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RESEARCH ARTICLE

Clinical & Radiological Spectrum of Histoplasmosis in Delhi-NCR: A Retrospective Analysis and Evidence of Emerging Endemicity

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ABSTRACT

Background: Histoplasmosis, caused by the dimorphic fungi Histoplasma capsulatum and acquired primarily through inhalation of spores from soil enriched with bird or bat droppings, has traditionally been considered rare in India. Recent reports, however, suggest rising recognition from previously non-endemic regions. This case series describes nine patients from Delhi-NCR, highlighting diagnostic challenges and signals of emerging endemicity.

Methods: Nine patients with histopathologically confirmed histoplasmosis were retrospectively reviewed at a tertiary care centre in Delhi-NCR. Clinical presentation, radiology, diagnostic modalities, treatment, and outcomes were analyzed. Histopathology using Periodic acid–Schiff (PAS) and Gomori methenamine silver (GMS) stains confirmed the diagnosis in all cases.

Results: Nine patients with a median age of 64 years were identified, with a male predominance (88.9%). Eight of nine patients were immunocompromised, either due to diabetes mellitus (77.8%), prior tuberculosis (55.6%), or other causes of immunosuppression. Most presented with cough (100%), fever (66.7%), weight loss (44.4%), hemoptysis (22.2%), chest pain, or non-resolving pneumonia. Radiologically, consolidation and cavitation were most common (66.7%), frequently mimicking pulmonary tuberculosis, mediastinal lymphadenopathy (44.4%), and occasionally complicated by pneumothorax or pleural effusion. All diagnoses were confirmed through tissue histopathology. Localized histoplasmosis responded well to azole monotherapy (66.7%), while disseminated disease was associated with poorer outcomes and often required amphotericin B in combination with or followed by an azole. Seven patients (77.8%), improved clinically and radiologically; two patients succumbed to progressive disease (22.2%).

Conclusion: Histoplasmosis in the Delhi-NCR region commonly masquerades as tuberculosis and is likely underrecognized. A high index of suspicion in patients with non-resolving pneumonia or cavitary lung lesions, early tissue diagnosis, and timely institution of antifungal therapy are critical to improving outcomes. The present series supports the possibility of emerging endemicity of histoplasmosis in North India and underscores the need for heightened clinical awareness and further epidemiological studies.

Introduction

Histoplasmosis is a systemic fungal infection caused by the dimorphic fungi Histoplasma capsulatum.[1] Two main varieties are recognized:

- H. capsulatum var. capsulatum (2–4 µm), which has a worldwide distribution
- H. capsulatum var. duboisii (8–15 µm), predominantly reported from sub-Saharan Africa.[1]

The disease can present as:

1. Acute pulmonary histoplasmosis — often asymptomatic or self-limiting
2. Chronic cavitary pulmonary histoplasmosis — resembles TB
3. Progressive disseminated histoplasmosis (PDH) — more severe, especially in immunocompromised patients.

Other manifestations include pancytopenia, disseminated intra vascular coagulation, focal parenchymal lesions, renal failure, gastrointestinal manifestations like diarrhea and vomiting, encephalopathy, skin lesions and adrenal in sufficiency.[2]

Globally, the Ohio and Mississippi river valleys in the United States constitute classic endemic foci where histoplasmosis represents a common cause of community-acquired fungal pneumonia.[1,3] In India, histoplasmosis was historically regarded as rare[4], but an increasing number of reports have emerged over the past few decades, suggests that the disease is significantly underrecognized. Most Indian reports originate from eastern and northeastern regions—particularly West Bengal, Assam, and the Gangetic belt—where disseminated and adrenal forms predominate[4-11] Various case reports of cutaneous histoplasmosis have also been reported in both immunocompetent and immunosuppressed individuals in India.[12,13,14]

The Delhi-National Capital Region (NCR) has not traditionally been considered endemic for histoplasmosis. However, recent sporadic cases and small series suggest that the disease may be underdiagnosed in this region.[15,16,17]

The diagnostic challenge of histoplasmosis in India is compounded by the country's high tuberculosis (TB) burden.[18] Pulmonary histoplasmosis frequently mimics TB both clinically and radiologically, presenting with chronic cough, fever, weight loss, cavitary lung lesions, and mediastinal lymphadenopathy.[19,20] Consequently, many patients are empirically treated with anti-tubercular therapy, resulting in delayed diagnosis, disease progression, and increased morbidity and mortality. This overlap is particularly problematic in patients with diabetes mellitus, prior TB, or chronic structural lung diseases.

