

Role of the Fungal Serological Marker Galactomannan Antigen in the Diagnosis of Invasive Fungal Infection: A Three-Year Retrospective Study

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Abstract

Background and objectives

Invasive fungal infections (IFIs) are the most prevalent potentially fatal opportunistic mycoses in immunocompromised individuals. Early detection is essential for treating these patients. Biomarkers, measured in blood or other clinical samples, are effective supplements to conventional pathologic and microbiologic diagnostic methods for IFIs. We conducted this study to determine the positivity of serum galactomannan (GM) in clinically diagnosed IFI. Also, we assessed the diagnostic power of GM for predicting invasive aspergillosis (IA), the most common cause of IFIs.

Methodology

This retrospective study analyzed clinically diagnosed patients with IFIs admitted to Kalinga Institute of Medical Sciences (KIMS), Bhubaneswar, India, from January 2023 to December 2025. The study included the blood samples received for serum GM antigen testing during the study period. We evaluated the diagnostic performance of the GM antigen. We also plotted the receiver operating characteristic (ROC) curve and determined the area under the curve (AUC).

Results

We analyzed data from 172 clinically diagnosed patients with IFIs. *Aspergillus flavus* consistently showed the highest GM levels, followed closely by *Aspergillus fumigatus* and *Aspergillus niger*. The test demonstrated high sensitivity (82.4%) but low specificity (46.8%). The AUC was 0.69. Differences in age, gender distribution, and year-over-year prevalence were statistically significant ($P < 0.001$).

Conclusions

GM antigen detection is useful for identifying IA. It demonstrated improved sensitivity and specificity compared with conventional methods (potassium hydroxide (KOH) and culture). Future multicenter studies incorporating polymerase chain reaction (PCR) and β -D-glucan are warranted to validate these findings.

Categories: Internal Medicine, Infectious Disease

Keywords: aspergillosis, fungal culture, galactomannan antigen, invasive fungal infection, koh mount

Introduction

Invasive fungal Infections (IFIs) are systemic infections in which fungi enter the body, replicate in tissues, causing direct tissue damage as well as inflammatory cascades [1,2]. Over 650 million individuals globally suffer from fungal infections each year; of these, over 7 million have severe IFIs, which cause over 1.35 million fatalities [3]. The use of broad-spectrum antibiotics, immunosuppressants, various catheters, surgery, prolonged hospital stays, glucocorticoids, and serious underlying illnesses such as organ transplantation and hematologic malignancies is strongly associated with the incidence of IFIs [4,5]. *Candida*, *Mucorales*, *Aspergillus*, *Cryptococcus*, and *Pneumocystis* species are the most frequent causal agents [6].

The incidence rates of invasive aspergillosis (IA), the most prevalent IFI, range from 5.5% to 20% [7,8]. The primary risk factors for IA are allogeneic stem cell transplantation and sustained severe neutropenia (neutrophils <500 cells/mL) with long-term corticosteroid use [9,10]. Early detection and intense treatment may improve the prognosis, but IA-related mortality ranges from 36% to 90% [11,12].

The most prevalent *Aspergillus* species that causes IA is *Aspergillus fumigatus*. In untreated or complicated

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cases, *A. fumigatus* may cause hematogenously disseminated fungal infection by spreading from the lungs to other organs [13].

The clinical signs of IFIs are frequently challenging to differentiate from those caused by viral or bacterial illnesses. It is still difficult to diagnose IFI in patients with impaired immune systems. Furthermore, the standard conventional culture procedures have shortcomings and lack sensitivity in diagnosing IFIs. Non-culture-based methods (PCR-based DNA detection or fungal serological biomarker) in blood or respiratory samples are crucial supplementary tests [14,15].

Fungal serological tests include tools that detect antigens of fungal pathogens or antibodies formed by the host to the corresponding invading pathogen [16]. These tests can be used as screening tools, as standalone diagnostics, or in combination with culture and microscopy to confirm a fungal infection [16-18]. However, variables like the test's timing in relation to antifungal therapy or the course of the infection can affect test sensitivity, potentially producing false-positive or false-negative results [17].

The cell walls of *Aspergillus* species contain a polymer called galactomannan (GM). The *Aspergillus* galactomannan antigen titer (AGT) test detects this polysaccharide [19]. Antigen testing is more specific in patients with compromised immunity because, even in cases of IA, there is frequently no *Aspergillus* GM antibody response [20]. Serum and bronchoalveolar lavage fluid versions of the AGT test are commercially available. The physician can select the specimen to use for diagnosis based on the patient's circumstances. Because serum is typically an easily available test, AGT analysis has been demonstrated to greatly enhance the identification of fungal infections in individuals with suspected IFIs [21]. The creation of a straightforward, indirect test for IA detection has sparked intense global debate.

According to studies, the circulating GM can be found five to eight days before aspergillosis manifests clinically [22]. The EB-A2 rat monoclonal antibody is used in the enzyme-linked immunosorbent assay (ELISA) (Platelia test) to detect GM [22]. Results are reported using an optical density index (ODI) [23]. To strengthen the test's specificity without changing its sensitivity, the threshold for serum positivity has been raised to 1 in recent years. The test's sensitivity can reach 71%, and its specificity can approach 90% with an ODI cutoff of 1 [24].

Given the high mortality associated with IA, there is an ongoing need to refine noninvasive, rapid, and reliable diagnostic methods. In this context, the present study was carried out to determine the positivity of GM antigen in serum of clinically diagnosed patients with IFIs. We also assessed the diagnostic power of GM in predicting IA.

Materials And Methods

In this retrospective study, we analyzed patients with clinically diagnosed IFIs admitted to Kalinga Institute of Medical Sciences (KIMS), Bhubaneswar, Odisha, between January 2023 and December 2025. The Institutional Ethics Committee of KIMS granted ethical clearance for the study (reference number: KIIT/KIMS/IEC/2617/2026). Because the study was retrospective, informed consent was not required.

Study procedure

Demographic and laboratory data of the participants were retrieved retrospectively from hospital records and laboratory registers. Demographic data included age and gender. Laboratory data included (serum GM level and interpretation (positive/negative), fungal culture findings). These data were compiled into a structured electronic database for analysis.

As part of routine clinical practices, blood samples from clinically suspected cases were processed in the Microbiology laboratory for IFI detection using serology. The patients were closely monitored throughout their hospital stay to identify sinopulmonary signs and symptoms. Commercially available ELISA kits were used to detect this antigen.

Aspergillus GM antigen was detected in patient serum using an enzyme-linked immunosorbent assay. Serum samples were processed using the Platelia *Aspergillus* Galactomannan EIA kit (Bio-Rad, France) in accordance with the manufacturer's instructions. For each sample, 300 μ L of test serum was mixed with 100 μ L of treatment solution, vortexed, and heated at 120°C for 6 minutes in a dry heat block. Samples were then centrifuged at 10,000 rpm for 10 minutes. Approximately 50 μ L of supernatant was mixed with 50 μ L of conjugate in microtiter wells pre-coated with the monoclonal antibody EBA-2. After incubation at 37°C for 90 minutes, the plates were washed, and 200 μ L of tetramethylbenzidine (TMB) substrate was added. The plates were incubated in the dark for 30 minutes, after which 100 μ L of stopping solution was added to halt the reaction. Absorbance was measured at 450 nm with a reference wavelength of 620 nm. Results were expressed as an optical density (OD) index (ODI) relative to internal controls.

Each run included appropriate positive and negative controls. The index of each test sample was determined by dividing the sample's OD by the mean cutoff control OD; the findings were interpreted as index (I). The

mean cutoff control OD was obtained by adding the OD values for each cutoff control and dividing the result by 2. For both male and female patients, serum samples with an index ≥ 1 and < 1 were regarded as positive and negative for GM antigen, respectively [25].

Statistical analysis

This retrospective study employed convenience sampling. Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), accuracy, and 95% confidence intervals were computed to evaluate the serum GM performance. Analysis of variance (ANOVA) was used to evaluate quantitative variables among more than two groups with normally distributed data. The relationships between qualitative variables were evaluated using the chi-square test (χ^2). Fisher's exact test was applied whenever any expected cell count was fewer than five. Data analysis and plot creation were performed using R software version 4.3.2 (R Foundation for Statistical Computing, Vienna, Austria). *P*-values < 0.05 were considered statistically significant [26].

Results

We analyzed data from 172 clinically diagnosed patients with IFIs. The mean age was 46.6 ± 14.2 years. The study population consisted of 98 males (57%) and 74 females (43%). Forty-nine (28.5%) cases were infected with *A. flavus*, followed by 41 (23.8%) cases with *A. fumigatus* and 35 (20.3%) with *A. niger*. The study spanned three years: 2023 (51 participants), 2024 (68 participants), and 2025 (53 participants). Differences in age, gender distribution, and year-over-year prevalence were statistically significant ($P < 0.001$) (Table 1).

