Acquired Immune Deficiency Syndrome (AIDS) presenting as a nasal septal perforation*

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SUMMARY *Patients infected with the Human Immunodeficiency Virus (HIV) and those with AIDS may present with many head and neck manifestations. We report a case of an undiagnosed HIV positive male who presented with symptoms due to a nasal septal perforation, and rapidly developed AIDS. The histopathology of the perforation margins revealed active chronic inflammation with no evidence of neoplasia or granuloma. No viral or fungal infection was demonstrable on immunological testing and fungal stain. This is the first reported case of a patient developing AIDS presenting with a nasal septal perforation.*

Key words: Acquired Immunodeficiency Syndrome, HIV, nasal septum, nose disease.

INTRODUCTION

Patients with AIDS can exhibit various head and neck conditions which will bring them into contact with otolaryngologist (Marcuson and Sooy, 1985; Corey et al., 1991; Young, 1997). Acute and chronic rhinosinusitis may occur in HIV patients (Drake-Lee, 1997). These patients present with symptoms of nasal obstruction, rhinorrhoea, facial pain, alteration of smell and a post nasal drip. Allergic rhinosinusitis may arise de novo, or be increased in HIV patients (Small et al., 1993). Opportunistic infections, including fungal, viral, or mycobacterial infections which rarely occur in the immunocompetant patient may occur (Godofsky et al., 1992). In these patients there may be mixed infection. Nasal polyps and nasal tumours, including lymphomas and Kaposi's sarcomas, have been reported in AIDS patients (Drake-Lee, 1997).

Nasal septal perforation in HIV and AIDS patients has been attributed to *Varicella zoster* (Colebunders et al., 1997) and Non-Hodgkin's lymphoma (Gold et al., 1990). A palatal perforation due to *Histoplasma Capsulatum* has been reported as presenting symptoms in an AIDS patient by Fowler et al., 1989. To our knowledge there has been no previous reports of a patient with a nasal septal perforation as the presenting feature of AIDS.

CASE REPORT

A 42 year old homosexual man was referred with a three month history of nasal obstruction. He complained of a post nasal drip, occasional blood stained rhinorrhoea, mid-facial pain and sneezing. He had no history of nasal trauma or nasal surgery. He had been using topical Oxymetazoline for the duration of his symptoms.

Anterior rhinoscopy revealed a grossly deviated septum and a large septal perforation involving virtually the total extent of the quadrilateral cartilage (Figure 1). The nasal mucosa was noted to be inflamed and granular.

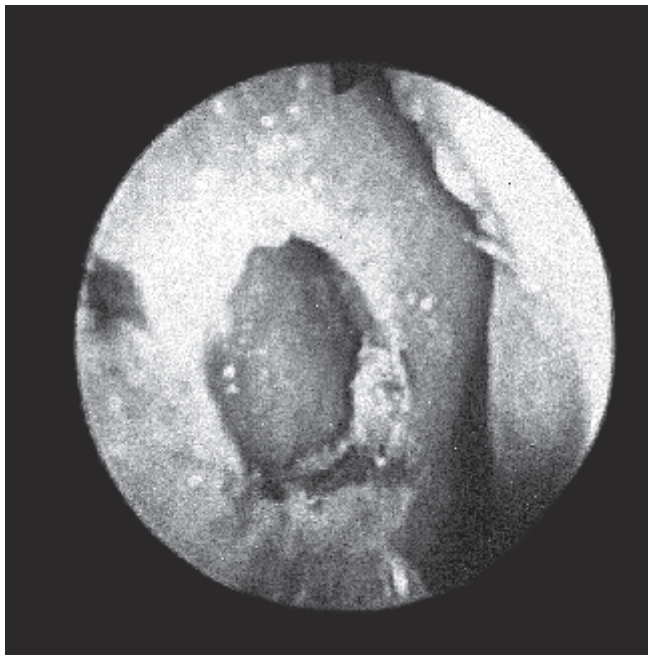

Figure 1. Large septal perforation.

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A full blood count, urea and electrolytes and C-reactive protein level was normal. The erythrocyte sedimentation rate was 100 millimetres per hour. Screening for anti-neutrophilic cytoplasmic antibody (ANCA) was negative. Due to the large and inflamed nature of the perforation and short history, a CD4 count was requested. His CD4 count was $0.1x10^9$ (normal 0.6-1.10) and the helper/suppressor cell ratio was 0.15 (normal 1-2.7).

The edges of the septal perforation were biopsied under local anaesthesia in the out-patient clinic. The histopathology of this biopsy showed chronic active inflammation only. There was no evidence of cytomegalovirus (CMV) inclusions, fungal hyphae, spores or acid fast bacilli. Immunohistochemistry for *Herpes simplex* was negative. The histopathology showed non-specific active chronic inflammation with ulceration.