Against this background, the emergence of histoplasmosis cases from Delhi-NCR raises important epidemiological and clinical questions regarding underdiagnosis, changing exposure patterns, and host susceptibility.

This study describes the clinical and radiological profile, diagnostic challenges, treatment, and outcomes of nine patients with histopathologically confirmed histoplasmosis from a tertiary care centre in Delhi-NCR. By comparing our findings with published Indian and international data, we aim to highlight pulmonary histoplasmosis as an important and underrecognized differential diagnosis in TB-endemic settings.

Aim: To study the clinical, radiological profile of patients of Histoplasmosis, diagnostic challenges and treatment outcomes.

Methodology:

This retrospective case series included nine consecutive patients diagnosed with histoplasmosis at a tertiary care centre in the Delhi-NCR region from June 2024 to November 2025. All patients had a confirmed diagnosis on histopathology.

CLINICAL AND RADIOLOGICAL EVALUATION:

Demographic details, comorbidities, clinical presentation, and prior TB or immunosuppression was recorded. All patients underwent chest imaging, including chest X-ray and high-resolution computed tomography (HRCT) of the thorax. Radiological patterns such as consolidation, cavitation, fibrocavitatory lesions, mediastinal lymphadenopathy, interstitial lung disease (ILD) patterns, pneumothorax, and pleural effusion were documented.

DIAGNOSTIC PROCEDURES

Diagnostic interventions included:

- Flexible bronchoscopy with bronchoalveolar lavage (BAL)
- Endobronchial and transbronchial lung biopsies (TBLB)
- Radial endobronchial ultrasound (EBUS)-guided transbronchial biopsies
- Conventional EBUS-guided nodal sampling, where indicated
- CT-guided percutaneous lung or pleural biopsies

BAL samples and other respiratory specimens were subjected to bacterial and fungal cultures, Mycobacterium tuberculosis GeneXpert testing, fungal polymerase chain reaction (PCR), galactomannan, and β-D-glucan assays where feasible.

HISTOPATHOLOGY

Biopsy specimens were processed with routine hematoxylin and eosin (H&E) staining and special fungal stains, including PAS and GMS. The diagnosis of histoplasmosis was established by demonstration of intracellular oval yeast forms of Histoplasma within macrophages (Figure 1-3)

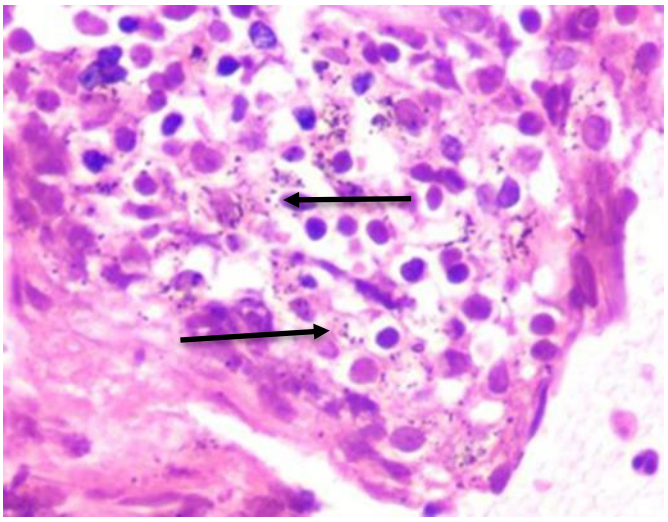


Figure 1A: Histopathology (H&E stain, ×400) showing macrophages with Intracellular oval forms of histoplasmosis

