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CASE REPORT

Invasive Pulmonary Aspergillosis Secondary to the Climate Changes

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ABSTRACT

Aspergillus fumigatus is a globally distributed mold and major opportunistic human pathogen, known for its ability to grow at high temperatures. This study examined 89 strains from 12 countries to assess variation in thermal growth and potential links to geographic origin. Strains were grown at four temperatures and genotyped at nine microsatellite loci. Results showed significant variation in growth among strains, even within the same region, but no strong correlation between thermal growth profiles and genetic makeup or geographic origin. These findings suggest that *A. fumigatus* populations have broad thermal adaptability, likely enabling rapid adjustment to changing temperatures. This adaptability has important implications for the pathogen's evolution and spread, especially under the pressures of global climate change.

KEYWORDS

• Invasive Pulmonary • Pathogen • Genotyped

INTRODUCTION

Aspergillus is a type of fungus commonly found in soil, food, plant matter, and indoor environments. Its spores can easily become airborne and inhaled. Once in the respiratory mucosa, the spores may germinate into hyphae, which can invade the tissue, leading to invasive pulmonary aspergillosis (IPA).[1] Aspergillus species thrive in the environment

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by obtaining nutrients from decaying organic matter and reproduce asexually through conidia. Over 24 species of Aspergillus can cause human disease, with *A. fumigatus* being the most common pathogen, followed by *A. terreus* and *A. flavus*.[2]

In most individuals, the body's innate immune response and inflammatory cells control fungal growth and prevent the development of disease. However, depending on the individual's immune system and specific immune deficiencies, Aspergillus may cause various pulmonary conditions.[3]

IPA typically affects individuals with severe immune suppression. The most common at-risk groups include those with prolonged neutropenia, which can result from chemotherapy or immunosuppressive treatments, as well as patients who have undergone hematopoietic stem cell transplants (SCT) or solid organ transplants.[4]

Risk factors for invasive pulmonary aspergillosis (IPA) include hematopoietic stem cell or solid organ transplants (especially lung transplants), hematologic malignancies, prolonged neutropenia, critical illness requiring ICU care, prolonged steroid use, hemodialysis, liver disease, chronic obstructive pulmonary disease (COPD), and chronic granulomatous diseases. These conditions compromise the immune system, increasing susceptibility to infection.[3]

There are three main types of bronchopulmonary aspergillosis: invasive aspergillosis, chronic aspergillosis, and allergic aspergillosis.[5] Although transmission occurs through the aspiration of conidia, most individuals who inhale these spores do not develop aspergillosis due to the body's immune response.

Importance of climate change and its effect on human health

Climate change is a major global challenge that poses significant risks to public health.[6] In October 2023, the World Health Organization (WHO) labelled climate change as the most significant threat to human health.[7]

Climate change has been recognized as a significant factor influencing the spread of communicable diseases and the emergence of new pathogens.[8] Global reports have increasingly highlighted the rising incidence and spread of vector-borne diseases like malaria, dengue, Lyme disease, West Nile virus, and the recent spread of the H5N1 avian influenza virus across species.[8] Extreme temperatures, heatwaves, rising sea levels, changes in rainfall leading to droughts and floods, and more frequent and intense hurricanes all contribute to injury, illness, and higher mortality rates.[9] Indirect health effects of climate change, such as air pollution, negatively impact respiratory and cardiovascular health, further increasing mortality rates.[10] Data from the US indicates that elevated air temperatures and pollution significantly raise the incidence of preterm births, low birth weight, and stillbirths.[11]

The primary cause of global climate change is the rising concentration of greenhouse gases in the atmosphere, largely due to the burning of fossil fuels by both developed and developing nations.[12] The consequences of climate change include more frequent and severe heatwaves, hurricane seasons, and heightened risks from droughts, wildfires, and floods. These effects have directly and indirectly impacted human health, weakened public health systems, including water and sanitation services, and increased the global burden of both infectious and noncommunicable diseases.[13]

Changes in temperature and rainfall patterns can alter the distribution, survival, and behavior of insects and other species that transmit diseases to humans, increasing the spread of vector-borne diseases and altering their geographic range.[14] Higher rainfall, storms, and rising sea temperatures are linked to a rise in water-borne diseases.[7] Heat can also jeopardize food safety, leading to contamination by infectious organisms and toxins, or through an increased need for chemicals to manage crop infestations and diseases in livestock. Impaired mental health and well-being have recently been recognized as consequences of climate change, linked to the growing risks to physical health and safety.[15]

