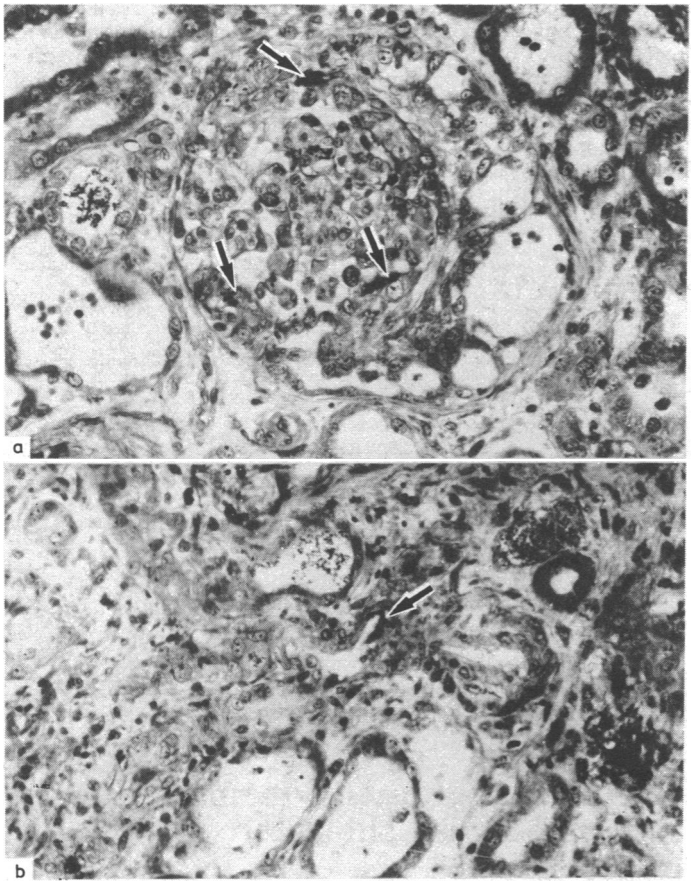
haemodialysis was started. Percutaneous renal biopsy two days later showed focal necrotising glomerulonephritis with the formation of crescents (figure (A)). Two small arterioles exhibited fibrinoid necrosis with the presence of needle shaped crystalline clefts (figure (B)). Immunofluorescence failed to detect deposits of immune reactants. Renal failure appeared to be irreversible, and he received maintenance treatment with haemodialysis. Six months later he died from a stroke. At necropsy the presence of characteristic crystalline clefts in several arteries and arterioles confirmed the diagnosis of renal cholesterol embolisation.



A: Glomerules showing focal necrosis (arrows) and extracapillary reaction. B: Arteriole with necrosis (arrow) around needle shaped crystalline cleft. Masson's trichrome × 300 (original magnification).

Comment

Our patient had evidence of atheroembolic disease in the retina, brain, and kidney. The necrotising glomerulonephritis was probably directly related to renal cholesterol embolisation because fibrinoid necrosis around a cholesterol crystal was observed in the renal biopsy specimen. Necrotising angiitis of distal vessels associated with cholesterol microemboli has been described in other organs,[3 4] and cholesterol embolisation may result in a multisystemic disease simulating polyarteritis nodosa.[3] Our case indicates that necrotising angiitis resulting from cholesterol embolisation may also occur in the kidney and lead to severe glomerulonephritis with formation of crescents similar to those observed in the microscopic form of polyarteritis nodosa.

Cholesterol embolisation may occur spontaneously but is usually a complication of angiographic procedures or aortic surgery.[1] In our case aortic catheterisation for carotid angiography may have had a role in the development of renal embolisation; the time between the angiography and the onset of renal failure was consistent with this hypothesis.[1] The nephropathy of our patient was rapidly progressive and irreversible, as usually occurs after renal cholesterol embolisation.[5]

Cholesterol embolisation may result in protean clinical manifestations and is recognised as a possible cause of renal failure in patients with aortic atheromatosis.[1] We suggest that necrotising glomerulonephritis should be included in the clinical manifestations of cholesterol embolisation.