Parameter	Total (n = 172)	<i>Aspergillus flavus</i> (n = 49)	<i>Aspergillus fumigatus</i> (n = 41)	<i>Aspergillus niger</i> (n = 35)	No growth (n = 47)	Statistical test used	Test statistics	P-value
Age (in years)	46.6 ± 14.2	45.2 ± 13.7	48.1 ± 11.6	47.4 ± 12.9	44.8 ± 11.5	ANOVA	10.781	<0.001
Gender, n (%)								
Male	98 (57%)	30 (61%)	27 (66%)	20 (57%)	21 (45%)	Chi-square test	23.413	<0.001
Female	74 (43%)	19 (39%)	14 (34%)	15 (43%)	26 (55%)			
Year, n (%)								
2023	51 (30%)	14 (28%)	11 (27%)	6 (17%)	20 (43%)	Chi-square test	34.508	<0.001
2024	68 (39%)	18 (37%)	18 (44%)	16 (46%)	16 (34%)			
2025	53 (31%)	17 (35%)	12 (29%)	13 (37%)	11 (23%)			

TABLE 1: Demographic data for the study population (n).

Continuous data are shown as the mean and standard deviation (SD). Categorical data are shown as numbers and percentages. Continuous data were analyzed using analysis of variance (ANOVA), and *F*-values were calculated. Categorical data were analyzed using the chi-square test, and chi-square values were calculated. A *P*-value < 0.05 was considered statistically significant.

In Figure 1, the bar plots show the mean GM values for different *Aspergillus* species and their fluctuations over the three years (2023-2025). The error bars show the standard deviations of the means. The mean values are indicated above the corresponding bars. Data from the three years are shown in different colors. *A. flavus* consistently shows the highest GM levels, followed closely by *A. fumigatus*. *A. niger* maintains the lowest mean level among the identified species. Across all three species, there is a distinct pattern in which values either increase slightly or remain stable between 2023 and 2024, followed by a sharp decline in 2025. *A. flavus* reaches a peak mean of 0.573 in 2024 before dropping to 0.479 in 2025. *A. fumigatus* peaks at 0.547 in 2024, dropping to 0.457 in 2025. *A. niger* starts at 0.503 in 2023 and falls to its lowest point of 0.386 in 2025. The GM test is more likely to yield higher readings for *A. flavus* and *A. fumigatus* infections than for *A. niger*.

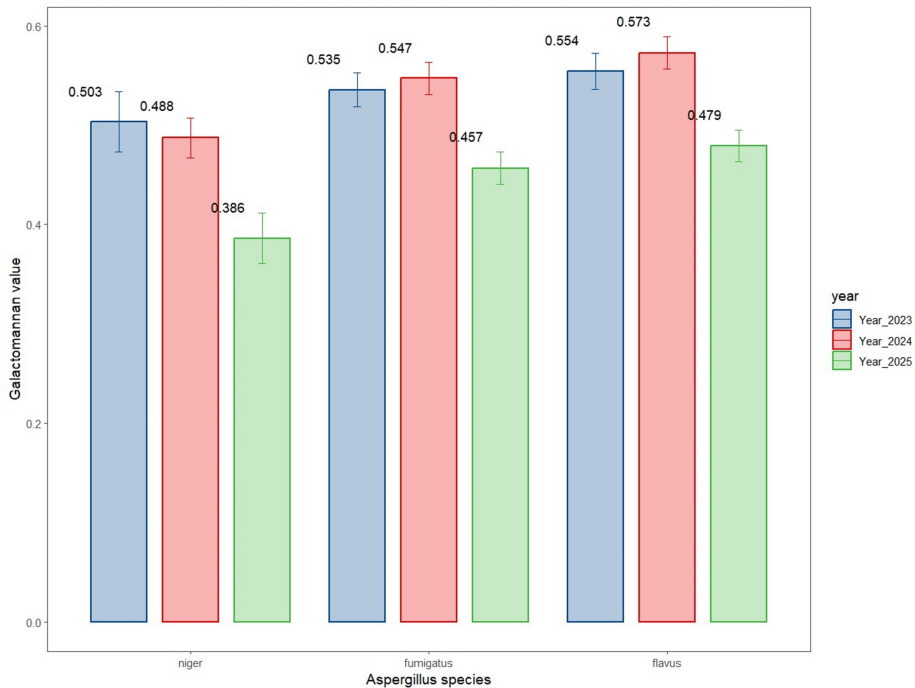


FIGURE 1: Mean galactomannan values of Aspergillus isolates.

The bar plot shows galactomannan values for different *Aspergillus* species.

Table 2 tracks the mean GM values across different species and years. The overall mean GM value was 0.490. *A. flavus* exhibited the highest mean GM value at 0.535, followed by *A. fumigatus* with a mean of 0.518 and *A. niger* with a lower mean of 0.453. For almost every category, GM values peaked in 2024 and were lowest in 2025.

Parameter	Mean	SD	SE	Test statistics	P-value
Total population (n = 172)	0.490	0.091	0.001	23.413	<0.001
Year 2023 (n = 51)	0.506	0.079	0.003		
Year 2024 (n = 68)	0.516	0.093	0.003		
Year 2025 (n = 53)	0.440	0.081	0.003		
<i>Aspergillus flavus</i> (n = 49)	0.535	0.079	0.003	23.413	<0.001
Year 2023 (n = 14)	0.554	0.068	0.010		
Year 2024 (n = 18)	0.573	0.070	0.008		
Year 2025 (n = 17)	0.479	0.066	0.008		
<i>Aspergillus fumigatus</i> (n = 41)	0.518	0.073	0.004	23.413	<0.001
Year 2023 (n = 11)	0.535	0.057	0.010		
Year 2024 (n = 18)	0.547	0.069	0.008		
Year 2025 (n = 12)	0.457	0.056	0.009		
<i>Aspergillus niger</i> (n = 35)	0.453	0.097	0.006	23.413	<0.001
Year 2023 (n = 6)	0.503	0.074	0.025		
Year 2024 (n = 16)	0.488	0.080	0.010		
Year 2025 (n = 13)	0.386	0.091	0.014		
No growth (n = 47)	0.446	0.085	0.004	23.413	<0.001
Year 2023 (n = 20)	0.456	0.073	0.007		
Year 2024 (n = 16)	0.447	0.103	0.013		
Year 2025 (n = 11)	0.425	0.082	0.015		

TABLE 2: Mean galactomannan values of the study population (n) analyzed from 2023 to 2025.

The standard deviation (SD) and standard error (SE) of the mean galactomannan values were calculated. The data were analyzed using analysis of variance (ANOVA), and F-values were calculated. A P-value < 0.05 was considered statistically significant.

In Figure 2, the plot shows sensitivity and 1-specificity on the y-axis and x-axis, respectively. The AUC was 0.69, suggesting that the test has a 69% chance of correctly distinguishing patients with IA from those without the disease. The curve shows high sensitivity but at the cost of specificity. This aligns with the data showing a high sensitivity of 82.4% but a low specificity of 46.8%. Since the brown stepped line is above the diagonal, the GM test is a significantly better diagnostic tool. The AUC of 0.69 and low specificity (46.8%) indicate that the test is an excellent screening tool.

