Invasive aspergillosis in a patient with idiopathic thrombocytopenic purpura without underlying lung disease

Dr L B Palmer and others (January 1991;46:15-20) describe six patients with invasive aspergillosis whose only impairment of defence comprised underlying lung disease and corticosteroid treatment. They pointed out two features—firstly, that invasive aspergillosis can also be observed in non-immunosuppressed patients and, secondly, that chronic lung disease is a facilitating factor for its development. We report a patient with idiopathic thrombocytopenic purpura—a condition in which the immunology of the next decade is not impaired—receiving corticosteroids, who developed disseminated aspergillosis without any previous lung disease.

A 63 year old man diagnosed two months earlier as having idiopathic thrombocytopenic purpura presented with fever and progressive dyspnoea. He was having high dose corticosteroids (60—100 mg prednisone daily) and had been diabetic for 10 years.

On admission a chest radiograph showed bilateral pulmonary infiltrates. The white blood cell count was 12.3 × 10^9/l (90% neutrophils), haemoglobin 14 g/dl, and platelet count 65 × 10^9/l. He required intubation and mechanical ventilation. Broncho-alveolar lavage showed *Pneumocystis carinii*. Co-trimoxazole and corticosteroid treatment was started on the fifth day. The patient was pronounced as the human immunodeficiency virus. He improved during the next 10 days but then deteriorated and empirical antibiotic treatment with erythromycin, cefazidime, amikacin, and amphotericin B was started. A chest radiograph showed a cavity in the right middle lobe, a right pneumothorax, and pneumomediastinum. Culture of bronchial secretions from a second lavage was positive for *Aspergillus fumigatus* but no *P carinii* was identified this time. Despite full dose treatment with amphotericin B, the patient deteriorated and died a week later after invasive aspergillosis was diagnosed and at necropsy *A fumigatus* was recovered from multiple abscesses in lungs, kidneys, thyroid gland, and heart. The fungus was also found in mitral valve vegetations and in an embolus in the right pulmonary artery.

There are several interesting points in this patient. Firstly, serious opportunistic infections developed without a major cause of immunosuppression (that is, neoplasia or HIV infection), much as in patients 1 and 6 in the series of Palmer and colleagues. Immune disorders caused by diabetes might have played a part along with corticosteroid treatment in the development of this fulminant illness.

In recent years the range of underlying diseases associated with aspergillosis has expanded substantially and diabetic patients have developed invasive infections. Secondly, the infection was extensive as invasive pulmonary aspergillosis was rapidly followed by fulminating dissemination to the kidneys, heart, thyroid gland, and probably brain. The disseminating pattern differs from that reported by Dr Palmer and colleagues, in which aspergillosis was confined to the lungs. Finally, the presence of *A fumigatus* endocarditis is an exceptional finding in thrombocytopenic patients, probably related to platelet malfunction and the low platelet count. Taken together these confirm that the spread of invasive aspergillosis is broader than was originally considered. This infection should be suspected even in patients with minimally compromised immunity.

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Treatment of pulmonary aspergillosis with itraconazole

We read with interest the paper of Dr J H Campbell and coworkers (November 1991; 46:839—41). We have had a similar experience with a patient presenting with cough and haemoptysis secondary to aspergillosis (mycetoma), which was successfully treated with oral itraconazole (100 mg twice daily) given over 10 months. During the course of treatment the size of the mycetoma gradually and progressively decreased and at the completion of treatment could not be identified on a computed tomogram of the chest. Twelve months after cessation of treatment the mycetoma remained unidentifiable.

These observations suggest that aspergillosis can be successfully treated with itraconazole. We agree, however, with Dr Campbell’s group that itraconazole should not, at the present time, be recommended for treatment of aspergillosis but should be reserved for cases where treatment is required and a surgical approach is contraindicated. Further experience with this new triazole antifungal agent is necessary, and in the case of its use in the treatment of aspergillosa a multicentre study in which substantial numbers of patients could be recruited should be considered.

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