In the light of his depressed CD4 count he was referred to the department of Genitourinary Medicine for appropriate counselling and an HIV test. He had previously had an HIV test in 1986 that was negative. On this occasion his HIV test was positive.

His Syphilis serology (VDRL, THA and FTA) was negative as were nuclear and tissue autoantibody screens. His serum immunoglobulins were raised with an IgG level of 19.11 (normal 6-16), Ig A 5.8 (normal 0.8-2.8) and IgM 8.39 (normal 0.5-1.9). His chest x-ray was normal.

One month after presentation to the ENT clinic he developed oral candidiasis which was treated with oral Fluconazole. A month later he was noted to have an active herpetic infection in the perianal region and oral hairy leukoplakia of the tongue. He subsequently developed cytomegalovirus retinitis for which he was treated with intravenous Gancyclovir and a course of antiretroviral treatment. Eight months following his presentation he is stable and under regular review.

DISCUSSION

The known causes of nasal septal perforation include trauma (surgical, repeated cautery, digital trauma); malignant disease (carcinoma and lymphoma), chronic inflammation (Wegenerís, syphilis, TB, candida, lupus erythromatosus and rheumatoid arthritis) and poisons (industrial, cocaine addicts, topical corticosteroids and topical decongestants). In a large proportion of patients the aetiological process remains unidentified (Brain, 1997).

Nasal septal perforation in patients known to be suffering from AIDS may result from a variety of causative organisms. Wiest et al., 1987 described a nasal septal perforation due to *Staphylococcus aureus* while Henry et al., in 1988 described a perforation as the result of *Alternaria alternata* (a filamentous fungus). Gold et al. described a perforation resulting in a T-cell Non-Hodgkin's lymphoma and Colebunders attributed nasal septal perforation to *Varicella zoster* in 1997. These nasal septal perforations occurred in HIV patients in the stage IV of the 1993 Communicable Disease Centre Classification of HIV (Centres for Disease Control, 1993).

A palatal perforation in an AIDS patient infected with *Histoplasma capsulatum* and *Candida albicans* was described by Fowler et al., 1989.

Our patient presented with symptoms due to his nasal septal perforation. He rapidly developed features of full blown AIDS (HIV stage IV), including oral candidiasis and CMV retinitis.

The aetiology of his nasal septal perforation remains unknown. There was no histological evidence of active infection, granuloma or malignancy. There was no serological evidence for autoimmune disease. The inflamed nasal mucosa surrounding the perforation and active chronic inflammation demonstrated histologically, are not in-keeping with a septal perforation of traumatic origin.

The patient had used nasal decongestants after the onset of his symptoms but not before. The use of phenylephrine has been reported as producing nasal septal perforations (Vilensky, 1982). This was after prolonged use (two years), the perforation was only 1 centimetre in diameter and the aetiology of the perforation was questionable as the patient had TB and postnasal discharge when she started using the decongestant.

During the year prior to presentation to the ENT clinic, our patient had presented to his General Practitioner with an upper respiratory tract infection and had received Erythromycin for one week. It is remotely possible that his symptoms of an upper respiratory tract infection were complicated by a septal abscess due to *Staphylococcus aureus,* which was subsequently eliminated by this Erythromycin in a similar fashion as described by Henry et al., (1988), but he had persisting symptoms within the three months before referral for an ENT opinion. Unusual or multiple opportunistic pathogens may infect an immunocompromised patient and these may be difficult to demonstrate despite good clinical evidence of infection (Upadhyay et al., 1995).

Besides the well documented immunocopromised state an atopic tendency may develop in AIDS patients (Small et al., 1993). This suggests that the immune system can be modulated in both a depressed and an exaggerated direction. Our patient had an ESR of 100. Although no specific autoantibodies known to be associated with septal perforations were detected, such as ANCA, it is possible that a destructive autoimmune or exaggerated allergic process may have contributed to his septal perforation.

The management of patients with nasal septal perforations aimed at the identification of the underlying aetiology, treatment of this and repair of the perforation if indicated. In the previous case reports of nasal septal perforation in AIDS patients, none lived long enough to have the perforation dealt with. In any immunocompromised patient opportunistic infections should be dealt with as a matter of urgency and the clinician should maintain a high index of suspicion, particularly for fungal infestations and pathogens which do not commonly occur in immunocompetent patients.

In our patient the treatment of his other AIDS opportunistic infection has taken precedence over dealing with his septal perforation. His CMV retinitis has been dealt with and the antiretroviral treatment has re-established a more competent immune system. He will be treated at a later stage once his condition is entirely stable.

CONCLUSION

This is the first reported case of nasal septal perforation being the presenting feature of an HIV patient progressing to full blown AIDS.

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