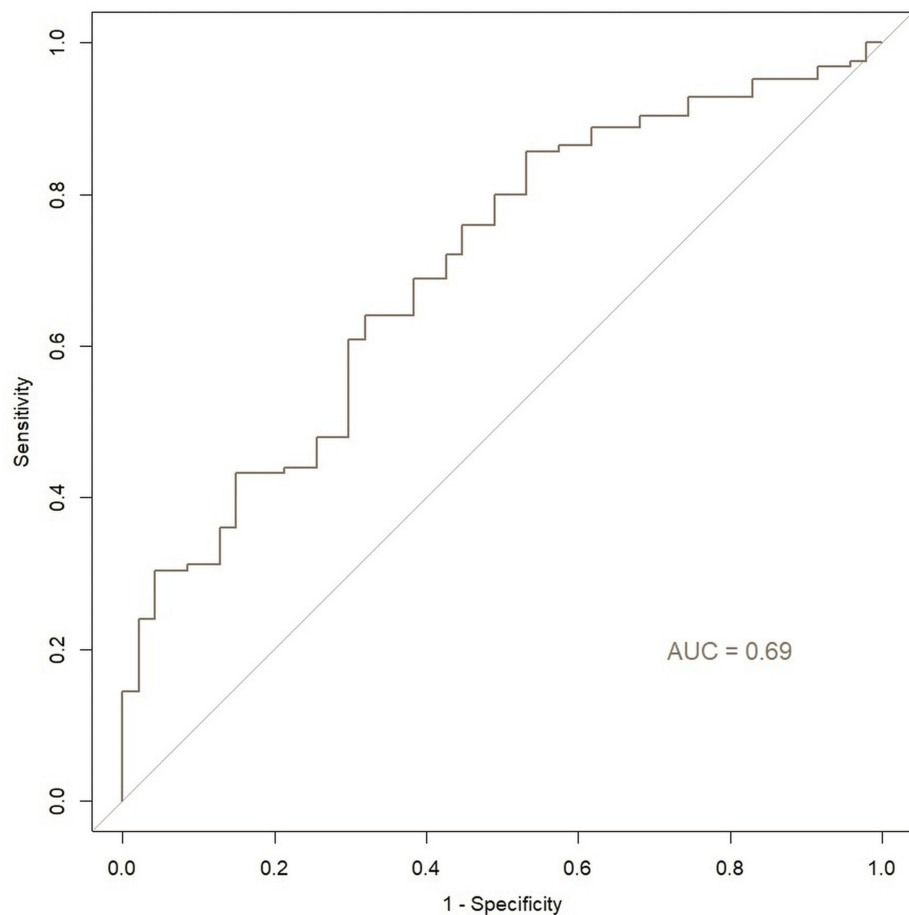


FIGURE 2: Receiver operating characteristic curve for galactomannan.

The x-axis and y-axis represent specificity and sensitivity, respectively.

AUC, area under the curve

Table 3 compares GM test results (positive/negative) with the actual presence of IA.

Parameters	Invasive aspergillosis	No infection	Total
Galactomannan-positive	TP = 103	FP = 25	128
Galactomannan-negative	FN = 22	TN = 22	44
Total	125	47	172

TABLE 3: Contingency table for galactomannan values.

We calculated true-positive (TP), false-positive (FP), true-negative (TN), and false-negative (FN) values for galactomannan in assessing invasive aspergillosis.

Table 4 evaluates the GM test's effectiveness as a diagnostic tool. The test is strong at correctly identifying those with the infection, with a sensitivity of 82.4%. The test is relatively weak at correctly identifying those without infection, with a specificity of 46.8%. The positive predictive value is 80.5%, indicating the highest likelihood of having IA. The negative predictive value is 50%, indicating a 50% chance of true IA clearance. The overall diagnostic accuracy of the GM test in this study was 72.7%.

Statistics	Value	95% CI
Sensitivity	82.4%	74.6%-88.6%
Specificity	46.8%	32.1%-61.9%
Positive likelihood ratio	1.55	1.17-2.05
Negative likelihood ratio	0.38	0.23-0.61
Disease prevalence	72.7%	65.4%-79.2%
Positive predictive value	80.5%	75.7%-84.5%
Negative predictive value	50.0%	38.1%-61.9%
Diagnostic accuracy	72.7%	65.4%-79.2%

TABLE 4: Statistical measures of galactomannan values and their 95% confidence intervals (CIs).

Discussion

Patients with weakened immune systems are particularly vulnerable to IA. The use of biomarkers is necessary to improve the detection of fungal pathogens due to current diagnostic constraints. Cases are identified based on a positive culture for aspergillosis because most patients usually do not undergo invasive histological testing. Sensitive assays are, therefore, required for a precise diagnosis of the fungal infection [27,28].

The retrospective study presents a three-year overview of the role of serum GM in clinically diagnosed IFIs at a tertiary care hospital. In our study, the overall mean age was 46.6 ± 14.2 years, which is similar to the findings of Taghizadeh-Armaki et al., who reported a mean age of 46 ± 15.4 years [29]. Forty-nine (28.5%) cases were infected with *A. flavus*, followed by 41 (23.8%) cases with *A. fumigatus* and 35 (20.3%) with *A. niger*, which contradicts the findings of Namuwenge et al., who reported *A. flavus* in 7 (26.9%) cases, *A. niger* in 7 (26.9%) cases, and *A. fumigatus* in 3 (11.5%) cases [30].

The underlying disease, age, and assay cutoff values all affect the GM test's accuracy. The GM antigen sensitivity and specificity in our study were 82.4% and 46.8%, respectively, which can be compared to the findings of Hu et al., in which the sensitivity was 84.1%, and the specificity was 75.68%, which may be a useful tool in conjunction with clinical risk factors to guide diagnosis and treatment [31]. In our study, 103 test results were true positives (TPs), and 22 were false negatives (FNs), which were much higher than those reported by Hu et al., in which there were 25 TPs and 14 FNs [31].

