ORIGINAL ARTICLE

COVID-associated pulmonary aspergillosis and azole-resistant *Aspergillus* species; A laboratory – based study from Pakistan

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ABSTRACT

Background: COVID-19-associated pulmonary aspergillosis (CAPA) has emerged as a significant complication in critically ill patients, leading to increased morbidity and mortality, particularly when diagnosis is delayed or antifungal resistance is present. A previous study from our centre reported a CAPA incidence of 21.7% with high associated mortality. This study aimed to evaluate azole resistance in *Aspergillus* species isolated from COVID-19 patients using a standardized screening approach.

Materials and Methods: This study included *Aspergillus* isolates recovered from respiratory specimens of COVID-19 patients admitted to Aga Khan University Hospital, Karachi, between July 2020 and January 2022. Azole resistance screening was performed using the CDC agar-based methodology with itraconazole, voriconazole, and posaconazole. Isolates that demonstrated growth in any azole-containing well were further tested for minimum inhibitory concentrations (MICs) using broth microdilution. Quality control strains included *Candida parapsilosis* ATCC 22019, *Candida krusei* ATCC 6258, and *A. flavus* ATCC 204304.

Results: A total of 174 *Aspergillus* isolates (*A. flavus* 85, *A. niger* 41, *A. fumigatus* 36, *A. terreus* 8, *A. nidulans* 3, *A. versicolor* 1) from 125 patients (40 CAPA; 85 colonization) were analysed. Growth was observed on voriconazole screening agar in 24 (13.7%) isolates. No growth was seen in the itraconazole or posaconazole wells. MIC testing of 18 voriconazole-screened isolates confirmed that all were azole-susceptible.

Conclusion: Although initial screening suggested possible azole resistance, none were confirmed resistant by MIC testing. These findings highlight the need for continued surveillance to monitor emerging resistance in *Aspergillus* species in Pakistan.

***Keywords**:* Azole resistance, CAPA, COVID-19

BACKGROUND

The emergence of severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), the causative agent of Coronavirus Disease 2019 (COVID-19), has been associated with profound effects on host immunity and respiratory epithelial integrity. The virus disrupts mucociliary clearance and the epithelial barrier of the respiratory tract, creating a permissive environment for secondary infections.[1] Opportunistic fungal infections, particularly COVID-19-associated pulmonary aspergillosis (CAPA), have emerged as frequent and severe complications in critically ill patients.

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This article can be cited as: Zaka S, Shahid M, Farooqi J, Raheem T, Naqvi MF, Jabeen K. COVID-associated pulmonary aspergillosis and azole-resistant *Aspergillus* species; A laboratory-based study from Pakistan. Infect Dis J Pak. 2025; 34(4): 227-232.

DOI: https://doi.org/10.61529/idjp.v34i4.432

Receiving date: **17 Jul 2025** Acceptance Date: **30 Oct 2025**
Revision date: **22 Oct 2025** Publication Date: **30 Dec 2025**

Initial reports on CAPA varied in incidence due to differences in diagnostic criteria and regional surveillance capacity. However, more recent studies using standardized definitions proposed by the European Confederation for Medical Mycology (ECMM) and the International Society for Human and Animal Mycology (ISHAM) report a CAPA prevalence of approximately 10% in critically ill patients.[2] A meta-analysis encompassing 28 studies and 3,184 intensive care unit (ICU) patients reported a pooled CAPA incidence of 10.2%.[3] Similarly, in a cohort of 335 mechanically ventilated COVID-19 patients, CAPA was diagnosed in 33% of cases.[4] Identified risk factors include underlying chronic liver disease, hematologic malignancies, chronic obstructive pulmonary disease (COPD), diabetes mellitus, cerebrovascular disease, renal replacement therapy, and the use of immunosuppressive therapies such as corticosteroids and IL-6 inhibitors.[5] Importantly, CAPA is associated with poor clinical outcomes, with all-cause mortality often exceeding 50% significantly higher than in COVID-19 patients without fungal co-infection.

The high mortality associated with CAPA may be further exacerbated by antifungal resistance, particularly to azoles, the first-line agents in the treatment of *Aspergillus* infections.[6-8] Since the beginning of the pandemic, several studies have reported azole resistance in *Aspergillus* species isolated from CAPA patients.

In response, the 2020 ECMM/ISHAM consensus guidelines recommended routine antifungal susceptibility testing, particularly in regions with known environmental azole resistance or in patients exhibiting poor clinical response to therapy.[9] Agar-based screening followed by confirmatory broth microdilution is recommended for accurate detection.

