GASTRIC MUCORMYCOSIS : REPORT OF CASE IN A SWAZI

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Fungi such as the aspergilli, penicillia and mucors that were once familiar chiefly as laboratory contaminants are sometimes the cause of severe and often fatal disease. This is perhaps part of a changing pattern of disease effected by antibiotic-, steroid- and chemotherapy. Cases of mucormycosis are being reported in increasing numbers from the Americas and Europe. Usually they occur secondarily to some other disease, especially uncontrolled diabetes and leukaemia. Cortisone, broad-spectrum antibiotic and antileukaemic therapy may be predisposing factors.[1] The following case is the second to be published from South Africa although several others have been seen at the South African Institute for Medical Research in Johannesburg,[2] at the University of Natal,[3] and possibly elsewhere.

CASE REPORT

J.D., a Swazi man of about 50 years, came to the hospital on 31 May 1958 because of cough, blood-stained sputum, and sharp pain in the chest which began 3 weeks previously. Occasionally there was postprandial abdominal pain. The patient was weak and emaciated. Moist rales could be heard in both lungs and there was a palpable epigastric tumour. The temperature was subnormal. The urine was not tested. 600,000 units of procaine penicillin were given after admission and again the following morning. The patient died early in the morning of the third day in hospital. The clinical diagnosis was pulmonary tuberculosis and carcinoma of the stomach.

An autopsy was performed 48 hours *post mortem*. The principal findings were in the lungs and stomach. The left lung was dark, heavy and firm and from the cut surface a frothy haemorrhagic fluid could be expressed. No cavities were noted. The lower lobe of the right lung was similar to the left but in a lesser degree. Within the stomach there was a greyish-white tumour, about 3 × 2 inches, along the greater curvature near the cardia. The surface of this tumour was elevated about 3/8th inch above the surrounding mucosa and was covered with a gelatinous exudate. The remainder of the gastro-intestinal tract was normal by palpation, but was not opened. Sections of the stomach, lungs, liver, one kidney and aorta were put in 10% formalin and sent to the Armed Forces Institute of Pathology in Washington, D.C., whose report, by Elson B. Helwig, M.D., was as follows:

'The lesion of the stomach is composed of necrotic and haemorrhagic material which is diffusely infiltrated by numerous and non-septate hyphae typical of mucormycosis (Fig. 1). Vascular invasion, a characteristic feature of mucormycosis is also prominent in this case (Fig. 2). Since this fungus is not liable to penetrate intact mucosa it is likely that the infection followed some mucosal defect, possibly a gastric ulcer. Diligent search of sections of other organs did not yield any evidence of systemic spread by the fungus. Only one other case of gastric mucormycosis, very similar to the present one and contributed from Korea, is on file at the A.F.I.P. In addition, sections of the liver and lung show evidence of schistosomiasis; several ova with minimal tissue reaction are present, but are not sufficiently characteristic

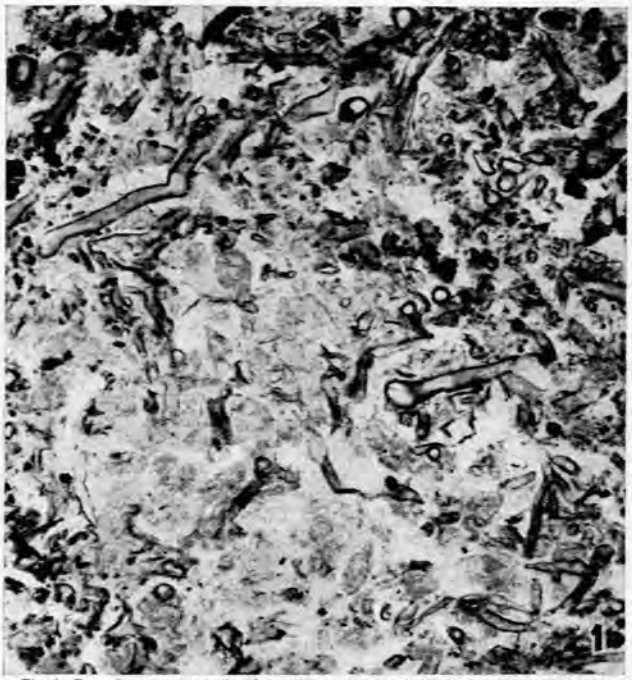

Fig. 1. Broad, non-septate hyphae of mucormycosis in the wall of the stomach, H & E, × 380. (*Armed Forces Institute of Pathology, Washington.*)

