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Histoplasmosis – More common than we realize

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ABSTRACT

Histoplasmosis is primarily associated with immunocompromised individuals; however, its presentation in immunocompetent patients is increasingly recognized. This series of 5 cases from eastern India, a potential Histoplasmosis hotspot, describes five immunocompetent individuals with the disease. It emphasizes the diverse clinical spectrum of Histoplasmosis, often mimicking other conditions, thereby complicating diagnosis. Four patients presented with adrenal masses, emphasizing the importance of considering histoplasmosis in the differential diagnosis of adrenal enlargement. One patient developed hemophagocytic lymphohistiocytosis (HLH), underscoring the severe complications associated with disseminated histoplasmosis. Additionally, one patient exhibited localized disease, demonstrating the variable clinical presentations of this infection. Our findings emphasize the need for heightened clinical suspicion of histoplasmosis in patients with adrenal masses or unexplained fever, even in immunocompetent individuals in a relevant epidemiological setting. Early diagnosis and appropriate antifungal therapy are crucial for optimal outcomes.

Introduction

Histoplasmosis is a global disease endemic to regions of all six inhabited continents. Most epidemiological research on histoplasmosis has been conducted in the United States, which has a widespread geographic presence. Recent compilations of several previous studies have revealed a scattered distribution of histoplasmosis cases worldwide, with case series reported in Brazil, South Africa, and India, and isolated cases in Central and South America, northern sub-Saharan Africa, Oceania, and Europe [1]. Although Randhawa's 1970 review identified 30 autochthonous cases of histoplasmosis in Southeast Asia, the epidemiology of this fungal infection in the region has remained relatively unexplored [2].

In India, cases are likely underdiagnosed and under-reported. Still, areas of high prevalence include the Eastern states of West Bengal and Assam, through which the Ganges and Brahmaputra rivers flow. While histoplasmosis has a specific epidemiologic niche, several other states in the eastern and northern parts of India like Bihar, Delhi, Haryana, Rajasthan, Punjab, and Uttar Pradesh, have documented cases [3]. India may represent a region of underdiagnosis [4]. In this study, we identified five individuals, all residents of the Eastern part of India with histoplasmosis between Dec 2022 and April 2024. Notably, all these patients were HIV-negative but had underlying health conditions such as diabetes or renal impairment.

Case reports

Case 1

A 59-year-old diabetic male subject presented with respiratory distress for the past two days which was progressive. He was clinically diagnosed with pulmonary tuberculosis four months ago and has been and has been taking anti-tuberculosis medication since then. At the time of admission, his vitals were stable and oxygen saturation was found to be 98 % on room air. Chest X-ray showed bilateral pleural effusion and a small pericardial effusion. A whole-body positron emission tomography (PET) scan to look for occult malignancy or infection revealed FDG (fluorodeoxyglucose) avid ground glass opacity in the right upper lobe of the lung (anterior segment), and FDG enhancing well-defined mediastinal nodes. Bilateral adrenal masses were also noted. CT-guided biopsy was performed from the adrenal mass and sent for relevant investigations. The histopathological section showed predominantly necrosis and scattered foamy histiocytes studded with intracellular yeast like spores [Fig. 1]. Fungal stain revealed the presence of numerous budding yeast cells 2–4-μm, ovoid, uninuclear with thin walls and narrow-based budding inside foamy macrophages, consistent with

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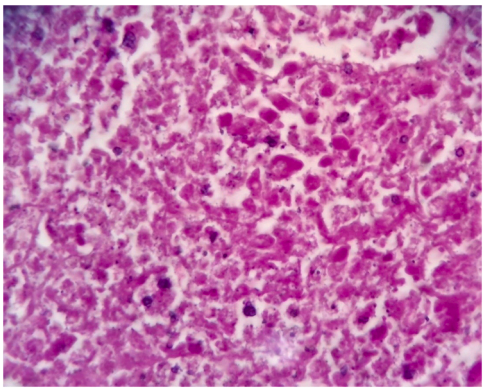

Fig. 1. H and E, 40X section showed predominantly necrosis and scattered foamy histiocytes studded with intracellular yeast-like spores.

