Is invasive aspergillosis community acquired or nosocomial: an ongoing debate

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Invasive aspergillosis presents a formidable problem for both diagnosis and therapy. Therefore, prevention is a very important strategy in controlling this disease. Currently, the environmental mycology of most cases of invasive aspergillosis is poorly defined. However, the development of molecular biology techniques more directly applicable to *Aspergillus* spp. may help resolve some of these difficulties.

Preventing invasive aspergillosis demands a clear understanding of the environmental sources of Aspergillus spp. and how this mould is transmitted to patients before, during and after hospitalization for transplantation. Insight into the sources of exposure, mechanisms of transmission, and host susceptibility to infection are vital to appropriately direct preventive strategies to those settings where the risk of infection is the highest and consequently the impact of prevention the greatest. Increasingly, there is mounting evidence that some cases of invasive aspergillosis may be community acquired. Most human subjects are colonised with a low number of *Aspergillus* conidia. This has been confirmed by Aspergillus PCR on respiratory specimens from patients before entering the hospital and culture of tissue biopsies from patients undergoing thoracic surgery for a variety of reasons. Exposure to *Aspergillus* in hospital environments, especially during heavy construction or demolition, has been well documented. There appears to be a strong correlation between outbreaks of invasive aspergillosis and hospital building work. However, prevention of aspergillosis is relatively difficult. Simple precautions – such as eliminating potted plants from patients’ rooms and using barriers during hospital construction – are recommended. The use of high-efficacy particulate (HEPA) filters appears to be the only currently effective means of decreasing the incidence of aspergillosis infection.

The principles of environmental control of nosocomial aspergillosis are complex given that even HEPA units are not completely effective in preventing disease. Fungal exposure would be more precisely studied using a personal air sampler for the patient, but there is no fungal sampler currently available which can be used in this way, and there are also severe technical limitations on the duration of the sampling time of available fungal samplers. Alternatively, a systematic programme of longitudinal patient and environmental surveillance may predict cases of invasive aspergillosis. Indeed, there appears to be a correlation between the recovery of *Aspergillus* species from the nose and mouth of patients in an open haematology ward and an elevated number of conidia in the air. The relationship between aspergillosis in predisposed patients and building work is also complex. Hospitals are buildings of continuous change and adaptation, so construction is an inevitable prospect which may extend throughout the year. Whether or not this activity is complicated by an outbreak of infection in the susceptible patients nearby, or is a risk related directly to the amount of disruption or some other factor, is unknown. In one study, an increase in the number of patients with invasive aspergillosis could not be explained by an increase in the number of *Aspergillus* conidia in the outside air (Leenders et al. 1998). Genotyping of the isolates from the unit where the survey was
carried out showed that clonally related isolates were persistently present for more than 1 year. Clinical isolates of *A. fumigatus* obtained during the outbreak period were different from the clones normally persistent in that environment.

*Aspergillus* spp. have a major reservoir in organic debris, bird droppings, dust and building material. Further steps consist of elimination of obvious sources of aspergilli, such as removing plants from the surrounding environment of a patient. Susceptible patients should not be treated in areas where there is construction or demolition activity, and if such activities are under way, measures should be instigated to seal these sites to prevent air exchange with the patients’ environment. Certain foodstuffs, such as cereals, nuts and spices, e.g. ground black pepper, have been found to be contaminated with aspergilli and should not be offered to patients at risk of developing invasive pulmonary aspergillosis. Although outbreaks of invasive aspergillosis have been associated with construction within or around a hospital, the precise source of the fungus is very difficult to trace. There have been few studies that have prospectively examined the aeromycology in and around a hospital during major building alterations and then compared these findings with samples from patients and the incidence of invasive aspergillosis. New data suggest that patients may be exposed to *Aspergillus* conidia in water supplies in hospitals and as a result of showering in patient bedroom facilities. Quite often the investigation of the hospital environment starts at the moment an increased number of patients is diagnosed with an invasive mould infection, while no adequate background information exists about the level of environmental fungal contamination before the outbreak, or whether the patients had been exposed in their home or work environment. Increasingly, cases of aspergillosis are being reported many months after transplantation and discharge from hospital. This scenario raises the question of community acquired disease following exposure to *Aspergillus* in the home, the workplace or the outdoor environment.

Returning to work after transplantation is a much-discussed topic today, especially as a measure to avoid permanent work disability. Many transplant patients regain their ability to work 2-6 months after transplantation. However, returning to work should not endanger their health. This means that occupational risks such as occupational exposure to *Aspergillus* spores must be evaluated. On one hand the risk is linked to the exposure to microorganisms that the patient is likely to be subjected to, and on the other hand to the factors that modify his state of susceptibility or resistance to these infectious agents. The necessity of immunosuppressive therapy or the treatment for chronic graft versus host disease after transplantation elevates the aspergillosis risk, especially 1-6 months after transplantation. There are many professions in which exposure to Aspergillus spores can occur. The risk of acquiring aspergillosis at work exists, but is not easily quantifiable. Nevertheless, the risk should be minimized during the period of vulnerability by preventive measures such as restriction of certain activities, changing work methods and reorganizing the work day to adapt to the risk, and wearing personal protective equipment, as well as attention to information about aspergillosis risk and about the likelihood of exposure in the patient's professional and leisure activities.

From an epidemiological point of view, molecular investigation of moulds either isolated from patients or the environment will increase our understanding of the acquisition and route of infection. Various molecular techniques are available to genotype moulds. Discriminative typing methods are now available
Key references:


