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Conidiobolus coronatus infection of the para-nasal sinuses masquerading as a sino-nasal tumour

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Introduction

Fungal infections of the para nasal sinuses are uncommon [1, 2]. We report a rare case of Conidiobolus coronatus infection of the para-nasal sinuses in a 32-year old immuno competent male presenting as a sino-nasal tumour extending to the brain which completely resolved following resection and anti fungal therapy. Nasal sinus infection due to C. coronatus has not been reported previously in Sri Lanka.

Case report

A 32-year old man working in a hardware store presented with right nasal obstruction, nasal discharge and frequent sneezing of three months duration. He had no other significant medical illness. Examination revealed enlarged right turbinate obstructing the nasal cavity and fullness over the maxillary area. CT scan of the para-nasal sinuses revealed an extensive soft tissue growth in the right maxillary antrum, involving both ethmoids and sphenoid sinuses with erosion of the cribriform plate with extension into the frontal lobe of the brain. A biopsy was taken under anaesthesia by the infra turbinate route and sent for histology. Heamatoxylin and eosin (H&E) stained sections revealed fibro connective tissue showing extensive areas of necrosis with eosinophil infiltration and eosinophilic vasculitis. There was no evidence of a neoplasm in multiple sections examined. A possibility of Wegener’s granulomatosis was suggested. Anti neutrophilic cytoplasmic antibody to cytoplasm and peri-nuclear antigen were negative. A second biopsy was taken and a piece was sent to the Medical Research Institute for mycological studies. C. coronatus / Entomophth oraceous fungus was isolated on direct microscopy and culture. H&E stained sections revealed granulomas with foreign body type multinucleated giant cells, chronic inflammation with abundant eosinophils. Periodic acid schiff and Grocott’s stain revealed fungal filaments. Right lateral rhinotomy and medial maxillectomy was done. The patient was treated with potassium iodide (KI), ithaconazole and cotrimoxazole for six months. Liver function tests, blood urea and creatinine was monitored every two weeks. After four weeks there was an increase in alanine transaminase enzymes (ALT) and KI dose was adjusted to maintain the ALT level within the normal range. CT scan was done two months after commencing therapy and compared with the initial films and showed a marked reduction in soft tissue swelling of the nasal cavity, ethmoids, maxillary and sphenoid sinuses, indicating a good response to anti fungal therapy. There was mucosal thickening in all the sinuses but no soft tissue growth in the nasal cavity or erosion of the cribriform plates. Four months later right rigid nasal endoscopy revealed nasal adhesions. Nasal septoplasty with submucous resection was done. CT scan six months after initial presentation showed complete clearance of the sinuses. The patient was free of recurrence 60 months later.

Discussion

Invasive paranasal sinus fungal infections are mostly described in South East Asian and Chinese people and occur in immunocompetant hosts [1-3]. Intra cranial extension of sinus infection with bone erosion is described in aspergillosis and mucormycosis with a very good response to anti fungal treatment [4,5]. Intra cranial extension has not been previously reported in C. coronatus infection [1]. Radiological imaging of chronic sinusitis due to fungi sometimes mimic a tumour with bone erosion and intracranial spread [6]. Infection with C. coronatus causes chronic granulomatous lesions of the nasal sinuses spreading to contiguous skin and invading blood vessels. Bone erosion mimics a malignant tumour. This fungus is also called Entomophthora mycosis, Conidobolus mycosis, Conidibolus and Rhinoentomoph thromycosis [6]. This infection affects healthy adult males but is also reported in immuno compromised individuals [7]. Patients commonly present with chronic sinusitis and nasal obstruction often mimicking a sino-nasal tumour. The mode of transmission is inhalation of fungal spores.

C. coronatus has a universal, mainly tropical distribution. It is a saprophyte in soil and on decaying vegetation. It has been isolated in tropical rain forest of Asia, insects and in dolphins. This fungus grown on culture media produce villous conidia giving a lacie like
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appearance. Colonies grow rapidly and are flat, cream coloured and become radially folded covered by a fine powdery white surface consisting of mycelium and conidiospores. Tan coloured colonies become brown with age [7].

Treatment consists of a combination of three or more drugs and surgery, KI, amphoteracine B, ketaconazole and itraconazole [1]. Our patient had complete clearance of the nasal sinuses on CT scan 6 months after the initial presentation and was free of recurrence 72 months later on follow up. Long term follow up is required as recurrences are common.

References


Neck stiffness and papilloedema due to Harada syndrome

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Case report

A 41-year old previously healthy woman presented with severe headache, vomiting, low grade fever, bilateral eye pain, photophobia and deteriorating vision over one week. Examination showed neck stiffness and bilateral papilloedema [Figures 1 and 2]. She had no evidence of autoimmune disorders. Contrast CT brain and routine investigations were normal. CSF showed pleocytosis with negative microbiological tests. Worsening visual acuity in the absence of meningitis or an intracranial space occupying lesion prompted an urgent ophthalmological assessment which showed bilateral disc oedema with multiple exudative detachments of the sensory retina with vitreal cells, suggestive of posterior uveitis. This led to the clinical diagnosis of an incomplete form of Harada syndrome, as she had no auditory or cutaneous signs [1]. She had an excellent response to intravenous methylprednisolone followed by oral steroids. Serial visual and fundoscopy assessments showed gradual improvement in visual acuity, disc oedema and retinal detachments, and visual acuity became normal after one month of treatment [Figures 3 and 4]. Further supporting our diagnosis, four months later she presented with significant hair loss, but without any poliosis or vitiligo ( integumentary signs).

Vogt-Koyanagi-Harada (VKH) syndrome is a rare inflammatory granulomatous disorder involving melanocyte containing organs causing bilateral pan-uveitis and retinitis (ophthalmic), meningismus and cerebrospinal fluid pleocytosis (neurological), tinnitus (auditory) and alopecia, poliosis, and vitiligo ( integumentary) manifestations [1]. It has HLA-DR4, HLA-DR53, and HLA-DQ4 associations [2]. The disease commonly affects dark skinned people, women more than men, of any age group. Although an infectious aetiology is often postulated, autoimmune origins have also been considered as VKH is associated with certain autoimmune disorders such as type I diabetes mellitus, Hashimoto’s thyroiditis, polyglandular syndrome and IgA nephropathy (3, 4, 5). Exclusion of a history of ocular trauma, surgery or

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