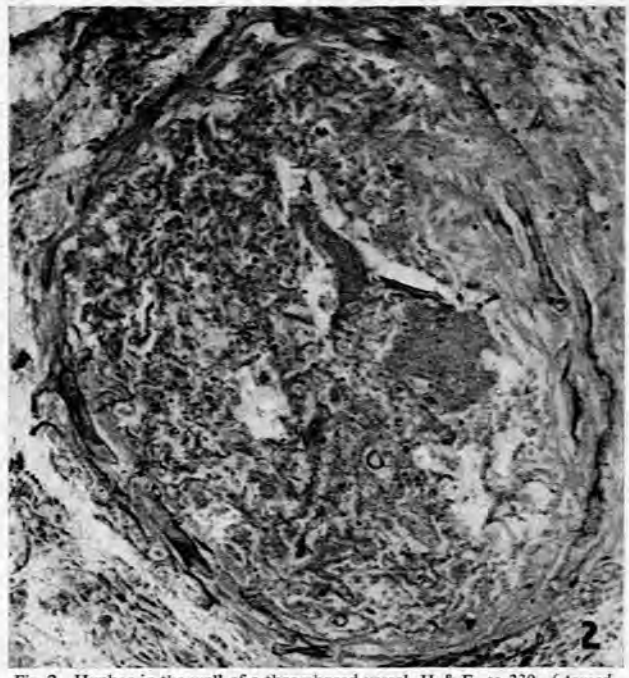

Fig. 2. Hyphae in the wall of a thrombosed vessel, H & E, × 330. (*Armed Forces Institute of Pathology, Washington.*)

to allow any further classification. Both lungs exhibit a severe and necrotizing pneumonitis. The aorta shows mild arteriosclerotic changes with thickening of the subintimal tissue, but does not manifest any evidence of syphilis.'

A nephew, contacted after the patient's death, said that the patient had been ill with a chronic cough for 3 years. He was fond of Bantu beer, especially *ngodwana*, or strong beer, and his family attributed his illness to this habit. During the last 3 years he worked as a policeman on various farms in the Lowveld. He never worked in the mines. His nephew last saw him 1 year before he died.

DISCUSSION

Mucormycosis is infection by fungi of the family Mucoraceae, which includes 3 genera, viz. Mucor, Absidia and Rhizopus.[4] They are ubiquitous, occurring as saprophytes on dead or decaying plants and animals and they often contaminate laboratory cultures.[5] In mucormycosis the characteristic histological finding is the presence of large, non-septate hyphae in arterial walls, associated with thrombosis and infarction.[1] It is necessary to culture the organism to decide in which genus it belongs and in the present case the identification cannot be refined beyond that of one of the Mucoraceae.

The most frequent sites for mucormycosis are the lungs and brain.[1] In the lungs there may be bronchopneumonia[6] or a lung abscess.[7] Both air-borne and haematogenous infections may occur.[1] In the brain the lesion is that of meningo-encephalitis, often with ophthalmoplegia, and usually found in the presence of uncontrolled diabetes.[8,9] Thrombosis of the lateral sinus has occurred.[2] The route of infection is probably from the nose *via* the paranasal sinuses or through the cribriform plate or from the eyes.[1,9]

Reports of gastro-intestinal mucormycosis in the literature are scanty so that as late as 1957 Baker could review only 5 cases, including 2 of his own.[10] Usually there is haemorrhagic necrosis and ulceration of the intestinal wall, sometimes with perforation.[5,10,11] Tumour-like lesions similar to the present case have been reported.[12] Mesenteric thrombosis may occur. All portions of the gastro-intestinal tract, including the oesophagus, may be involved. The clinical features are those of an ulcerative colitis, peritonitis and intestinal obstruction. Embolic abscesses may occur in the liver.[10,12] In none of the cases reviewed by Baker *et al.*[10] was the fungus noted clinically in the stool. The previous South African case is one of gastric mucormycosis in a 26-month-old Bantu child suffering from advanced kwashiorkor.[13] Malnutrition was also a prominent feature in another South African case, a Bantu infant,[2] and in 2 Brazilian cases, aged 10 months and 1 year.[11]