According to the newly released 2025 Climate Risk Index (CRI), India ranks among the countries most severely affected by extreme weather over the past three decades. From 1993 to 2022, the nation experienced over 400 extreme weather events such as floods, heatwaves, and cyclones which resulted in approximately 80,000 deaths and nearly $180 billion in economic damages.[16]

In 2023, individuals in India experienced more than 2,400 hours or roughly 100 days of moderate to extreme heat stress, representing a substantial rise from past decades. The report highlighted that in India, infants and the elderly have been the most vulnerable to recent climate-related effects. In addition to the physical strain caused by extreme heat, the report also pointed to significant economic losses.[17]

Effect of climate change on fungus

Climate change is impacting vegetation patterns and plant physiology by altering temperature, humidity, and atmospheric carbon dioxide (CO2) levels.[18] These changes affect plant biology, including photosynthesis, growth, and biomass production, and can influence the spread of invasive plants, pollination, fruiting patterns, and the allergenicity of pollen, fungal spores, and other biological aerosol particles.[19] It also alters microbial surface communities, which could lead to increased release of allergens or adjuvants from microbial sources.[20] Moreover, the frequency of dust storms is expected to increase, carrying pathogenic, allergenic, and adjuvant components that can worsen respiratory conditions like asthma. Dust particles, which may include pollen and fungal spores, can aggravate respiratory disorders, and "thunderstorm asthma" may occur due to inhaled allergenic particles from pollen and fungal spores.[21]

Climate change-related changes in outdoor humidity and indoor dampness may further exacerbate respiratory symptoms and asthma. Pollen grains, although part of the coarse fraction of air particulate matter, and fungal spores are also found in fine particulate matter (PM2.5), which can penetrate deep into the lungs.[20] Pollen and fungal spores release allergenic proteins after damage or under humid conditions, and osmotic shock during rain can trigger outbreaks of thunderstorm asthma. Increased concentrations of pollen, fungal spores, and other biological aerosol particles have been observed following heavy rainfall. Prominent airborne fungi like Cladosporium herbarum, Alternaria alternata, and Aspergillus fumigatus release more allergens after germination under humid conditions, with some allergens being expressed only post-germination.[22] Air pollutants, such as ozone, nitrogen oxides, and acids, can interact with these particles, damaging their envelopes and facilitating the release of allergenic substances.[23]

Pollen and fungal spores also release nonallergenic compounds, such as lipid mediators (PALMs), which have pro-inflammatory and immunomodulatory effects that can enhance allergic reactions. These substances can be influenced by climate conditions and air pollution, and studies have shown higher levels of PALMs in pollen from areas with heavy traffic.[24] PALMs like leukotrienes attract immune cells, such as neutrophils and eosinophils, which can intensify allergic reactions. Additionally, some PALMs like phytoprostanes can inhibit immune responses, fostering an allergenic environment. Nonallergenic substances in pollen may aggravate IgE-mediated allergies, contributing to the exacerbation of allergic responses.[24]

Fine aerosol particles can accumulate on the surface of pollen, fungal spores, and other biological aerosol particles, including pollutants like dust, soot, and diesel exhaust particles, which may carry allergens into deeper airways.[23] This interaction between allergens and pollutants can promote allergic sensitization and responses, with particles serving as a delivery mechanism for allergens and adjuvants.

Effect of climate change on Aspergillus

Aspergillosis, including allergic, chronic, and invasive forms, affects millions worldwide, with over 11 million cases of allergic forms, 3 million chronic cases, and 300,000 invasive aspergillosis cases annually.[25] Climate change and air pollution contribute significantly to the rise in allergic diseases by promoting fungal growth and dispersal. Warmer temperatures increase fungal allergen bioavailability, and storms may further disperse fungi. Pollutants can enhance the immunogenic potency of Aspergillus fumigatus allergens and damage lung barriers, allowing spores to germinate and release allergenic proteins.[26]

Chronic pulmonary aspergillosis is common in patients with chronic obstructive pulmonary disease (COPD) and tuberculosis (TB).[27] While rising temperatures' effects on TB incidence are unclear, increasing air pollution and CO_2 levels are projected to increase deaths from COPD and TB, making people more susceptible to aspergillosis.[28]

Azole resistance, a major concern in aspergillosis treatment, is rising. Climate change may accelerate resistance by promoting mutations in A. fumigatus that enhance survival. Resistance typically results from mutations in ergosterol biosynthesis genes, and climate-induced changes in membrane composition may increase thermotolerance. However, the full impact of reduced ergosterol content on the fungus's pathogenicity is unclear.[29]

From an evolutionary perspective, drug resistance is shaped by a complex interplay of factors such as the level of exposure to antifungals, the size and diversity of microbial populations, the existence of pre-resistant strains, and the fitness costs associated with resistance.

