with a clot activator (for biochemical analyses) and with EDTA (for complete blood count). The complete blood count was performed on a Sysmex XN-1000 haematology analyser (Sysmex, Japan) with determination of standard parameters.

Biochemical blood analysis was carried out on an Architect c8000 automated analyser (Abbott, USA). Serum ACE level was measured by enzyme-linked immunosorbent assay using the ACE-ELISA reagent kit (Alcor Bio LLC, Russia) on a ChemWell 2910 analyser (Awareness Technology, USA). The reference range established by the manufacturer was 20–70 U/L. The intra-assay coefficient of variation

was less than 5%, and the inter-assay coefficient of variation was less than 8%.

Ionized calcium in serum was determined by potentiometry on an EasyLyte electrolyte analyser (Medica, USA) using whole blood specimens delivered to the laboratory within 30 minutes of collection (reference range: 1.16–1.32 mmol/L). Twenty-four-hour urinary calcium was measured by colorimetry with arsenazo III on the same Architect c8000 analyser; urine was collected over 24 hours into a preservative-free container (reference range: 2.0–7.5 mmol/24 h).

Instrumental Methods

Computed tomography of the chest was performed on a 16-slice spiral CT scanner Somatom Emotion 16 (Siemens, Germany) according to a standard protocol: tube voltage 110 kV, effective exposure 80–120 mAs, slice thickness 1 mm, reconstruction increment 0.8 mm. Intravenous bolus contrast enhancement (iohexol 350 mgI/mL, 100 mL) was administered when the nature of lymphadenopathy was uncertain. Images were evaluated in pulmonary and mediastinal windows by two radiologists independently.

Pulmonary function testing was performed on a MasterScreen PFT spirometer (Jaeger, Germany) with determination of forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), the FEV_1/FVC ratio (Tiffeneau–Pinelli index), peak expiratory flow, and maximal expiratory flow rates at 25%, 50%, and 75% of FVC. Vital capacity (VC) and total lung capacity (TLC) were additionally assessed where indicated. Results were interpreted in accordance with ATS/ERS criteria.

Electrocardiography was performed in 12 standard leads on a Cardiax electrocardiograph (IMED, Hungary) at a recording speed of 50 mm/s.

Morphological Methods

Lung biopsy was performed by video-assisted thoracoscopic surgery (VATS) under general anaesthesia with selective bronchial intubation. Resection of segment S3 of the right lung was carried out. The obtained specimen was fixed in 10% neutral buffered formalin for 24 hours, processed through a standard histological tissue processing protocol, and embedded in paraffin. Sections of 4–5 µm thickness were stained with haematoxylin and eosin and by the Ziehl–Neelsen method for AFB detection. Microscopic examination was performed on a Leica DM2500 light microscope (Leica Microsystems, Germany) at magnifications of ×100, ×200, and ×400.

The primary histological report was issued by the Department of Pathology of the Tambov Regional Clinical Tuberculosis Dispensary. Owing to diagnostic uncertainty, the slides were referred for review to the reference centre of the NMRC FPI (Moscow), where an additional consultation by three pathologists specializing in granulomatous diseases was conducted.

Ethical Considerations

Written informed consent was obtained from the patient for the processing of personal data and the publication of this case report for scientific purposes.

Results

Clinical Characteristics of the Patient

Patient N., a 34-year-old male, was admitted to the Tambov Regional Clinical Tuberculosis Dispensary on 25 April 2025 with a referral diagnosis of newly diagnosed disseminated pulmonary tuberculosis. On admission, the patient reported exertional dyspnoea (mMRC grade 2) and intermittent chest pain unrelated to respiration.

History of present illness: since August 2024, the patient had experienced chest pain for which he had been unsuccessfully treated by a neurologist under the diagnosis of thoracalgia (receiving non-steroidal anti-inflammatory drugs with transient relief). From December 2024, inspiratory dyspnoea, a 6 kg weight loss over four months, and decreased appetite developed. Routine fluorography performed on 14 April 2025 revealed miliary dissemination for the first time, which was subsequently confirmed by chest CT on 16 April 2025.

