HISTOPLASMOSIS AS THE CAUSE OF A PATHOLOGICAL FRACTURE

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We report the case of an 82-year-old man with a pathological fracture of the hip caused by infection with Histoplasma capsulatum var capsulatum. He was treated by a hemiarthroplasty and with oral itraconazole.

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Fungal spores of Histoplasma capsulatum infect man when inhaled in dust from animal droppings. Histoplasmosis is endemic in parts of the Americas, and sporadic cases are seen in tropical and temperate countries.1 Classical histoplasmosis is a self-limiting febrile illness with pulmonary symptoms. African histoplasmosis caused by Histoplasma duboisii affects the skin, lymph nodes, and bone.1 In HIV-infected patients, latent histoplasmosis may be reactive, causing fungaemia and visceral disease. We now report a case of histoplasmosis which resulted in a pathological fracture.

Case report

An 82-year-old Asian man presented with a four-month history of pain in the right hip, anorexia and loss of weight. He had lived in India until 1947, in Mombasa, Kenya until 1988, and subsequently in the UK. He had visited Mombasa in 1994. He was investigated but no diagnosis was made. In June 2000 he attended with further severe pain in the hip. There was no history of trauma. Radiographs revealed a subcapital fracture of the right femoral neck secondary to a lytic lesion (Fig. 1). All movements of the right hip were limited. The chest radiograph was normal. He underwent a Thompson hemiarthroplasty. At operation the femoral head was macroscopically abnormal and was sent for histopathological
examination. After an uneventful recovery, he was discharged nine days after surgery.

At six weeks, he was mobile, the wound had healed and the radiographs were satisfactory. The histopathological examination revealed infiltration of the bone marrow by chronic inflammatory cells, including numerous plasma cells and macrophages and a few granulomata. There were also numerous intracellular and extracellular budding yeasts. Periodic acid-Schiff and the Grocott silver stain (Fig. 2) identified numerous small intracellular organisms as *Histoplasma capsulatum* var *capsulatum*. Staining for acid-fast bacilli was negative. He was treated successfully with oral itraconazole for one year.

At follow-up he was HIV-negative, with a normal white cell count, lymphocyte count and lymphocyte subsets, but with a low level of vitamin D (<13 nmol/l). His ESR was 54 mm/1h and the level of C-reactive protein was 4 mg/l. Protein electrophoresis showed elevated levels of IgG of 27.4 g/l (normal: 7.0 to 16.0), with two monoclonal bands (one lambda, one kappa). A bone-marrow aspirate contained an excess of plasma cells. It is probable that he will progress to myelomatosis in the future, but he currently remains well.

**Discussion**

This patient’s histoplasmosis was probably acquired in Kenya or India decades earlier. Immunosuppression by a combination of advanced age, deficiency of vitamin D, and his plasma cell dyscrasia, probably allowed active histoplasmosis to develop, localised to the femoral neck. He had *Histoplasma capsulatum* var *capsulatum* (up to 5 µm), and not the larger *Histoplasma capsulatum* var *duboisii* (8 to 14 µm), which causes histoplasmosis in Central and West Africa, south of the Sahara and north of the Zambezi river (Fig. 2).

Histoplasmosis is an acute or opportunistic fungal infection which only rarely causes isolated lesions in bone. It has not before, to our knowledge, been reported as the cause of a pathological fracture.

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**References**