Gastro-intestinal mucormycosis may occur in the absence of an antecedent disease and is usually considered to be caused by ingested spores.[10] It is interesting to speculate on the role of *ngodwana* in this case. This is the Shangaan name for strong beer which is made as follows. Corn meal is mixed with water and allowed to stand for a week or 10 days. Then it is cooked in the manner of porridge, cooled and thinned with water. Kaffir corn (*Sorghum vulgare*) which has previously been allowed to sprout and then dried and partially ground is added with sugar to the corn-meal mixture. This final mixture is allowed to ferment 2 or 3 days, when drinking usually begins. With such favourable conditions for the growth of a fungus, perhaps many spores are ingested in drinking *ngodwana*. Fermented foods might have been a factor in other South African cases. The soft porridge given to Bantu children is usually cooked after fermenting. Sometimes, however, it may ferment after cooking (*amahewu*). The 2 Brazilian cases were also associated with malnutrition. The staple diet of the lower classes in Brazil is low in protein and rich in carbohydrates. Fermentation, however, is not a part of its preparation.[14]

Ingested spores are thought incapable of penetrating

intact mucosa and the possibility of a gastric ulcer in this case is suggested. In the absence of an adequate history we can only speculate. Recent autopsy studies in Uganda suggest a higher incidence of both gastric and duodenal ulcers amongst the Bantu than was previously suspected.[15]

Death in this case was probably caused by pneumonia. We are unable to say whether or not this preceded the gastric infection. Clark suggests that the presence of a tumour-like mass in gastro-intestinal mucormycosis may signify an infection of longer duration than usually occurs.[12]

An additional feature was the bilharzial ova in the liver and lungs. Bilharziasis is common here in the Lowveld. Dissemination of the ova probably decreased the patient's resistance and may have been a contributory factor in the fungus infection.

The clinical diagnosis of mucormycosis is not easy. In most of the reported cases an antemortem diagnosis was not made, perhaps because it was not suspected. At least 6 cases reported since 1955, however, have recovered.[9] Of these, 5 were cranial cases and the diagnosis was confirmed either by a culture or by a biopsy in 4 of them. The 6th was a pulmonary case, in which a lobectomy was done for lung abscess.[7] All 6 cases were in uncontrolled diabetics.

The possibility of mucormycosis should be kept in mind in any severe disease that is being intensively treated with antibiotics, steroids or antileukaemic agents. Also, in such a disease, worsening of the patient's condition should arouse suspicion that a fungus infection has possibly occurred. To confirm the diagnosis, cultures on Sabouroud's medium can be made from sputum, bronchial washings, nasal smears, gastric contents, stool, cerebrospinal fluid and the exudate from chronic ulcers. Biopsies may be taken from suspicious lesions. Laboratory contamination of cultures is likely and the spores of these fungi may be found in the nose, sputum and stomach of healthy persons. Thus close co-operation is necessary between clinician and mycologist to assess the significance of positive cultures.[5]

The treatment of mucormycosis is difficult. If diabetes is present, the hyperglycaemia should be carefully controlled and all steroid and antibiotic therapy stopped or at least re-evaluated.[1] Diabetic control was the chief feature in the 6 recovered cases. The following have also been used in one or more of these cases: potassium iodide, desensitization to the fungus, parenzyme, antibiotics and mycostatin.[9] The focus of infection was excised in 2 cases. The use of antibiotics in the treatment of 1 case, and also mycostatin is interesting, although of doubtful value. In another report the use of amphotericin B is suggested.[11]

SUMMARY

A case of gastric mucormycosis in a Swazi man, associated with a necrotizing pneumonitis, is presented. Mucormycosis, especially of the lungs and brain, usually occurs secondarily to some other disease such as uncontrolled diabetes and leukaemia. Gastro-intestinal mucormycosis is less often seen and may occur primarily. The fermented foods and beer of the Bantu may possibly be factors in gastro-intestinal fungus infections by the introduction of excessive numbers of spores.

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