Medical history: the patient denied any prior history of tuberculosis; no contact with tuberculosis patients was established. He had received BCG vaccination at birth, with a 4 mm scar on the left shoulder. Social history: smoking since the age of 18, 10–15 cigarettes per day (pack-year index: 12). Comorbidities: Gilbert's syndrome (diagnosed in 2019; total bilirubin up to 35 µmol/L outside exacerbations). No history of allergies. No occupational hazards.

Laboratory Data

The principal laboratory parameters of the patient over the course of the diagnostic and treatment period are presented in Table 1.

Table 1. Dynamics of laboratory parameters in patient N.

Parameter (reference range)	20 Jun 2025	07 Jul 2025	25 Aug 2025
Haemoglobin (130–160 g/L)	142	138	140

Parameter (reference range)	20 Jun 2025	07 Jul 2025	25 Aug 2025
White blood cell count (4–9×10⁹/L)	6,8	7,1	6,5
Lymphocytes (1.2–3.5×10⁹/L)	2,1	2,3	2,4
ESR (2–20 mm/h)	18	16	14
Total protein (65–85 g/L)	76	74	78
ALT (10–40 U/L)	32	35	30
AST (10–40 U/L)	28	30	27
Total bilirubin (5–21 µmol/L)	31*	28*	26*
Creatinine (62–115 µmol/L)	82	84	80
ACE (20–70 U/L)	**187**	–	145
Ionized calcium (1.16–1.32 mmol/L)	**1,32**	1,30	1,28
24-hour urinary calcium (2.0–7.5 mmol/24 h)	**8,3**	–	6,9

Note: The elevated bilirubin level is attributable to the comorbid Gilbert's syndrome (predominantly unconjugated fraction).

Noteworthy findings included a markedly elevated serum ACE level (2.7 times above the upper limit of the reference range) and hypercalciuria in the presence of normal ionized calcium, a combination characteristic of active sarcoidosis. These measurements were obtained only on 20 June 2025, two months after admission, which precluded the use of these biomarkers at the early stage of differential diagnosis.

Instrumental Findings

Chest CT performed on 16 April 2025 and repeated on 9 June 2025 (following two months of anti-tuberculosis therapy) demonstrated multiple small focal disseminations (miliary and larger nodules measuring 2–5 mm) throughout all pulmonary fields with a predominantly perilymphatic distribution (along bronchi, vessels, and interlobular septa). The pulmonary hila were enlarged owing to increased intrathoracic lymph nodes (bronchopulmonary and tracheobronchial groups) measuring up to 13–15 mm in short axis bilaterally, forming the so-called "curtain sign" (Fig. 1). No evidence of lung tissue destruction or pleural effusion was identified.

Pulmonary function testing performed on 7 July 2025 revealed moderate restrictive impairment: VC 68% of predicted, FEV_1 72% of predicted, FEV_1/FVC ratio 82% (within normal limits). Conclusion: moderate restrictive ventilatory impairment.

ECG performed on 7 July 2025 demonstrated sinus rhythm at 72 beats per minute, right axis deviation (α angle +100°), and incomplete right bundle branch block. These findings were interpreted as indirect evidence of right heart strain and possible early development of cor pulmonale.

Morphological Findings

VATS lung biopsy performed on 17 May 2025 yielded material for histological examination. The primary report issued by the Department of Pathology in Tambov read as follows: "The specimens show lung tissue containing multiple epithelioid cell granulomas admixed with multinucleated giant cells. A subset of granulomas contains small areas of fibrinoid necrosis. Ziehl–Neelsen staining revealed isolated AFB in the field of view (1–2 per 100 fields). Conclusion: morphological picture of productive inflammation, most consistent with tuberculous etiology (A15.2)."