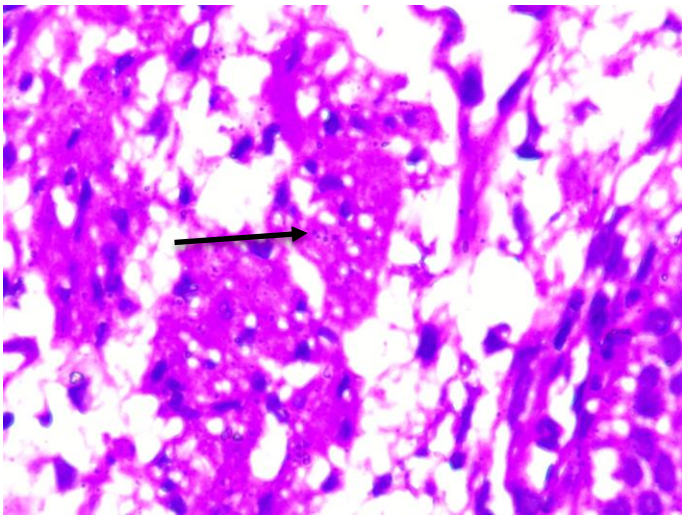


Figure 2: PAS stain showing numerous small (2-4 um), oval, budding yeasts

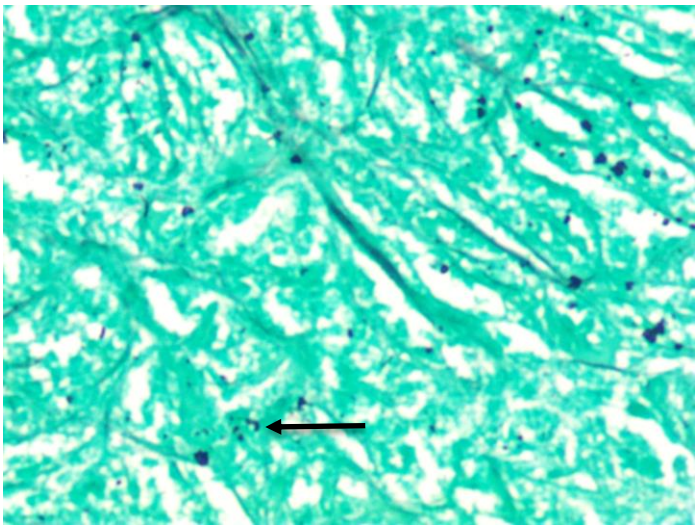


Figure 3: GMS Stain showing round to oval yeasts with narrow– based budding

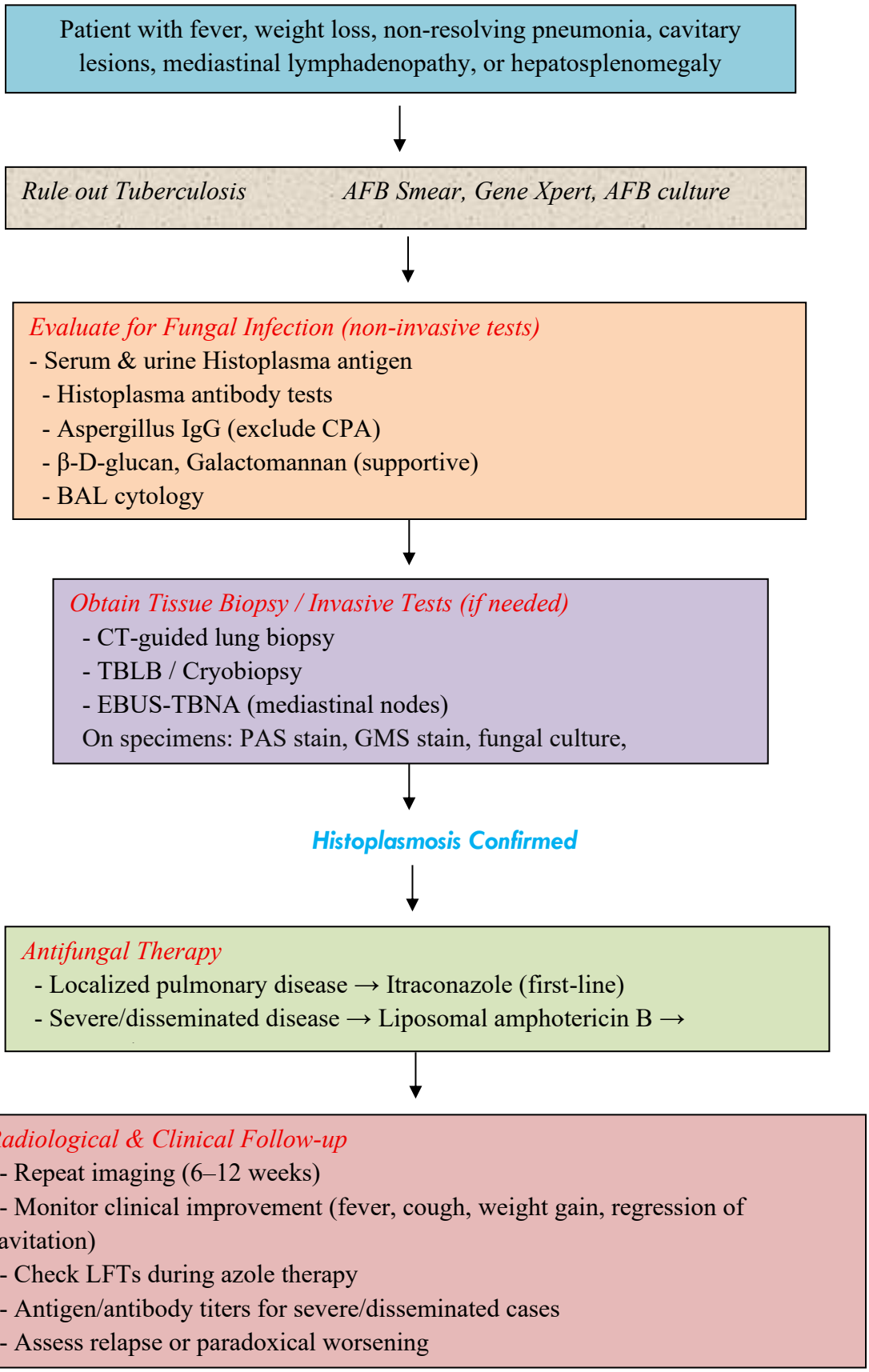
TREATMENT AND FOLLOW-UP

Treatment was individualized based on disease severity and extent:

- Localized pulmonary disease: oral itraconazole or posaconazole
- Disseminated or severe disease: intravenous amphotericin B, followed by or combined with azole therapy

Clinical and radiological follow-up was recorded, including duration of therapy, response, and survival outcomes (Figure 4)

Figure 4 :diagnostic and treatment algorithm for pulmonary / disseminated histoplasmosis in tb endemic regions



Results:

DEMOGRAPHIC AND CLINICAL PROFILE

Nine patients with histopathologically confirmed histoplasmosis were identified and included in the study. (Table 1). The median age was 64 years (range 44–77 years).

Table 1: *Summary of Clinico-radiological profile of patients with histoplasmosis*

S. No.	Age	Symptoms	Co-morbidities	HRCT	Serology	BAL	Biopsy	Treatment	Follow-up	Prognosis
1.	54	Fever, right side Chest pain, Weight loss	PTB	Right lower lobe Consolidation with cavitation	Negative	• Culture: klebsiella	Ct guided biopsy: histoplasmosis	Itraconazole	3 months	Clinical and radiological improvement
2.	74	Fever, cough with sputum, weight loss	DM, SHTN, CAD	Right lower lobe consolidation with cavitation+ Hilar Lymphadenopathy	Aspergillus IgG positive	• Culture: klebsiella • Galactomannan positive	Ebus: histoplasmosis	Posaconazole	4 months	50% clinical and radiological improvement