The negative predictive value was 92%-98%, and the positive predictive value was 25%-62% in a study by Sarrafzadeh et al., which differed from our findings, in which the positive predictive value was 75.7%-84.5%, and the negative predictive value ranged from 38.1% to 61.9% [32]. The AUC in our study was 0.69, which was close to the findings of Dichtl et al., in which the AUC was 0.76 [33].

We advise routine GM antigen testing for each patient suspected of having a fungal infection because it is a reliable, quick, inexpensive test. In patients who also meet host characteristics and clinical criteria, a positive GM antigen test result may support a diagnosis of likely fungal infection and prompt the doctor to initiate empirical antifungal therapy. In patients with chronic neutropenic fever without radiologic or clinical symptoms, negative GM antigen assay results are more useful for confirming the absence of IA and for preventing empirical anti-mold therapy.

Limitations

The limitations of this study are discussed. The study is weaker because it is retrospective. Single-center designs may limit generalizability, as they may not be broadly applicable across populations and geographic regions. There was lower statistical power since the serum GM subgroup was small ($n = 172$). Clinical judgment was used to allocate patients to serum testing, which could introduce selection bias. Because studies with positive results are more likely to be published, potential publication bias may lead to an overestimation of diagnostic accuracy. We excluded 1,3- β -D-glucan testing and PCR assays, which could have improved diagnostic performance and enabled more thorough comparisons. Variations in reagent batches, individual patient differences, and test timing can affect the sensitivity and specificity of detection methods, leading to fluctuations in findings. Interpretation is further complicated by heterogeneity in cutoffs and by preceding antifungal therapy, as reported by Marr et al. [34]. Additionally, the development of IA and its identification may be affected by factors not accounted for in the study, such as patients'

concurrent drug use and specific immunological states.

Conclusions

Serum GM antigen detection is a helpful and reliable diagnostic test for IFIs. For the diagnosis of IA in serum, the GM assay frequently offers great specificity, enabling early, focused antifungal treatment. The ability to distinguish invasive infection from colonization or non-infectious cases is greatly improved by incorporating GM testing into clinical protocols, particularly in immunocompromised patients. To improve the generalizability and reliability of the results, the limitations should be addressed in future studies by increasing the sample size and conducting investigations across multiple medical facilities in various locations.

An improved understanding of the developmental process and risk factors for IFIs would result from longitudinal patient tracking. Developing more precise and sensitive biomarker detection techniques for IFI is expected to increase diagnostic precision.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Kalinga Institute of Medical Sciences (KIMS), KIIT Deemed to be University issued approval KIIT/KIMS/IEC/2617/2026. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

- Adzic-Vukicevic T, Mladenovic M, Jovanovic S, Soldatović I, Radovanovic-Spurnic A: Invasive fungal disease in COVID-19 patients: a single-center prospective observational study. *Front Med (Lausanne)*. 2023, 10:1084666. [10.3389/fmed.2023.1084666](https://doi.org/10.3389/fmed.2023.1084666)
- Liu Q, Chen P, Xin L, Zhang J, Jiang M: A rare intestinal mucormycosis caused by *Lichtheimia ramosa* in a patient with diabetes: a case report. *Front Med (Lausanne)*. 2024, 11:1435239. [10.3389/fmed.2024.1435239](https://doi.org/10.3389/fmed.2024.1435239)
- Denning DW: Global incidence and mortality of severe fungal disease. *Lancet Infect Dis*. 2024, 24:428-38. [10.1016/S1473-3099\(23\)00692-8](https://doi.org/10.1016/S1473-3099(23)00692-8)
- Suleyman G, Alangaden GJ: Nosocomial fungal infections: epidemiology, infection control, and prevention. *Infect Dis Clin North Am*. 2021, 35:1027-53. [10.1016/j.idc.2021.08.002](https://doi.org/10.1016/j.idc.2021.08.002)
- Monzó-Gallo P, Chumbita M, Lopera C, et al.: Real-life epidemiology and current outcomes of hospitalized adults with invasive fungal infections. *Med Mycol*. 2023, 61:10.1093/mmy/myad021
- Anandani G, Bhise M, Agarwal A: Invasive fungal infections and the management in immunocompromised conditions. *J Family Med Prim Care*. 2025, 14:2643-52. [10.4103/jfmpc.jfmpc_1582_24](https://doi.org/10.4103/jfmpc.jfmpc_1582_24)
- Patterson TF, Kirkpatrick WR, White M, et al.: Invasive aspergillosis. Disease spectrum, treatment practices, and outcomes. I3 Aspergillus Study Group. *Medicine (Baltimore)*. 2000, 79:250-60. [10.1097/00005792-200007000-00006](https://doi.org/10.1097/00005792-200007000-00006)
- Teng JC, Slavin MA, Teh BW, et al.: Epidemiology of invasive fungal disease in lymphoproliferative disorders. *Haematologica*. 2015, 100:e462-6. [10.3324/haematol.2015.126698](https://doi.org/10.3324/haematol.2015.126698)