In Pakistan, one of the first countries to report CAPA during the early phase of the pandemic, an initial study documented a CAPA prevalence of 21.7% among ICU-admitted COVID-19 patients, with a case fatality rate of 44%.[10] A subsequent case-control analysis identified advanced age, chronic kidney disease, and critical illness at presentation as significant predictors of CAPA.[11] Despite these insights, both studies lacked data on antifungal resistance, particularly azole resistance in *Aspergillus* isolates.

Given the clinical importance of azole resistance in guiding therapy and improving outcomes, this study aimed to determine its frequency in *Aspergillus* species isolated from COVID-19 patients at a tertiary care centre in Pakistan.

MATERIAL AND METHODS

This cross-sectional study was conducted at the Aga Khan University Hospital clinical laboratory, Karachi, Pakistan, between July 2020 and January 2022. The Aga Khan University Hospital served as one of the major tertiary care centres managing COVID-19 patients in the country during the pandemic. Respiratory specimens were submitted to the microbiology laboratory based on the clinical discretion of the primary physician. All respiratory samples were routinely processed for bacterial culture using sheep blood colistin nalidixic agar, chocolate agar, and MacConkey agar. For fungal isolation, a high volume (approximately 1 mL) of each specimen was inoculated onto Sabouraud's dextrose agar (SDA). *Aspergillus* species were identified using conventional macroscopic colony morphology and microscopic examination with lactophenol cotton blue stain. These phenotypic methods are routinely used in clinical microbiology laboratories across Pakistan; however, they offer limited discriminatory power, particularly for distinguishing cryptic species. Molecular identification techniques, which provide greater accuracy, were not utilized in this study due to their unavailability at the time. All *Aspergillus* isolates were preserved at –80 °C in glycerol stocks and later sub-cultured onto SDA for antifungal susceptibility testing.

Preliminary azole resistance screening was performed using the agar-based triazole screening method. Conidial suspensions (adjusted to 0.5 McFarland) of each *Aspergillus* isolate were inoculated into Roswell Park Memorial Institute (RPMI) 1640 agar supplemented with individual antifungal agents: itraconazole (4 mg/l), voriconazole (1 mg/l), posaconazole (0.5 mg/l), powders from the Sigma-Aldrich Company (St. Louis, MO, USA) and a control plate without azoles. The plates were incubated at 35 °C and examined at 24, 48, and 72 hours. Isolates showing growth on any azole-containing plate were considered potentially resistant (azole-non-susceptible), while those with growth restricted to the control plate were considered susceptible[12]. Azole screening results were compared in CAPA and colonizer groups using a chi-square test on R software [version 4.4.2 (2024-10-31 ucrt) R Foundation for Statistical Computing, Vienna, Austria]. A p-value of < 0.05 was considered statistically significant.

Isolates demonstrating growth on any azole screening plate were further tested by the broth microdilution method, as per the CLSI M38-A2 reference standard. Serial twofold dilutions of antifungal agents were prepared in 96-well U-bottom microtiter plates (Costar®, Corning Incorporated) and stored at –80 °C until use[13]. Conidial suspensions (0.5 McFarland) were diluted 1:50 in RPMI 1640 broth and inoculated into each well. Plates were incubated at 35 °C, and minimum inhibitory concentrations (MICs) were determined visually after 46–50 hours. Quality control was ensured using *Candida parapsilosis* ATCC 22019, *Candida krusei* ATCC 6258, and *Aspergillus flavus* ATCC 204304. MIC interpretations were based on CLSI M59, 3rd edition[14].

(Reference number of the Institutional Review Committee (IRC): 2019-0438-2659)

RESULTS

A total of 174 non-duplicate *Aspergillus* isolates were recovered from 122 patients with COVID-19. The majority of isolates originated from lower respiratory tract specimens, including tracheal aspirates, sputum, and bronchoalveolar lavage. Additionally, one isolate each was obtained from pus, a sinonasal swab, and a combined bone and tissue sample. Among the 122 patients, 75 (61.47%) were male and 47 (38.5%) were female. Forty patients had probable CAPA, and 82 patients had *Aspergillus* colonization. All patients were adults except one, a 9-month-old infant from whom *Aspergillus* was isolated from a tracheal aspirate. The overall mean patient age was 58.7 years (median: 60 years; range: 9 months–95 years). The age was not normally distributed with a Median (IQR): 61 (47-71).