Histoplasma. The sample was negative for tuberculosis (cartridge-based nucleic acid amplification test, *Xpert MTB/*RIF assay, Cepheid), other bacterial pathogens, and malignancy. Following the diagnosis of histoplasmosis, the patient was started on liposomal amphotericin B (4 mg/kg body weight/day), and anti-tubercular drugs were discontinued. The patient improved clinically and symptomatically. He received amphotericin B for 2 weeks and was discharged with the advice to take oral itraconazole 200 mg bd for one month. At follow-up, at one and six months the patient was doing well.

Case 2

A 71-year-old (recently diagnosed diabetic patient on oral hypoglycaemics) with renal impairment presented with fever, shortness of breath, and altered sensorium for the past two days. Further history revealed that he was having intermittent fever associated with anorexia, nausea, and vomiting for the last 3 months. He was admitted to two other hospitals in previous month but without any improvement. The laboratory workup at admission showed pancytopenia, liver insufficiency, and high triglycerides. Increased ferritin levels were also detected (70073 *n*g/dL, reference value: 24–336 *n*g/dL). An ultrasound scan of the abdomen showed mild hepatosplenomegaly and a well-circumscribed hypoechoic lesion in the right suprarenal.

CT scan of the abdomen revealed hepatosplenomegaly, adrenal SOL (3.8 cm × 2 cm), and bilateral lung nodularity along with mediastinal lymphadenopathy. Further workup with bone marrow biopsy showed scattered macrophages containing yeast-like microorganisms (Fig. 2A) also highlighted by fungal stains. Also noted were macrophages showing hemophagocytosis. (Fig. 2B). Histoplasma urinary antigen was found to be positive. Based on the biopsy findings along with other biochemical markers assay, the patient was diagnosed with Histoplasma-induced HLH. Liposomal Amphotericin B was started but the patient expired 3 days later.

Case 3

A 63-year-old male patient presented with bilateral pedal swelling and generalized weakness for the past 3 weeks. Significant findings include hepatosplenomegaly and cervical and axillary lymphadenopathy. Routine investigations showed pancytopenia (Table 1) and renal dysfunction with hypercalcemia and raised alkaline phosphatase.

PET-CT findings revealed FDG avid bilateral suprarenal and cervical masses, right axillary lymph nodes, and FDG avid lingual lymphoid tissue. There was diffuse FDG uptake in bone marrow as well suggestive of disseminated inflammatory or neoplastic condition. Bilateral minimal pleural effusions and hepatosplenomegaly were also identified. HRCT revealed a small area of focal basal and segmental atelectasis at both lower lobes with minimal pleural effusion.

The conditions considered in the differential diagnosis included infectious diseases such as tuberculosis and histoplasmosis, metastasis from an unidentified primary site, lymphoma, and bilateral adrenocortical carcinoma.

A cervical lymph node biopsy revealed effaced architecture by sheets of histiocytes (Fig. 3A) containing numerous intracytoplasmic yeast-like forms (arrow Fig. 3B) highlighted by fungal stains, a finding consistent with *Histoplasma capsulatum*. Treatment was initiated with intravenous amphotericin B but his condition worsened. He developed altered mental status and deteriorating kidney parameters. Haemodialysis was initiated, but the patient ultimately succumbed.

Case 4

A 66-year-old diabetic patient (controlled with anti-diabetics) presented with a history of significant weight loss (10 kg over the past two months) associated with generalized weakness and loss of appetite. Clinical examination was significant for anemia and cervical lymphadenopathy. Suspecting occult malignancy, a whole-body PET CT scan was done which revealed FDG avid bilateral adrenal nodules along with bilateral neck nodes. A CT-guided biopsy from the adrenal nodule was done. The histopathology of the adrenal gland was predominantly necrotic, with moderately mixed inflammatory cells, foreign body type multinucleated giant cells, and histiocytes containing multiple intracytoplasmic yeast-like bodies (Fig. 4A) highlighted by fungal stains (Fig. 4 B). Subsequently, Histoplasma capsulatum var capsulatum was

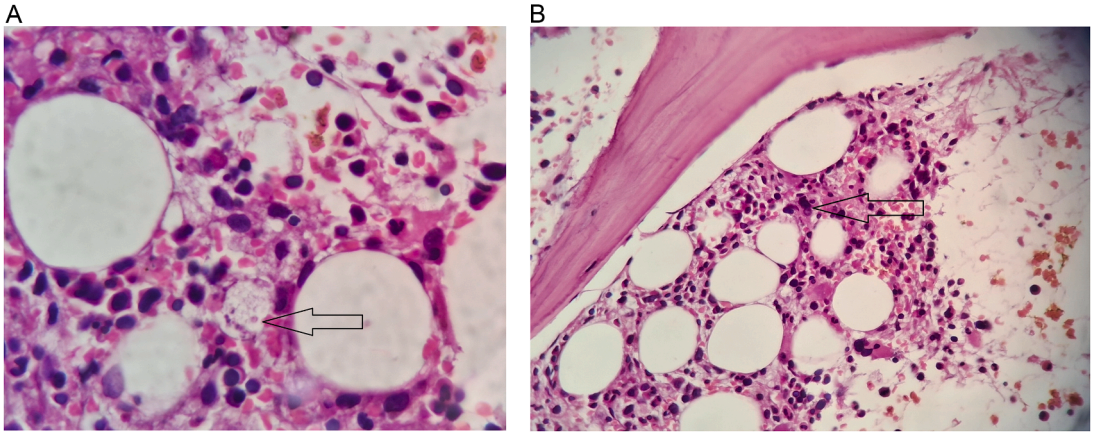

Fig. 2. A H and E, 40X Bone marrow biopsy showed scattered macrophages containing yeast-like microorganisms. B H and E, 40X Bone marrow biopsy showed macrophages with hemophagocytosis.

Table 1
depicts the laboratory and Radiological findings of patients at the time of presentation.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Haemoglobin (mg/dl)	9.5	8.3	7.8	10.6	10.5
Total Count /cu mm	12700	1200	2800	5800	6400
Platelet Lacs/cu mm	1.7	0.18	1.2	2.06	2.1
Bilirubin mg/dl	0.7	8.5	1.5		
ALT U/l	26	47	23		
AST U/l	30	138	21		
ACTH mcg/dl				27.3	
Cortisol mcg/dl				11	
Blood sugar (random) mg/dl	140	61	97	163	89
HBA1C	6.4			6.3	
Fibrinogen mg/dl		279			
Ferritin ng/ml		70073	1295		
Triglycerides mg/dl		589			
CT scan					
Bone marrow		Features of HLH with scanty yeast cells	Reactive bone marrow with epitheloid granuloma		
PET CT			Lymphadenopathy (cervical, inguinal, axillary), bilateral adrenal SOL		
Adrenal aspirate	Foamy macrophages studded with fungi			Histiocytes Containing multiple intracytoplasmic yeast-like bodies	
Lymph node aspirate			Yeast cells		
Tissue from oral lesion					Round to oval yeast-like organisms noted inside macrophages
Urea mg/dl	22	86	243	23	21
Creatinine mg/dl	0.9	2.7	3.4	1.2	1.1
Histoplasma urinary antigen ng/ml	Not done	Positive 0.93	Not done	Positive 0.89	Not done
Growth in SDA	Yes	No	Yes	Yes	Not done

Normal Range: Hb 13–17 mg/dl; Total Count 4000–10000 /cu mm; Platelet 1.5–4 lacs/cumm; Blood glucose (random) 70–140 mg/dl; Urea 17 – 49 mg/dl; Creatinine 0. 8–1.3 mg/dl;
Bilirubin 0.3–1.2 mg/dl; ALT < 50 U/l; AST < 50 U/l; Fibrinogen 200–400 mg/dl; Ferritin 24–336 ng/ml; Triglycerides upto150 mg/dl; ACTH 7.2 – 63.3 mcg/dl; Cortisol 07–10 AM: 6.7–22.6 mcg/dl: 04–08 PM: < 10 mcg/dl
Histoplasma urinary antigen; Positive cut off ≥ 0.5 ng/ml