3.	68	Fever, cough with sputum, hemoptysis	DM	Left upper lobe consolidation with cavitation	• Aspergillus l gG positive • B-d glucan positive	• Negative	Ebb: histoplasmosis	Amphotericin b	2 weeks	Clinical and radiological improvement
4.	64	Fever, cough with sputum, sob	None	Right upper lobe fibrocavitatory lesion+ Mediastinal Lymphadenopathy	Aspergillus IgG positive	• Culture: pseudomonas • Galactomannan positive	Ebus + radial Ebus: histoplasmosis	Amphotericin b and itraconazole	2 weeks	Death
5.	44	Fever, dry cough, right chest pain	Asthma, DM, recurrent pneumonia, PTB	Right upper lobe Consolidation+ Mediastinal Lymphadenopathy	Negative	• Negative	Ebus: histoplasmosis	Itraconazole	2 weeks	Clinical and radiological improvement
6.	72	Cough with sputum, sob, left chest pain	• DM, SHTN, hypothyroid, OSA, post covid • PTB	Left upper lobe consolidation with cavitation	Negative	• Gene X pert: positive	Radial ebus: histoplasmosis	Itraconazole	2 weeks	Clinical and radiological improvement
7.	64	Cough with sputum, sob, Weight loss	• F-ILD, DM, OSA, post covid	F-ILD with left side pneumothorax+ Consolidation	Negative	• Culture: Klebsiella	TBLB: histoplasmosis	Itraconazole+ amphotericin b	2 weeks	Death
8.	59	Hemoptysis, sob, cough with sputum	• DM, SHTN, CAD, PTB	Fibro cavitatory lesion in right upper lobe	Negative	• Fungal pcr: neg • Gene X pert: negative • Culture: neg • Galactomannan positive	Radial Ebus: histoplasmosis	Itraconazole	2 weeks	Clinical and radiological improvement