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Micro-organisms isolated from skin under wedding rings worn by hospital staff

Little evidence exists to support theories on the risk of infection from rings worn by hospital staff. The bacterial flora of skin under rings is not predictable because changes encouraged by occlusion could be offset by the release of toxic metal ions, such as silver and copper, from gold alloys. We surveyed the microflora isolated from skin under rings worn by hospital staff.

Methods and results

Fifty nurses working on medical and surgical wards who permanently wore rings took part in the survey. Rings were removed and the skin underneath sampled with a swab that neutralised the residual action of any skin disinfectants. A similar area on a non-adjacent finger of the same hand served as a control site. Bacteria were grown on caesin, yeast extract, lactose, and glucose agar and MacConkey agar number 3 (Oxoid) and Gram negative bacilli identified by API 20E (API Laboratory Products) and conventional tests. The lower limit of detection was 10 colony forming units/swab. Nurses whose ring site yielded Gram negative bacilli were sampled at intervals for five months. At the end of the survey samples were obtained from all of the nurses who were still working at the hospital.

Occurrence and distribution of Gram negative species colonising skin under rings

Organism	No of staff colonised	No of colony forming units/swab
Enterobacter cloacae	10	10-24 000
Klebsiella pneumoniae ssp aerogenes	5	10-2 200 000
Acinetobacter calcoaceticus	3	110-560 000
Pseudomonas aeruginosa	2	7 200-40 000
Serratia marcescens	1	48 000
Proteus mirabilis	1	50
Providencia stuartii	1	14 000

The numbers of organisms comprising the normal Gram positive flora were significantly increased at ring sites, with a geometric mean of 1600/swab from the skin under rings and 180/swab from control sites ($p < 0.001$, paired t test). Of the 50 nurses originally sampled, 20 had Gram negative bacilli on the skin under their rings and one also had Gram negative bacilli on the control site. The geometric mean number of Gram negative bacilli on these ring sites was 730/swab (range 10 to 2 760 000). The table shows the distributions of some species found. Other species isolated (and the number of nurses colonised) were: *Enterobacter agglomerans* (three), *Ent aerogenes* (one), *Klebsiella oxytoca* (three), *Pseudomonas paucimobilis* (one), *Ps putida* (one), *Serratia liquifaciens* (one), *Aeromonas hydrophilia* (one), *Kluyvera sp* (one), *Citrobacter freundii* (one), and *Escherichia coli* (one). The single control site that showed Gram negative bacilli yielded 50 *Kleb pneumoniae ssp aerogense*, which were indistinguishable from those on the ring site. The ring site was colonised by one species in eight cases, by two species in eight, by three species in three, and four species in one.

Over the five months of the study 16 of the original 20 colonised nurses had Gram negative bacilli on the ring site on at least one other occasion. In most cases the strains were present each time the nurses were sampled (up to six samples). The final sample from two nurses yielded Gram negative bacilli although these had not been present in the initial sample.

Serological and bacteriophage typing of the organisms showed that the same strains were persistently isolated from most subjects, and sometimes two different strains of the same species were repeatedly present.

Comment

Little work has been done on the effect of wearing rings on the microflora of skin. One study showed an increase in the normal Gram positive flora but did not mention colonisation by Gram negative organisms.[1] The pattern of isolation of Gram negative bacilli in our study suggested that these organisms are colonisers rather than transient contaminants because the same strains were persistently isolated over several months. Repeated isolation of *Ent agglomerans*, *Kleb pneumoniae*, and *Ps aeruginosa* from the whole hand has previously been shown.[2 3] Much attention has been given to hands as vectors of infection in hospitals, and direct contact is an effective method of transfer.[4] The clinical importance of such carriage remains to be evaluated, but as the organisms isolated include those responsible for many infections in hospitals,[5] the possibility that such bacteria can permanently colonise the hands of hospital staff wearing rings should be borne in mind in high risk wards and theatres.

We thank Mrs J A Crees-Morris, Mr H Todd, and Mr M A Gaston for typing isolates, and Mr C A Mackintosh for statistical analysis.

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Frontal sinusitis caused by Myriodontium keratinophilum

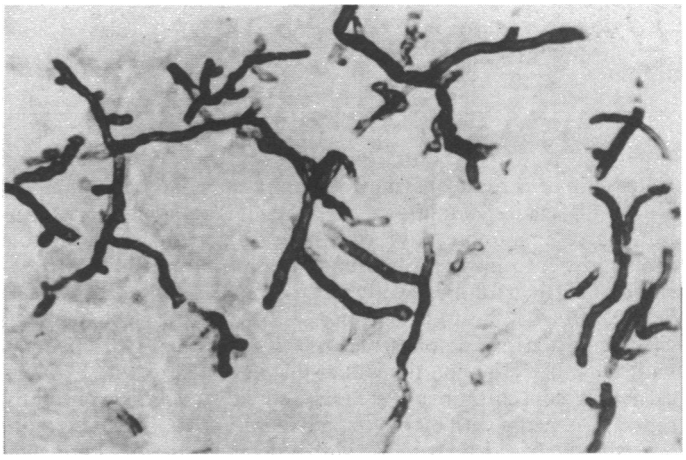
We report the first case of infection with *Myriodontium keratinophilum* in man.

Case report

A 53 year old Nigerian business man was healthy until early 1980, when he suffered from facial pain. Sinusitis secondary to nasal polyps was diagnosed, and nasal polypectomy was performed in Ibadan in March. The polyps recurred often and at short intervals, so polypectomy was repeated in April 1980, May 1982, and August 1982. Four months after this last operation he noticed a swelling on his forehead, which gradually enlarged and was accompanied by left proptosis.

A mucocele of the frontal sinus was diagnosed clinically and radiologically, but when an osteoplastic frontal flap was raised in September 1983 the sinus was found to contain brown necrotic like material. This had eroded the roof of the left orbit and both ethmoid bones, causing polypoidal mucosa to block both sides of the nose. The posterior wall of the frontal sinus was thinned and eroded. The eosinophil count was 4% and the erythrocyte sedimentation rate 61 mm in the first hour. The frontal and ethmoidal sinuses were exenterated and drainage tubes placed in the frontal sinus via the nose.

The tissue lining the sinus consisted of floridly inflamed granulation tissue containing large numbers of plasma cells and eosinophils; most of the mucosal lining was ulcerated. Few macrophages were present, there were no giant cells, and there was no evidence that the tissue had been invaded by the fungus in the lumen. The contents of the sinus were mainly cellular debris containing Charcot-Leyden crystals with some foci of viable cells, including eosinophils. Scattered throughout were fungal hyphae, which were scanty in most areas. The hyphae were branched and septate



Histological section of contents of frontal sinus, showing hyphae of *M keratinophilum*. Methenamine silver ×750 (original magnification).

(figure) and were initially thought to be a species of *Aspergillus*. Closer examination showed that they differed from aspergillus because they branched non-dichotomously and appeared ribbon like and folded. These features are characteristic of mycetoma of the paranasal sinus seen in the Sudan[2] and of allergic aspergillosis of the paranasal sinus.[3]

Growth of the hyphae yielded a slow growing white mould that produced a few single celled conidia on long denticles along the sides of the fertile hyphae. This isolate was identified by Dr B L Brady, Commonwealth Mycological Institute, Kew, Surrey, as *M keratinophilum*. An antigenic extract prepared from the isolate produced a single diffuse precipitin line when it was tested against the patient's serum by double diffusion. No reaction was observed when comparable extracts of *A flavus*, *A fumigatus*, and *A versicolor* were tested against the serum.

The patient was treated with ketoconazole 200 mg daily for three weeks, returned to Nigeria, and had recovered by October 1984 with no recurrence.

Comment

M keratinophilum was reported in 1978 as a new species.[1] It was isolated from soil in Italy and California, and from the penis of a bull in Germany. The Commonwealth Mycological Institute has identified isolates from the hair of shrews and cats in the United Kingdom and also from an unknown source in Nigeria. The species therefore appears to be widespread in nature, especially where keratinous substrates are present. It is not related to other species that are pathogenic in man.

Although antifungal treatment is indicated in the management of fungal sinusitis, it is unlikely to be effective without surgical intervention, which enables a fresh specimen to be used for isolation and identification of the causal organism. Frontal sinuses should be drained via an enlarged frontal nasal duct and maxillary sinuses through an intranasal antrostomy.

This is the first documented case of fungal infection with *M keratinophilum* in man. It exhibits many features of aspergillosis of the paranasal sinuses, which affects fit, healthy subjects. Our patient was neither immunosuppressed nor diabetic.

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