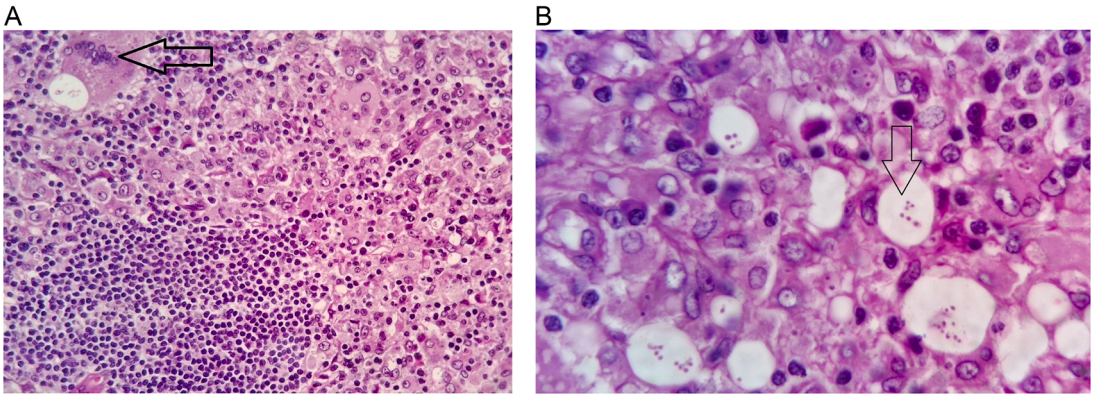

Fig. 3. A H and E, 40X cervical lymph node biopsy revealed effaced architecture by sheets of histiocytes. The arrow points at multinucleated giant cell B H and E, 40X cervical lymph node biopsy showed histiocytes containing numerous intracytoplasmic yeast-like forms (pointed by arrow head).

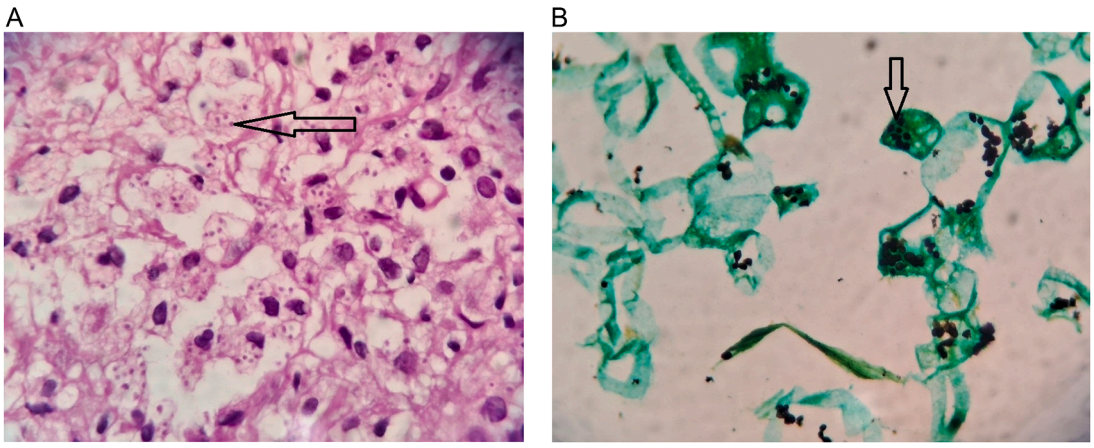

Fig. 4. A H and E, 40X arrowhead shows histiocytes containing multiple intracytoplasmic yeast-like bodies. B GMS stain, 40X showed histiocytes containing multiple intracytoplasmic yeast-like bodies.

recovered in culture. The patient was administered liposomal amphotericin B for 2 weeks followed by oral itraconazole.