9. Svensson T, Lundström KL, Höglund M, Cherif H: Utility of bronchoalveolar lavage in diagnosing respiratory tract infections in patients with hematological malignancies: are invasive diagnostics still needed?. *Ups J Med Sci.* 2017, 122:56-60. [10.1080/03009734.2016.1237595](https://doi.org/10.1080/03009734.2016.1237595)
10. Baddley JW: Clinical risk factors for invasive aspergillosis. *Med Mycol.* 2011, 49:S7-12. [10.3109/13693786.2010.505204](https://doi.org/10.3109/13693786.2010.505204)
11. Blyth CC, Gilroy NM, Guy SD, et al.: Consensus guidelines for the treatment of invasive mould infections in haematological malignancy and haemopoietic stem cell transplantation. *Intern Med J.* 2014, 44:1333-49. [10.1111/imj.12598](https://doi.org/10.1111/imj.12598)
12. Lewis RE, Cahyame-Zuniga L, Leventakos K, et al.: Epidemiology and sites of involvement of invasive fungal infections in patients with haematological malignancies: a 20-year autopsy study. *Mycoses.* 2013, 56:638-45. [10.1111/myc.12081](https://doi.org/10.1111/myc.12081)
13. Jenks JD, Hoenigl M: Treatment of aspergillosis. *J Fungi (Basel).* 2018, 4: [10.3390/jof4030098](https://doi.org/10.3390/jof4030098)
14. Rives MM, Wingard JR: Diagnostic methods for invasive fungal diseases in patients with hematologic malignancies. *Expert Rev Hematol.* 2012, 5:661-9. [10.1586/ehm.12.53](https://doi.org/10.1586/ehm.12.53)
15. Lamoth F: Galactomannan and 1,3- β -D-glucan testing for the diagnosis of invasive aspergillosis. *J Fungi (Basel).* 2016, 2: [10.3390/jof2030022](https://doi.org/10.3390/jof2030022)
16. Freeman Weiss Z, Leon A, Koo S: The evolving landscape of fungal diagnostics, current and emerging microbiological approaches. *J Fungi (Basel).* 2021, 7: [10.3390/jof7020127](https://doi.org/10.3390/jof7020127)
17. Lass-Flörl C: How to make a fast diagnosis in invasive aspergillosis. *Med Mycol.* 2019, 57:S155-60. [10.1093/mmy/myy103](https://doi.org/10.1093/mmy/myy103)
18. Hoenigl M, Seidel D, Carvalho A, et al.: The emergence of COVID-19 associated mucormycosis: a review of cases from 18 countries. *Lancet Microbe.* 2022, 3:e543-52. [10.1016/S2666-5247\(21\)00237-8](https://doi.org/10.1016/S2666-5247(21)00237-8)
19. Moura S, Cerqueira L, Almeida A: Invasive pulmonary aspergillosis: current diagnostic methodologies and a new molecular approach. *Eur J Clin Microbiol Infect Dis.* 2018, 37:1393-403. [10.1007/s10096-018-3251-5](https://doi.org/10.1007/s10096-018-3251-5)
20. Kono Y, Tsushima K, Yamaguchi K, et al.: The utility of galactomannan antigen in the bronchial washing and serum for diagnosing pulmonary aspergillosis. *Respir Med.* 2013, 107:1094-100. [10.1016/j.rmed.2013.04.007](https://doi.org/10.1016/j.rmed.2013.04.007)
21. Sarwar M, Gardezi SA, Zaman G, Ikram A, Satti L, Khadim MT: Evaluation of galactomannan and beta-d-glucan assays for the diagnosis of invasive aspergillosis in clinically suspected cases. *J Pak Med Assoc.* 2020, 70:442-6. [10.5455/JPMA.1476](https://doi.org/10.5455/JPMA.1476)
22. Aquino VR, Goldani LZ, Pasqualotto AC: Update on the contribution of galactomannan for the diagnosis of invasive aspergillosis. *Mycopathologia.* 2007, 163:191-202. [10.1007/s11046-007-9010-2](https://doi.org/10.1007/s11046-007-9010-2)