Resistance in Aspergillus fumigatus may increase due to climate change, which can indirectly create selective pressures favoring drug-resistant strains. Azole resistance often results from mutations affecting ergosterol biosynthesis, such as in lanosterol demethylase or hmg1, leading to altered membrane composition. While links between thermotolerance and reduced ergosterol are noted in other fungi, this connection remains unproven in A. fumigatus.

Climate driven changes in agriculture like intensified farming and increased fungicide use may further promote resistance. These resistant strains can spread through air, trade, human activity, and migratory birds. Overall, resistance evolves through drug exposure, population dynamics, and fitness costs, all of which may be influenced by a changing climate.[29]

Climate change will also affect agriculture, increasing fungicide use, including azoles, which could lead to more resistant fungal strains. The spread of resistant strains may be influenced by environmental factors such as air currents, human activity, and migratory birds.[30]

Aspergillus fumigatus is the primary cause of aspergillosis and is resilient to high temperatures.[31] While models predict it will increase in warmer regions, Aspergillus flavus remains dominant in areas like Asia, the Middle East, and Africa, despite being less heat-tolerant. This species is resistant to amphotericin B, limiting treatment options. The full effects of climate change on A. fumigatus's pathogenic potential and its adaptation to rising temperatures remain uncertain, highlighting the need for further research.[32]

In a retrospective study the relation between aspergillus lung infection and the seasonal change was detemined. They concluded a noticeable rise in Aspergillus lung infections, particularly among individuals with underlying lung conditions and multiple comorbidities during the monsoon season.[33]

Research indicates that the distribution of airborne fungal spores, such as Aspergillus species, varies across different regions in India and is influenced by climatic conditions. For instance, in Delhi, Aspergillus spore counts peak between September and October. Such seasonal variations correlate with increased respiratory allergy cases, suggesting a link between climate patterns and fungal exposure. at their institution, 52.5% of adult asthma patients showed sensitization to Aspergillus.[34]

In 2024, severe floods in Guwahati led to multiple fatalities and significant health challenges among survivors. Many required ventilator support due to pulmonary complications. Post-flood conditions, characterized by damp environments, facilitated mold growth inside buildings. Exposure to high concentrations of fungal molds, including Aspergillus species, was associated with acute respiratory infections, asthma exacerbations, and allergic reactions.[35]

CASE REPORT

A man 61 years old, went to other hospital with the complaint of fever and breathlessness. A month ago he was treated with antibiotics, bronchodilators and inhalational steroids for acute bronchitis. After being discharged then he was symptom free and alright. Patient visited our emergency department complaining of episodic fever since a month. At present patient had cough with mucoid expectoration and no blood in sputum. He had no history of rashes, headache, burning micturition and sore throat. The patient had a medical history of diabetes since 5 years for which he is on insulin therapy 10IU subcutaneously twice a day. He had the habit of smoking since 20 years 4 to 5 cigarettes a day. He is a known case of hypertension.

On examination he was febrile, and normal

vitals, saturation and blood sugar levels. There were no other abnormal signs. Respiratory system examination demonstrated bronchial breath sounds in right supraclavicular areas whereas other areas were normal. On per abdomen examination the abdomen was non tender and there were no other abnormalities noted. On cardiovascular examination heart sounds were normal.

Blood examination revealed higher red blood cells count, Prothrombin time, International normalised ratio, absolute neutrophil count, serum creatine kinase, creatinine and lower absolute monocyte count, serum chloride, phosphorus. The patient was admitted in the Department of Pulmonology and Critical Care in RICU and supportive measures were performed with antimicrobial therapy. But there was no change in the condition of the patient. Thus HRCT Chest was done on a 128 slice multidetector CT scanner (Figure 1). Interval appearance of cluster of centrilobular nodules were seen in superior segment of right lower lobe. Two large bullae were seen in right upper lobe adjacent to each other separated by a thin septa measing about 11x8.9cm and 7.4x8.1cm. Fibrocalcific and fibronodular lesions were seen in the posterior segment of right apex with the fibrous nodule measuring approximately 31x32mm. Multiple small calcific nodules were noted around this lesion with thin parenchymal strands. Multiple mediastinal lymph nodes were unchanged. Emphysematous changes were seen in the right upper lobe.