In view of the absence of clinical and radiological response to anti-tuberculosis therapy and persistent suspicion of sarcoidosis, the histological slides were referred for review to the reference centre of the NMRC FPI. The expert panel report (three pathologists) stated: "The lung tissue contains multiple, focally confluent, well-defined non-caseating granulomas composed of epithelioid cells and Pirogov–Langhans-type multinucleated giant cells. Asteroid bodies are identified within a subset of granulomas. Caseous necrosis is absent. Fibrotic changes surrounding the granulomas are minimal. Ziehl–Neelsen staining (200 fields examined) revealed no acid-fast organisms. The morphological picture is fully consistent with sarcoidosis (D86)" (Figs. 2, 3).

Treatment Course and Final Diagnosis

A chronology of the key diagnostic and therapeutic events is presented in Table 2.

Table 2. Chronology of diagnostic and therapeutic interventions

Date	Event	Result / Comment
August 2024	Onset of symptoms (chest pain)	Treated by a neurologist for thoracalgia
December 2024	Development of dyspnoea and weight loss	Medical consultations did not yield a diagnosis
14 Apr 2025	Routine fluorography	Miliary dissemination identified for the first time
16 Apr 2025	Chest CT	Dissemination confirmed; mediastinal lymphadenopathy
25 Apr 2025	Admission to Tambov Regional Clinical Tuberculosis Dispensary	Referral diagnosis: disseminated tuberculosis
13 May 2025	Initiation of anti-tuberculosis therapy	Isoniazid 0.6 g + rifampicin 0.6 g + ethambutol 1.6 g + amikacin 1.0 g IM
17 May 2025	VATS lung biopsy	Specimen obtained for histological examination
June 2025	Primary histological report	"Productive inflammation, probable tuberculosis; isolated AFB detected"
20 Jun 2025	Biochemical blood analysis (ACE measured for the first time)	ACE 187 U/L; ionized calcium 1.32 mmol/L
24 Jun 2025	24-hour urinary calcium	Calcium 8.3 mmol/24 h (hypercalciuria)
24 Jun 2025	Multidisciplinary case conference; anti-tuberculosis therapy discontinued	Sarcoidosis suspected; pentoxifylline and ibuprofen prescribed
July 2025	Histological slide review at NMRC FPI	Conclusion: sarcoidosis; no AFB detected
07 Jul 2025	Pulmonary function testing; ECG	Restrictive impairment; signs of right heart strain
20 Jul 2025	Discharge from hospital	Follow-up with pulmonologist recommended
August 2025	Follow-up outpatient visit	ACE decreased to 145 U/L; urinary calcium 6.9 mmol/24 h

Final Clinical Diagnosis

Primary: Sarcoidosis of the intrathoracic lymph nodes and lungs, chronic course, active phase (morphologically verified).

Complications: Respiratory failure, grade I–II.

Comorbidity: Gilbert's syndrome.

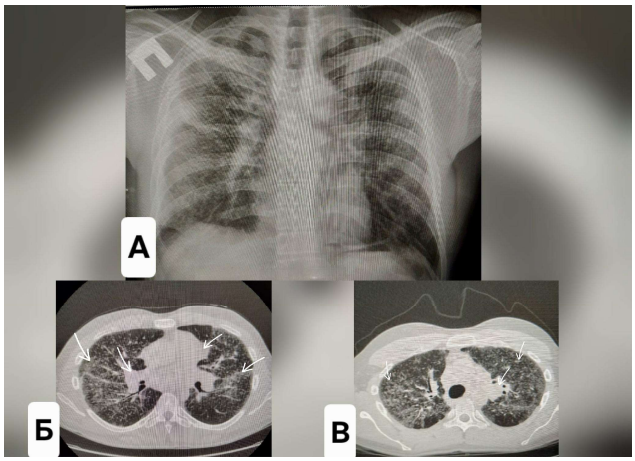

Figure 1. Chest computed tomography of patient N., axial section. Multiple small nodular shadows throughout all pulmonary fields with a predominantly perilymphatic distribution (arrows) and bilateral enlargement of bronchopulmonary lymph nodes (asterisks). CT semiotics characteristic of sarcoidosis.