9.	77	Fever, cough, Weight loss	• DM, SHTN, pemphigus foliaceous, PTB	Thickened costal pleura in lower lobe of right lung with loculated pleural effusion, few ipsilateral hilar and mediastinal lymph nodes.	Negative	• Fungal pcr: neg • Gene X pert: negative • Culture: neg • Galactomannan: positive	Ct guided biopsy: histoplasmosis	Posaconazole	6 months	Clinical and radiological improvement

Table 2: Gender Distribution

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Gender/Cases	**Number**	%
Male	8	88.89
Female	1	11.11
Total	9	100

There was male predominance 88.89% (table 2).

Table 3: Comorbidity

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Comorbidity	**Number**	%
DM	7	77.8
Old PTB	5	55.6
HTN	4	44.4
CAD	2	22.2
OSA	2	22.2
ILD	1	11.1
Recurrent pneumonia	1	11.1
Absent	1	11.1

Eight patients were immunocompromised (88.9%), primarily due to diabetes mellitus (77.8%), prior pulmonary tuberculosis (55.6%) (PTB) followed by hypertension (44.4%), cardiovascular disease (22.2%), OSA (22.2%) and other respiratory diseases including fibrotic interstitial lung disease and recurrent pneumonia (table 3).

Table 4: Symptoms

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Symptoms	**Number**	%
Cough	9	100
Fever	6	66.7
Weight loss	4	44.4
Hemoptysis	2	22.2
Chest pain	3	33.3

Table 4 shows patients presented with cough (100%), fever (66.7%), weight loss (44.4%), hemoptysis (22.2%) and chest pain (33.3%).

RADIOLOGICAL FINDINGS

HRCT Thorax consistently revealed consolidation with cavitation (66.7%), mediastinal or hilar lymphadenopathy (44.4%), fibrocavitatory lesions (22.2%) predominantly in the upper lobes or involving specific segments of the lungs. In some patients, additional findings included fibrotic ILD with superimposed pneumothorax, pleural thickening with loculated pleural effusion (11.1% each.) (table 5).

Table 5: Radiological findings on HRCT Thorax

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HRCT Thorax findings	**Number**	%
Consolidation with cavitation	6	66.7
Hilar or mediastinal lymphadenopathy	4	44.4
Fibro-cavitatory lesion	2	22.2
Fibrotic ILD with pneumothorax	1	11.1
Loculated pleural effusion	1	11.1

Overall, the radiological picture frequently mimicked active or post-TB sequelae, chronic necrotizing infection, or fungal colonization.

MICROBIOLOGICAL AND SEROLOGICAL FINDINGS

Table 6: Microbiological and Serological Findings

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Serology & Microbiology	**Number**	%
Serum Aspergillus Ig G	3	33.3
BAL Galactomannan	4	44.4
Serum β-D-glucan	1	11.1
BAL Culture Klebsiella	3	33.3
Bal Culture Pseudomonas	1	11.1
Gene Xpert Positive	1	11.1

Table 6 shows serological and microbiological data. Aspergillus IgG was positive in 33.3% patients, BAL galactomannan in 44.4% and serum β-D-glucan in 11.1% patients, reflecting co-colonization or cross-reactivity. Bacterial cultures yielded organisms such as *Klebsiella pneumoniae* or *Pseudomonas aeruginosa* in 4 patients (44.4%) GeneXpert for *M. tuberculosis* was positive in 1 patient (11.1%) while fungal PCR was variable.

Despite these confounding findings, definitive diagnosis in all patients required histopathological confirmation.

HISTOPATHOLOGICAL DIAGNOSIS

In every case, tissue biopsy (via CT-guided lung or pleural biopsy, EBUS-guided sampling, radial EBUS-guided biopsy, or TBLB) demonstrated features consistent with histoplasmosis (table 7). On H&E staining, macrophages containing intracellular oval yeast forms were identified, and these were highlighted on PAS and GMS stains (Figure 1).