23. Farmakiotis D, Le A, Weiss Z, Ismail N, Kubiak DW, Koo S: False positive bronchoalveolar lavage galactomannan: effect of host and cut-off value. *Mycoses.* 2019, 62:204-13. [10.1111/myc.12867](https://doi.org/10.1111/myc.12867)
24. Leeflang MM, Debets-Ossenkopp YJ, Wang J, et al.: Galactomannan detection for invasive aspergillosis in immunocompromised patients. *Cochrane Database Syst Rev.* 2015, 2015:CD007394. [10.1002/14651858.CD007394.pub2](https://doi.org/10.1002/14651858.CD007394.pub2)
25. Kumari P, Wadhwa T, Kumar A, Gaba M, Datta B: A prospective observational study on the evaluation of the galactomannan test in the diagnosis of invasive aspergillosis. *Cureus.* 2025, 17:e95230. [10.7759/cureus.95230](https://doi.org/10.7759/cureus.95230)
26. The R project for statistical computing. (2020). Accessed: April 4, 2026: <http://www.r-project.org/>.
27. Kontoyiannis DP, Marr KA, Park BJ, et al.: Prospective surveillance for invasive fungal infections in hematopoietic stem cell transplant recipients, 2001-2006: overview of the Transplant-Associated Infection Surveillance Network (TRANSNET) Database. *Clin Infect Dis.* 2010, 50:1091-100. [10.1086/651263](https://doi.org/10.1086/651263)
28. Boch T, Spiess B, Cornely OA, et al.: Diagnosis of invasive fungal infections in haematological patients by combined use of galactomannan, 1,3- β -D-glucan, Aspergillus PCR, multifungal DNA-microarray, and Aspergillus azole resistance PCRs in blood and bronchoalveolar lavage samples: results of a prospective multicentre study. *Clin Microbiol Infect.* 2016, 22:862-8. [10.1016/j.cmi.2016.06.021](https://doi.org/10.1016/j.cmi.2016.06.021)
29. Taghizadeh-Armaki M, Hedayati MT, Moqarabzadeh V, et al.: Effect of involved Aspergillus species on galactomannan in bronchoalveolar lavage of patients with invasive aspergillosis. *J Med Microbiol.* 2017, 66:898-904. [10.1099/jmm.0.000512](https://doi.org/10.1099/jmm.0.000512)
30. Namuwenge PM, Kyokushaba J, Kasone V, et al.: Prevalence and factors associated with Aspergillus galactomannan antigenemia among people with advanced HIV disease in Uganda, 2023. *Open Forum Infect Dis.* 2026, 13:ofaf620. [10.1093/ofid/ofaf620](https://doi.org/10.1093/ofid/ofaf620)
31. Hu X, Hu X, Zhu Y, Hu H, Wang Z: Sensitivity of serum galactomannan antigen in the diagnosis of invasive aspergillosis in patients with obstructive pulmonary diseases (asthma and chronic obstructive pulmonary diseases). *Postepy Dermatol Alergol.* 2025, 42:183-9. [10.5114/ada.2024.145208](https://doi.org/10.5114/ada.2024.145208)
32. Sarrafzadeh SA, Rafati AH, Ardalan M, Mansouri D, Tabarsi P, Pourpak Z: The accuracy of serum galactomannan assay in diagnosing invasive pulmonary aspergillosis. *Iran J Allergy Asthma Immunol.* 2010, 149-55.
33. Dichtl K, Forster J, Ormanns S, Horns H, Suerbaum S, Seybold U, Wagener J: Comparison of β -D-glucan and galactomannan in serum for detection of invasive aspergillosis: retrospective analysis with focus on early diagnosis. *J Fungi (Basel).* 2020, 6: [10.3390/jof6040253](https://doi.org/10.3390/jof6040253)
34. Marr KA, Schlamm HT, Herbrecht R, et al.: Combination antifungal therapy for invasive aspergillosis: a randomized trial. *Ann Intern Med.* 2015, 162:81-9. [10.7326/M13-2508](https://doi.org/10.7326/M13-2508)