Case 5

A 65-year-old non-smoker man involved in operating a farm and marketing its products presented with a non-healing painful progressive ulcer of the buccal mucosa near the left angle of the mouth for the last six months. Besides weakness, he reported no other constitutional symptoms such as fever, weight loss, shortness of breath, etc. He was variously treated with systemic, topical antibiotics, and topical steroids. As there was no relief of symptoms and the ulcer progressed so he sought treatment in our hospital. A well-defined tender, indurated ulcer of size 6 cm x 6 cm on the buccal mucosa adjacent to the left angle of mouth associated with left cervical lymphadenopathy was detected on examination. There were no other systemic signs and symptoms. All radiological investigations (Chest skiagram and ultrasound abdomen) and blood parameters (Table 1) were within normal limits. An incisional biopsy was done from the lower lip buccal mucosa. Histopathological examination showed ulcerated mucosa with dense mixed inflammation, multinucleated Langhans-type giant cells, and noncaseating epitheloid cell granulomata (Fig. 5A) in the submucosa. Round to oval yeast-like organisms highlighted by fungal stains were noted in the macrophages (Fig. 5B). Intracellular yeast cells, consistent with *Histoplasma capsulatum*, were identified using PAS-Diastase and Gomori's methenamine silver stains, confirming oral histoplasmosis.

Itraconazole therapy (100 mg BD) proved highly effective for this patient. The ulcer healed rapidly within 2 weeks with no recurrence observed at follow-up.

Discussion

Histoplasma was discovered in 1905 by Samuel T. Darling but only in the 1930s was it discovered to be a widespread infection. *H. capsulatum* is a dimorphic fungus that occurs as mold in the environment and yeast-like structures with septate hyphae at 37 degrees Celsius in tissues [5]. Human infection occurs after the fungus (in the form of microconidia or hyphal fragments) is inhaled, and reaches the alveoli where it is transformed into the yeast phase. Most subjects who acquire the infection remain asymptomatic but patients who become symptomatic have substantial risk of mortality and severe morbidity depending on the host's immune status.

The areas of highest endemicity for Histoplasmosis lie within the Mississippi and Ohio River Valleys of North America and parts of Central and South America like Brazil, Ecuador, Venezuela, Paraguay, Uruguay, and Argentina [6]. In India, the Gangetic region of West Bengal seems to be endemic to this fungus. A study by Sanyal et al. found that 9.4 % of the population tested positive in the histoplasmin skin test, indicating prior exposure to the fungus [7]. In a separate study, the same researchers isolated *H. capsulatum* from local soil, proving that the fungus thrives in the region's ecological niche [8]. All of our patients are from West Bengal and have not travelled outside the state in a manner that

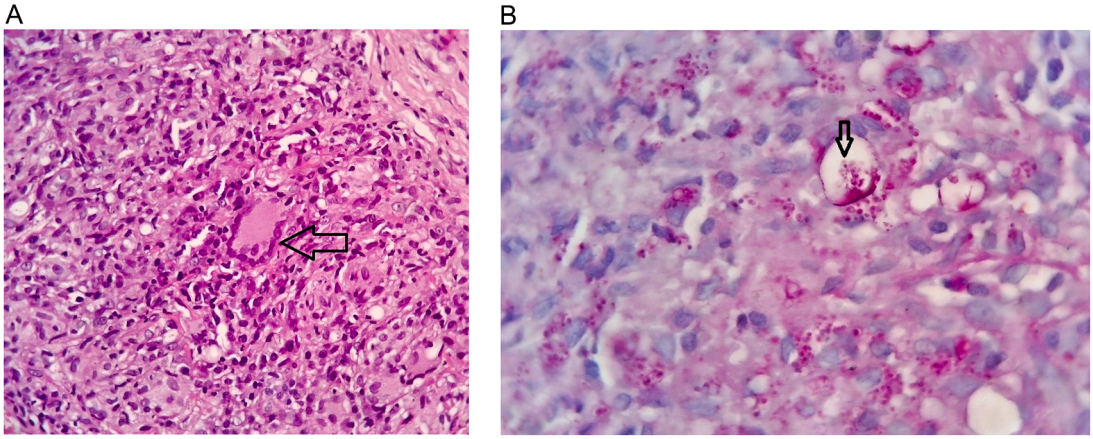

Fig. 5. A H and E stain, 40X arrowhead shows multinucleated Langhans-type giant cells and noncaseating epitheloid cell granulomata. B PAS stain, 100X arrowhead shows round to oval yeast-like organisms inside the macrophages.

could have exposed them to the infection and in all probability the infection was acquired locally.