Table 7: Diagnostic Tests

Table 7: Diagnostic Tests		
Tests performed	**Number**	%
CT guided biopsy	2	22.2
Peripheral EBUS guided Biopsy	3	33.3
Central EBUS guided biopsy	2	22.2
Endobronchial biopsy	1	11.1
Transbronchial lung biopsy	1	11.1

TREATMENT AND OUTCOMES

Treatment regimens included Itraconazole monotherapy in 44.4% patients with localized pulmonary disease, Posaconazole in 22.2% patients, particularly with pleural involvement or prolonged disease, Amphotericin B in 33.3% patients, either alone or followed by itraconazole, in those with more severe or disseminated disease or significant immunosuppression.

Seven of nine patients showed clinical and radiological improvement with antifungal therapy over follow-up periods ranging from 2 weeks to 6 months. Two patients with advanced comorbidities and severe pulmonary compromise died despite treatment, resulting in a mortality rate of approximately 22%.

Discussion

Histoplasmosis represents a major diagnostic pitfall in TB-endemic regions such as India. In our series, all patients presented with pulmonary symptoms and imaging findings—consolidation, cavitation, fibrocavitatory lesions, and mediastinal lymphadenopathy—that closely resembled active or post-tuberculosis disease. Notably, more than half of our patients had a prior history of pulmonary TB, and several had received empirical anti-tubercular therapy before the correct diagnosis was established. This reinforces the clinical reality that histoplasmosis remains under considered in routine practice, particularly in regions perceived as non-endemic.

WHY PREDOMINANTLY PULMONARY DISEASE IN DELHI-NCR?

Unlike reports from Rajasthan and eastern India, where adrenal and disseminated histoplasmosis are frequently described, our cohort predominantly exhibited pulmonary involvement. This difference likely reflects a combination of host and environmental factors. Diabetes mellitus, prior TB, fibrotic interstitial lung disease, and chronic airway damage were common in our patients, suggesting that altered pulmonary architecture and impaired local immunity may predispose to localized pulmonary infection rather than systemic dissemination. Urban exposure patterns, including construction activity and soil disruption, may also contribute to inhalational exposure in Delhi-NCR, although environmental sampling was not performed.

LIMITATIONS OF NON-INVASIVE DIAGNOSTICS

Our experience highlights the limited reliability of serological and biomarker-based tests in real-world Indian settings. Galactomannan, β-D-glucan, and Aspergillus IgG were variably positive, likely reflecting cross-reactivity, fungal co-colonization, or advanced lung disease rather than true invasive aspergillosis[21]. Similarly, fungal cultures and PCR assays were often negative. As a result,

histopathology emerged as the definitive diagnostic modality in all cases. This finding underscores the need for early tissue diagnosis in patients with non-resolving pneumonia or cavitary lung disease, particularly when TB investigations are negative or discordant.

COMPARISON WITH INDIAN DATA

Indian literature describes a wide clinico-anatomical spectrum of histoplasmosis. Case series from Rajasthan have reported disseminated histoplasmosis in immunocompetent hosts and adrenal histoplasmosis frequently associated with adrenal insufficiency.[22,23] A five-year experience from Kolkata documented nineteen cases of adrenal histoplasmosis, predominantly in middle-aged to older men.[24] Cutaneous, adrenal, and disseminated forms have also been described in immunocompetent as well as immunocompromised individuals.

In contrast, our Delhi-NCR cohort predominantly exhibited pulmonary involvement in patients with underlying immunosuppression (diabetes, prior TB, chronic lung disease, or systemic immunosuppression). This difference in anatomical predilection (pulmonary vs adrenal vs disseminated) underscores the interplay between environmental exposure, host immune status, and regional patterns of disease.

INTERNATIONAL PERSPECTIVE

In the United States, histoplasmosis is endemic in the Ohio and Mississippi river valleys, where the majority of cases are pulmonary.[1,3] Mortality among hospitalized patients is reported at approximately 5–7%.[25] In Southeast Asia and Latin America, histoplasmosis is increasingly recognized in association with HIV infection, with many patients presenting with progressive disseminated disease and higher mortality.[26]

Compared with these international data, the Indian experience reflects a broader spectrum, ranging from self-limited pulmonary disease to adrenal involvement and disseminated infection in both immunocompetent and immunocompromised hosts. Our series adds to this body of evidence by documenting predominantly pulmonary disease in a region (Delhi-NCR) not traditionally regarded as endemic.