Bronchoscopy was performed and BAL samples were collected. Samples showed few alveolar macrophages along with bronchial epithelial cells and squamous epithelial cells. There are numerous inflammatory cells predominantly polymorphs. Occasional atypical cells are seen having orangeophilic cytoplasm and hyperchromatic nuclei with irregular nuclei membrane. No granuloma was seen. TLC – 21,400 cells/cu.mm Differential count on BAL fluid: Polymorphs – 95% and lymphocytes – 05%.

Microscopic examination revealed few budding yeast cells. Galactomannan BAL readings were 5.63. The treatment was started with voriconazole injection 400mg intravenous stat dose followed by 200 mg intravenous twice a day for 2 weeks and was then shifted to oral dose of 200 mg twice a day for 7 more days. Other supportive measures were given continuously with the antimicrobial coverage. Repeat investigations after 6 weeks showed minimal changes thus requiring the continuation of treatment for 2 more months. Eventually the condition of the patient improved and patient was alright without any adverse events.

On discharge



Figure 1: Chest X-ray on day of admission



Figure 2: Chest X-ray on day of discharge

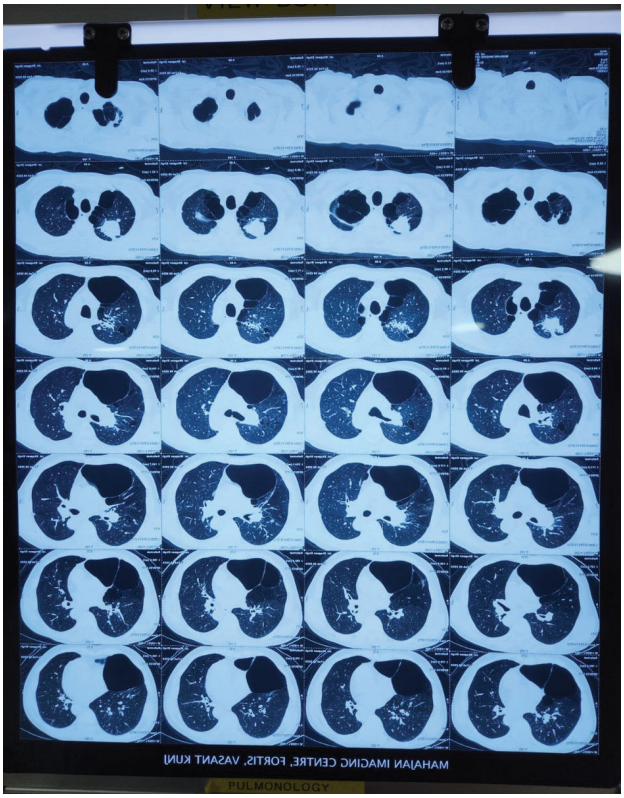


Figure 3: CT Chest on admission day

DISCUSSION

The spores of Aspergillus species are found everywhere in the environment, and the likelihood of infection spread is influenced by factors such as precipitation, humidity, temperature, and wind conditions. The patient's immune status plays a significant role in susceptibility to infections caused by Aspergillus species. These fungi can lead to diseases such as allergic bronchopulmonary Aspergillosis (ABPA), aspergilloma, chronic necrotizing Aspergillus pneumonia, and invasive infections.

The galactomannan test is a diagnostic assay used to detect Aspergillus antigens, primarily in serum or bronchoalveolar lavage (BAL) fluid. It is commonly employed in diagnosing invasive aspergillosis (IA), especially in immunocompromised patients.

Reference Range:

- Negative: Index < 0.5
- Indeterminate/Borderline: 0.5–0.99
- Positive: Index ≥ 1.0 (serum) or ≥ 1.0–1.5 (BAL, depending on guideline/lab)

The galactomannan (GM) assay demonstrates a sensitivity of approximately 71% and specificity of around 89% when used on serum samples for the diagnosis of invasive aspergillosis.[36] In contrast, testing galactomannan in bronchoalveolar lavage (BAL) fluid offers higher sensitivity, ranging from 85% to 100%, and a specificity between 85% and 95%, making it a more reliable diagnostic tool, especially in pulmonary cases.[37,38]

In this case patient was immunosuppressed due to hypertension, diabetes and smoking. Considering the fungal infections in immunocompromised patients with the changing climatic conditions proper diagnosis and treatment is essential. Triazoles like voriconazole and isavuconazole, along with lipid formulations of Amphotericin B, are the primary treatments for Aspergillus infections.

CONCLUSION

Climate change significantly affects the way we live arm, and interact with our environment, ultimately altering the epidemiology of pathogens. Fungi, in particular, are highly adaptable to changing conditions, and this adaptability will likely reshape the landscape of fungal diseases. Thus proper diagnosis and early treatment is essential and highly recommended.

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