Histoplasmosis is often difficult to diagnose because its symptoms are similar to many other illnesses. This fungal infection can mimic community-acquired pneumonia, tuberculosis, lymphoma, sarcoidosis, and cancer [9].

Risk factors

Histoplasma infection is acquired by inhaling spores, and the host's immune status determines the severity of symptoms and clinical manifestations. Clinical classification of Histoplasmosis include acute pulmonary infection, chronic pulmonary disease, and progressive disseminated histoplasmosis. Progressive disease can also be classified as acute, sub-acute or chronic.Although most infections are asymptomatic, histoplasmosis can be severe and progressive in patients who have experienced heavy exposure to the spores, have underlying immune defects such as HIV/AIDS, or those taking immunosuppressive medications, such as corticosteroids, anti-rejection drugs, TNF-alpha inhibitors, and cancer chemotherapeutic agents. Patients with symptomatic infection that is not recognized and treated may also develop progressive disease [10]. Histoplasma can also exist inside the host in a latent form with the potential for reactivation [9].

The four cases with disseminated Histoplasmosis elaborated here did not have any of the above-described risk factors. All the patients were HIV nonreactive. The only other identifiable risk factor in these patients is diabetes (though controlled as is evident from the laboratory findings) and two of the patients had chronic renal failure. None of the identified risk factors were present in the fifth patient with a localized infection. All five patients presented with non-specific symptoms like fever, weight loss, and generalized weakness.

Diabetes mellitus is not well described as a risk factor for disseminated histoplasmosis. However, several case reports do mention diabetes as being the only comorbidity [11].

Symptomatic Histoplasma can have various presentations ranging from acute pulmonary infection, disseminated disease, and chronic pulmonary infection. Similar to the fifth patient who presented solely with an oral ulcer, other localized variations of the disease might exist.

Adrenal involvement is frequently seen in disseminated histoplasmosis but symptomatic adrenal insufficiency is not necessarily present. Adrenal involvement is a prevalent manifestation of histoplasmosis in regions where the fungus is endemic. Adrenal histoplasmosis should be considered as one of the differential diagnoses in cases of adrenal masses with intense FDG uptake on PET scans. Adrenal insufficiency occurs in 20–50 % of cases[10]. Adrenal involvement is frequently observed, particularly in immunocompetent subjects. The reason why *Histoplasma* fungus preferentially infects the adrenal glands remains unknown. However, it is postulated that the high levels of glucocorticoids in adrenal cells, combined with the limited presence of reticuloendothelial cells, may play a role [11]. There was no clinical evidence of adrenal insufficiency in our patients with disseminated infection. In only one of the patients, a workup for adrenal function was done and the results were within normal limits.

Histoplasma causing HLH is a severe complication and is associated with high mortality [12]. This is caused by uncontrolled activation of the T cell resulting in a cytokine storm. The clinical features of HLH mimic that of severe sepsis and thus HLH may be missed in the setting of an overwhelming infection. Patient 2 exhibited clinical presentation and laboratory parameters suggestive of Hemophagocytic Lymphohistiocytosis (HLH), characterized by fever, an enlarged spleen, and abnormally high levels of ferritin and triglycerides. Further investigations were warranted to confirm the diagnosis. The identification of hemophagocytes within the bone marrow, as assessed by biopsy, fulfilled the diagnostic criteria for Hemophagocytic Lymphohistiocytosis (HLH) as outlined in the 2004 protocol. HLH triggered by Histoplasma has mostly been reported in the background of HIV. There is no current consensus regarding treatment options for infection-associated HLH apart from the fact that the inciting infection is to be treated with appropriate antimicrobials. Finding the right course of action for eliminating the infection and controlling the immune reaction at the same time is complex and there is no strong recommendation as to how to balance the two offenders in case of infection-induced HLH. Histoplasma being an intracellular pathogen, the possibility of HLH should be kept in mind. Patients with low counts and high inflammatory markers like ferritin should undergo investigations for HLH.