MORTALITY AND OUTCOMES

The overall mortality in our series was approximately 22% (2 of 9 patients), higher than that reported in hospitalized cohorts from endemic regions in the US (5–7%)[25] but similar to Indian tertiary-care data, where mortality has been reported around 18%.[17] Among immunocompromised populations, particularly people living with HIV/AIDS, mortality rates as high as 21–53% have been described.[27] Our findings are consistent with the poorer outcomes observed in immunosuppressed individuals, likely reflecting the combined impact of advanced age, diabetes, prior TB, chronic lung disease, and delayed diagnosis (Figure 5).

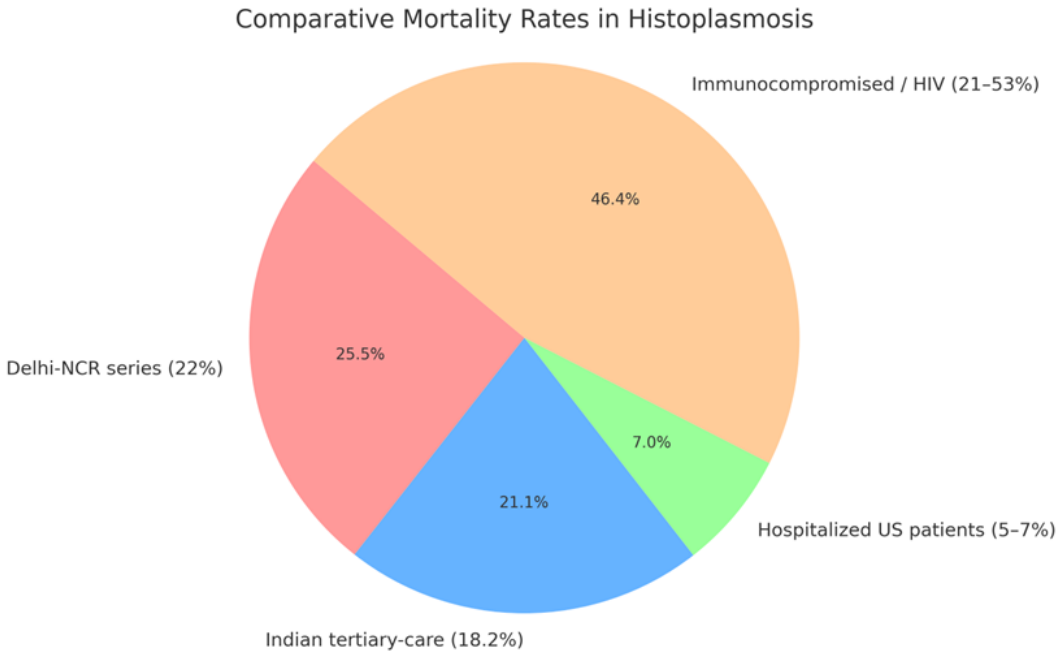

Figure 5: Comparative mortality rates in histoplasmosis across different cohorts

STUDY LIMITATIONS AND FUTURE DIRECTIONS

This study has several limitations. It represents a small, single-centre, retrospective case series and may not generalise to the wider population. Fungal cultures and antigen detection assays were not uniformly available, and molecular (PCR-based) diagnostics were limited. Environmental sampling was not performed.

Future directions include multicentre epidemiological studies to clarify the true burden and geographic distribution of histoplasmosis in India, particularly in North Indian regions such as Delhi-NCR. Improved access to rapid antigen detection and molecular diagnostics could facilitate early diagnosis. Prospective inclusion of histoplasmosis in the

differential diagnosis of non-resolving pneumonia and cavitary lung lesions should be encouraged.

Conclusion:

The clustering of cases from Delhi-NCR suggests under diagnosis of histoplasmosis due to limited availability & lack of sensitivity and specificity of available non-invasive serological markers. Heightened clinical vigilance, early tissue diagnosis, timely antifungal therapy, and further epidemiological and environmental studies are urgently needed to better characterize and manage this potentially underrecognized infection.

Conflicts of Interest:

There are no conflicts of interest.

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