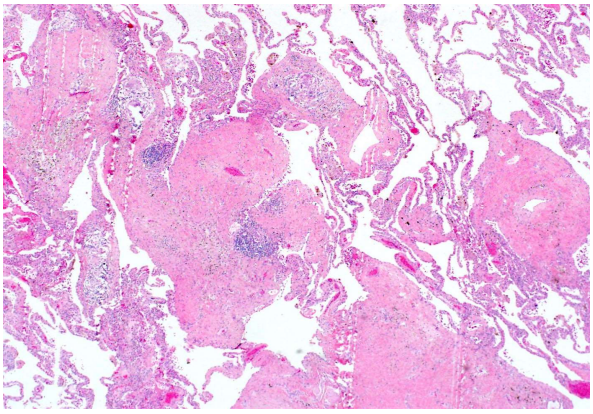

Figure 2. Micrograph of a lung biopsy specimen from patient N. Well-defined non-caseating granulomas composed of epithelioid cells and Pirogov–Langhans-type multinucleated giant cells (arrow). Caseous necrosis is absent. Haematoxylin and eosin stain, magnification ×100.

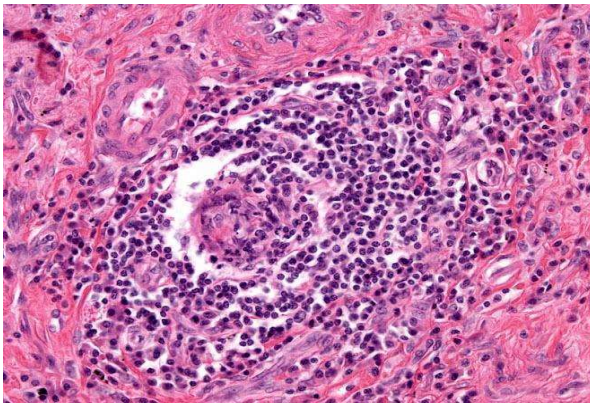

Figure 3. Fragment of a sarcoid granuloma containing a multinucleated giant cell with an asteroid body (inset). Haematoxylin and eosin stain, magnification ×200.

Discussion

The presented case illustrates the characteristic difficulties encountered in the differential diagnosis of disseminated pulmonary processes and highlights several key factors contributing to diagnostic errors.

False-Positive Microscopy as a Cause of Tuberculosis Overdiagnosis

The detection of isolated AFB in the surgical specimen on primary histological examination proved to be the decisive factor that directed the diagnostic workup toward tuberculosis. Such a scenario is not uncommon: in an analysis of 35 cases of sarcoidosis initially diagnosed as tuberculosis in tuberculosis hospitals, Lepikhina et al. [5] reported that false-positive AFB detection on fluorescence microscopy occurred in 6% of observations. Potential causes include specimen contamination, staining artefacts, the presence of saprophytic mycobacteria, and cross-reactivity with other acid-fast structures (e.g., residual cell wall components).

This case reinforces the principle that bacterioscopy alone cannot serve as the sole method of aetiological diagnosis of tuberculosis, particularly in the setting of scanty bacillary shedding, and must be supplemented by culture and molecular genetic methods (PCR) [10]. Regrettably, PCR testing of the biopsy specimen was not performed in the present case, which should be regarded as a diagnostic deficiency. Nevertheless, the negative culture result (the specimen was submitted, although no data were recorded in the medical notes) and the absence of a therapeutic response indirectly corroborate the unreliability of the microscopy findings.

The Role of Expert Histological Review

The decisive contribution to establishing the correct diagnosis was made by the review of histological material at the federal reference centre. Expert pathomorphologists identified features pathognomonic for sarcoidosis: the absence of caseous necrosis, the presence of asteroid bodies, and well-defined granuloma borders. As emphasized in the clinical guidelines of the Russian Respiratory Society [8] and international recommendations [9], the interpretation of granulomatous inflammation requires considerable expertise, and in complex cases, expert review is mandatory. Centralised assessment of complex biopsy specimens has been shown to prevent diagnostic errors in 15–20% of observations [5, 9].