Diagnosis and treatment

A definitive diagnosis of histoplasmosis through laboratory tests is crucial for effective treatment. In case of disseminated infection obtaining an aspirate from lymph nodes, adrenal mass, or bone marrow may clinch the diagnosis quickly by demonstrating typical intracellular yeast cells with a single narrow-based bud by special stains like GMS. Sensitivities of the direct visualization tests can vary widely (9–50%) depending on tissue type and disease syndrome [13]. The samples may be cultured in SDA agar to recover Histoplasma capsulatum. Culture is a slow process that may take up to 4 weeks for demonstrable growth and requires class 3 biosafety facilities. In all our patients' direct evidence for Histoplasma was obtained from lymph nodes, bone marrow, adrenal aspirate, and tissue biopsy in case of the oral lesion. In three of the patients, Histoplasma capsulatum was recovered in culture. Another test to detect disseminated Histoplasmosis is urinary antigen detection by EIA. The recommendation is to perform urinary and serum Histoplasma antigens in all patients with suspected disseminated histoplasmosis. The test's sensitivity depends on the extent of infection and can range from 30 % to 95 % [14]. The detection of Histoplasma urinary antigen is a good screening test in patients with a strong suspicion of disseminated Histoplasmosis. Performance of the antigen detection test in immunocompromised people is excellent (93.1%, 81/87) but less sensitive (73.3%, 11/15) among people without known immune-compromised condition [13]. A positive antigen detection test can be followed by histopathologic/ cytopathologic assessment and fungal culture. However, it has to be kept in mind that a negative antigen assay for Histoplasma does not exclude the diagnosis of Histoplasmosis since the sensitivity of the various commercially available assays is variable.

Treatment is indicated in all cases of disseminated disease. Lipid formulations of Amphotericin B are indicated in sick patients requiring hospitalization which can be stepped down to itraconazole after 1 or 2 weeks of therapy when patients have become afebrile and other vital parameters have stabilized.6 In all four of our cases of disseminated histoplasmosis, Amphotericin B was initiated following the diagnosis but two of the patients succumbed to the infection. Several factors, including shock, delayed diagnosis, acute kidney injury, mechanical ventilation need, and hemophagocytic lymphohistiocytosis (HLH), significantly worsen patient survival. Two patients had adverse outcomes. One, with a late presentation and HLH, and the other, with chronic kidney disease and widespread infection succumbed to the disease. In the remaining two surviving patients of disseminated disease, Amphotericin B was substituted with oral itraconazole at discharge 7–14 days later. The patient with the localized disease had an excellent response to oral itraconazole.

Conclusion

Histoplasmosis presents a diagnostic challenge due to its varied clinical manifestations and potential for mimicry of other diseases. This study highlights the importance of maintaining a high index of suspicion for histoplasmosis in individuals with compatible symptoms in a relevant epidemiological setting. Early diagnosis and prompt initiation of antifungal therapy are crucial for preventing severe complications and mortality. Furthermore, efforts should be directed to utilize rapid diagnostic tests such as Histoplasma urinary antigen detection in all

cases of suspected disseminated infection, and for subsequent follow-up, and improve our understanding of the pathogenesis of the disseminated disease, and also explore the potential for preventative measures. By raising awareness and establishing diagnostic tools or algorithms, we can effectively combat histoplasmosis and minimize its burden on public health.

Ethical approval

Ethics approval was obtained for this case report.

Consent for publication

The authors consent to have this paper published.

Patient consent

Patient consent was acquired for this publication.

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Declaration of Generative AI and AI-assisted technologies in the writing process

During the preparation of this work the author(s) used Grammarly in order to check spellings and for grammatical correction. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

CRediT authorship contribution statement

Ujjwayini Ray: Writing – original draft, Methodology, Formal analysis, Data curation. **Soma Dutta:** Writing – review & editing. **Arpita Sutradhar:** Writing – review & editing, Investigation, Data curation.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper

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Data availability

Consultation documents pertaining to the case are available upon request.

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