Of the 174 *Aspergillus* isolates, *A. flavus* was the most frequently identified species (85/174; 48.8%), followed by *A. niger* (41/174; 23.5%) and *A. fumigatus* (36/174; 20.7%). Less frequently isolated species included *A. terreus* (8/174; 4.6%), *A. nidulans* (3/174; 1.7%), and *A. versicolor* (1/174; 0.6%). This distribution of *Aspergillus* species in CAPA patients was as follows: *A. flavus* (21/40; 52.5%), *A. fumigatus* (9/40; 22.5%), *A. niger* (7/40; 17.5%), *A. terreus* (2/40; 5%) and *A. nidulans* (1/40; 2.5%).

Initial azole resistance screening using RPMI agar supplemented with triazoles revealed growth in the voriconazole-containing well in 24 (13.8%) of 174 isolates. Screen positive isolates were *A. flavus* (17/85; 20.0%), *A. fumigatus* (4/36; 11.1%), and *A. niger* (3/41; 7.3%). None of the isolates exhibited growth on either itraconazole or posaconazole screening plates. None of the *A. terreus*, *A. nidulans*, or *A. versicolor* isolates screened positive for resistance to any azole (Table-I). There was no statistically significant difference in azole screen positivity rate between CAPA isolates (5/40; 12.5%) and colonizer isolates (19/82; 23.2%) (p-value:0.25).

Minimum inhibitory concentrations (MICs) were determined for 18/24 isolates that grew on voriconazole screening agar. Six isolates could not be retested as they failed to revive upon subsequent subculture, likely due to loss of viability during storage or suboptimal preservation conditions, thereby precluding their inclusion in susceptibility testing. All isolates were found to be susceptible to all azoles by broth microdilution. The MIC ranges for *A. flavus* were as follows: posaconazole 0.03–0.5 µg/ml, voriconazole 0.125–2 µg/ml, and itraconazole 0.06–1 µg/ml. For *A. fumigatus*, MIC ranges were: posaconazole 0.5–1 µg/ml, itraconazole 1–2 µg/ml, and voriconazole 0.25–1 µg/ml. Single *A. niger* isolate exhibited MIC of 0.5 µg/ml for posaconazole, 0.5 µg/ml for voriconazole, and 0.5 µg/ml for itraconazole (Table-II).

Table-I: *Aspergillus* species distribution and growth on azole resistance screen agar

Specie	Number of isolates	Growth only in posaconazole well	Growth only in itraconazole well	Growth only in voriconazole well
Aspergillus flavus	85	0 (0%)	0 (0%)	17 (20%)
Aspergillus fumigatus	36	0 (0%)	0 (0%)	4 (11.1%)
Aspergillus niger	41	0 (0%)	0 (0%)	3 (7.3%)
Aspergillus terreus	8	0 (0%)	0 (0%)	0 (0%)
Aspergillus nidulans	3	0 (0%)	0 (0%)	0 (0%)
Aspergillus versicolor	1	0 (0%)	0 (0%)	0 (0%)
Total	**174**	**0 (0%)**	**0 (0%)**	**24 (13.7%)**

Table-II: Minimum inhibitory concentration of *Aspergillus* species that grew on voriconazole well using azole resistance screening agar (n=18)

Species	N	Posaconazole	Itraconazole	Voriconazole
Aspergillus flavus	13	0.03 - 0.5 µg/ml	0.06 - 1 µg/ml	0.125 - 2 µg/ml
Aspergillus fumigatus	4	0.5 -1 µg/ml	1 - 2 µg/ml	0.25 - 1 µg/ml
Aspergillus niger	1	0.5 µg/ml	0.5 µg/ml	0.5 µg/ml

DISCUSSION

COVID-19-associated pulmonary aspergillosis (CAPA) has emerged as a critical complication among patients with severe SARS-CoV-2 infection, especially those requiring intensive care and mechanical ventilation.[4] The immunological dysregulation, epithelial barrier damage, and corticosteroid use in COVID-19 patients create an ideal setting for fungal superinfections. One of the most concerning aspects of CAPA is the increasing emergence of azole non-wild-type *Aspergillus* species, which complicates antifungal therapy and is associated with poor clinical outcomes.[15] Globally, an estimated 3

million cases of chronic pulmonary aspergillosis (CPA) and invasive aspergillosis (IA) occur annually.[16] Azole resistance in invasive aspergillosis was recognized even before the COVID-19 pandemic and was associated with high morbidity and mortality.[17,18] The pandemic has likely accelerated this trend due to increased azole exposure and immunosuppressive treatment regimens, contributing to the emergence of azole non-wild type *Aspergillus fumigatus* strains in CAPA patients.[6,19]