Diagnostic Value of Non-Invasive Biomarkers

The markedly elevated serum ACE level (187 U/L) and hypercalciuria (8.3 mmol/24 h) provided compelling evidence in favour of sarcoidosis. According to the clinical guidelines of the Russian Ministry of Health [7], elevated ACE demonstrates a sensitivity of approximately 60% and a specificity of up to 90% for active sarcoidosis. An elevation of this magnitude is exceedingly rare in tuberculosis and represents an important non-invasive diagnostic marker. Hypercalciuria is likewise a characteristic feature of sarcoidosis, occurring in up to 40% of cases, and results from extrarenal production of 1,25-dihydroxyvitamin D_3 by activated macrophages [7].

A critically important observation is that these biomarkers were not measured until two months after admission. Had they been assessed at the initial diagnostic workup, the time to correct diagnosis could have been substantially reduced, and the prescription of ineffective anti-tuberculosis therapy avoided. The inclusion of serum ACE and urinary calcium in the mandatory diagnostic workup for patients presenting with unexplained disseminated pulmonary processes appears to be justified.

The Critical Role of Failure to Respond to Therapy

The absence of clinical and radiological improvement following two months of anti-tuberculosis therapy served as the key trigger for reassessment of the diagnostic concept. In accordance with current understanding, the lack of response to specific therapy over a period of 2–3 months should be considered a mandatory indication for reconsidering the diagnosis in favour of non-infectious granulomatoses [3, 5].

Factors Contributing to the Diagnostic Error

Based on the present observation, the following factors can be identified as having led to tuberculosis overdiagnosis:

- Overreliance on bacterioscopy results without accounting for the possibility of false-positive AFB detection.
- Absence of PCR testing of the biopsy specimen for mycobacterial verification.
- Insufficient pathological expertise at the initial stage of histological interpretation.
- Failure to measure serum ACE and urinary calcium at the time of admission.
- Delayed referral of histological material for expert centre review.

These factors are fully consistent with the findings of a study examining factors associated with diagnostic errors in sarcoidosis patients managed in tuberculosis hospitals [5], in which the principal causes identified were: radiologist error (54%), absence of biopsy (OR 0.09; $p<0.001$), and detection of AFB (OR 0.22; $p=0.026$).

Limitations

The present case report has limitations inherent to its retrospective design and the absence of certain diagnostic data (PCR testing of the biopsy specimen, serum ACE level at admission, and mycobacterial culture of the biopsy). Nevertheless, the observation retains its value in illustrating typical diagnostic errors and the means by which they may be prevented. The detailed description of methods and the inclusion of tabular data enhance reproducibility and allow readers to compare their own findings with those reported here.

Conclusions

- The presented case demonstrates that a false-positive AFB microscopy result (detection of isolated AFB) may lead to overdiagnosis of disseminated pulmonary tuberculosis in patients with sarcoidosis and to the prescription of ineffective anti-tuberculosis therapy.
- Measurement of serum ACE and urinary calcium represents a valuable non-invasive approach to the diagnosis of sarcoidosis; however, it must be applied in a timely manner at the early stages of the diagnostic workup. A markedly elevated serum ACE level (more than 2.5 times above the upper limit of normal) combined with hypercalciuria should be regarded as compelling evidence in favour of sarcoidosis, even when AFB are detected.
- The absence of a clinical and radiological response to anti-tuberculosis therapy over a period of 2–3 months should serve as a mandatory indication for diagnostic reassessment and exclusion of non-infectious granulomatoses.
- Expert pathological review of histological slides, with application of the appropriate stains (including Ziehl–Neelsen) and molecular genetic methods, is an obligatory step in the verification of granulomatous inflammation aetiology in complex diagnostic cases.
- To prevent similar diagnostic errors, it is advisable to implement a comprehensive diagnostic algorithm in clinical practice, incorporating mandatory measurement of serum ACE and urinary calcium in all patients presenting with unexplained disseminated pulmonary processes, as well as broader use of PCR-based diagnostics of biopsy specimens when tuberculosis is suspected.
- Detailed reporting of investigative methods and presentation of data in tabular form enhance the evidential quality of clinical case reports and are consistent with current standards for scientific publications.

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