In our study, out of 125 patients with COVID-19 and a growth of *Aspergillus* species, 40 cases of probable CAPA were identified, representing a substantial increase compared to an earlier study from our institution that reported only five cases.[10] This rise is likely attributable to the significant surge in COVID-19 hospitalizations during the study period, along with the widespread use of corticosteroids and other immunosuppressive therapies, which may have further compromised host defences.[20] Critically ill COVID-19 patients are known to exhibit profound immune dysregulation, including lymphopenia and cytokine imbalance, which predispose them to secondary fungal infections such as CAPA.[21] A study from the Netherlands found a 19.4% CAPA rate among 31 ICU patients[22], while reports from France and Germany observed rates of 33% and 26%, respectively, in smaller cohorts.[23,24]

Our study did not find azole resistance in CAPA patients. However, a study from Europe estimated 14.3% phenotypic azole resistance in a cohort of 21 patients.[15] In another study from Iran, 47% of the strains revealed itraconazole resistance with MICs of ≥ 16 µg/ml.[25] Additionally, a meta-analysis by Habibzadeh *et al*. reported an overall azole resistance prevalence of 15% among CAPA patients.[19] In line with these findings, Meijer *et al*. conducted a prospective dual-centre study in the Netherlands, identifying azole resistance in 15.4% of CAPA cases, including isolates harboring the TR34/L98H mutation, suggesting environmentally acquired resistance despite established antifungal stewardship practices.[26]

Our findings underscore the need for routine antifungal susceptibility testing, as well as, where feasible, molecular screening to guide appropriate therapy. Regular monitoring can aid in early detection of resistance trends and help prevent therapeutic failures. In addition, environmental surveillance and robust antifungal stewardship practices are crucial to limiting the emergence and spread of resistance, particularly in regions with extensive agricultural azole use. Despite the significance of these findings, the study has certain limitations.

This study was conducted at a single tertiary care centre, and therefore, the findings may not be fully generalizable to other healthcare settings or geographic regions within Pakistan. Respiratory samples were submitted to the microbiology laboratory based on the clinical judgement of the treating physicians, which could have introduced selection bias toward patients with more severe or atypical presentations. While this discretionary approach reflects real-world clinical practice, it may limit the external validity of the findings. The number of *A. fumigatus* isolates subjected to susceptibility testing was relatively small, potentially affecting the precision of the estimated prevalence of azole non-wild-type strains. Additionally, molecular characterization such as detection of *cyp51A* gene mutations or promoter tandem repeats were not performed, restricting insight into the underlying genetic mechanisms of resistance. The lack of clinical outcome data for patients with screen-positive isolates further limited our ability to correlate microbiological results with clinical significance or prognosis. These limitations underscore the need for future multicenter studies employing standardized sampling methods and integrating molecular and clinical data to provide a more comprehensive understanding of azole resistance in CAPA within the regional context.

CONCLUSION

In conclusion, although several *Aspergillus* isolates from COVID-19 patients initially screened positive for azole resistance, confirmatory testing revealed that none were truly resistant. These findings highlight the critical importance of periodic surveillance and monitoring to detect the potential emergence of azole resistance in *Aspergillus* species in Pakistan. However, as this was a single-centre study, the generalizability of the results may be limited, and we were unable to assess the clinical outcomes of patients with screen-positive isolates. Broader, multicenter investigations are therefore warranted to better define the epidemiology and underlying mechanisms of azole resistance in the region.

CONFLICT OF INTEREST

None

GRANT SUPPORT & FINANCIAL DISCLOSURE

This project was funded by the University Research Council, Aga Khan University Hospital, Karachi, Pakistan

AUTHOR CONTRIBUTION

Sadaf Zaka: Conceptualization, data curation, data analysis, manuscript writing, final approval, accountable for all aspects of publication

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Tooba Raheem: Data curation, manuscript writing, final approval, accountable for all aspects of publication

Muhammad Faheem Naqvi: Data curation, final approval, accountable for all aspects of publication

Kauser Jabeen: Conceptualization, data curation, data analysis, critical revisions, manuscript writing, final approval, accountable for all aspects of publication.

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