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Malignant otitis externa in HIV and AIDS

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Abstract
Malignant otitis externa is a necrotising infection of the external ear canal which may spread to include the mastoid and petrous parts of the temporal bone, leading to skull base osteomyelitis. It is almost exclusively caused by infection with *Pseudomonas aeruginosa*, and usually occurs in elderly non-insulin-dependent diabetic patients. However isolated cases have been reported in a small number of non-diabetic patients, particularly in children who are immunocompromised due to malignancy, malnutrition and severe anaemia. In 1984 a case of malignant otitis externa was reported in a child with an acquired immunodeficiency syndrome (AIDS)-like illness, prior to identification of the human immunodeficiency virus (HIV). Since that time further sporadic cases of this invasive infection have been reported in HIV and AIDS. We present two further cases and also a review of the current literature.

Key words: Otitis externa, malignant; Acquired immunodeficiency syndrome; HIV; *Pseudomonas aeruginosa*

Introduction

In the acquired immunodeficiency syndrome (AIDS), opportunistic infections occur which are usually related to defects in cell mediated immunity. However, in recent years there has been an increase in the incidence of bacterial infections in this population (Witt *et al.*, 1987; Schrager, 1988). This includes infections caused by *Pseudomonas* species and recent reports have cited *Pseudomonas aeruginosa* as the Gram negative enteric organism most frequently accounting for bacteraemia and pneumonitis (Witt *et al.*, 1987; Rolsten *et al.*, 1990). Malignant otitis externa in HIV and AIDS was first reported in 1984 in a seven-month-old Haitian boy with an acquired immunodeficiency like illness, manifest by failure to thrive, oral candidiasis, pulmonary infiltrates and protracted diarrhoea. At that time the human immunodeficiency virus had not been isolated. In 1990 a case of malignant otitis externa was reported in a HIV-seropositive Hispanic male, and since that time further sporadic cases have occurred. *Pseudomonas aeruginosa* has been the causative organism in all but two cases which involved the fungus *Aspergillus fumigatus*. A full review of all reported cases of malignant otitis externa in HIV and AIDS is presented in Table I.

The clinical manifestations of malignant otitis externa in HIV and AIDS are similar to those in non-AIDS patients. The commonest presenting symptom is otalgia which is severe, unremitting and tends to be worse at night. This may be associated with a severe headache located in the temporal or mastoid region and also pain and tenderness over the temporomandibular joint. Purulent otorrhoea occurs in the majority of cases, but may vary from a slight discharge to a profuse foul smelling greenish exudate. There may be a history of recent ear syringing. Examination of the ear reveals granulation tissue in the external ear canal particularly at the junction of the bony and cartilaginous regions. In addition erythema is present throughout the remainder of the ear canal and the tympanic membrane. Cranial nerve palsy may occur at any time during the course of the illness but usually occurs approximately two months after the onset of otitis externa. In non-AIDS patients the commonest cranial nerve to be affected is the facial nerve with an incidence ranging from 24 to 43 per cent. Other cranial nerve palsies secondary to the skull base osteomyelitis, have an incidence of 14 to 35 per cent (Rubin and Yu, 1988). Isolated reports of other complications include brain abscess (Soliman, 1978), sphenoidal sinusitis (Youngs and Bagley, 1986), mycotic aneurysm (Watson, 1977), parotitis (Caruso *et al.*, 1977), and venous sinus thrombosis (Evans and Richards, 1973; Chandler, 1974).

The diagnosis of malignant otitis externa depends on a high index of clinical suspicion, especially in AIDS patients where pseudomonas-related infections are increasing in frequency. It is therefore important to culture exudate from the external ear canal in any AIDS patient with a discharging ear. If malignant otitis externa is suspected, imaging may include technetium bone scanning or gallium scanning but computed tomography is the technique of choice. The treatment of malignant otitis externa in non-AIDS patients involves systemic and local antibiotics combined with frequent debridement of the ear canal. Recently monotherapy with ciprofloxacin has shown excellent results, probably because it reaches high concentrations in bone and cartilage. Other advantages include low toxicity and good systemic absorption from oral administration (Weinroth *et al.*, 1994). In patients with AIDS, there are some advantages in combination

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TABLE I

A REVIEW OF THE LITERATURE ON MALIGNANT OTITIS EXTERNA IN PATIENTS WITH HIV AND AIDS

Case Report Author	Age (y)	Sex	Presenting history	Prior HIV related disease	Examination	CD4 count	WBC count	Culture	CT scan	Treatment	Outcome
Scott *et al.* (1984)	7	M		Failure to thrive Oral candidiasis Pulmonary infiltrates Hepatosplenomegaly Lymphadenopathy Protracted diarrhoea No HIV test available at this time	External ear canal abscess	330/mm^3	4140/mm^3 (absolute lymphocyte count)	*Pseudomonas aeruginosa*			Cured (at time of publication)
Rivas Lacarte and Pumarola Segura (1990)	23	M	L otalgia and otorrhoea for 1 month L hemifacial parasthesia	HIV positive	Oedema and granulation tissue in ear canal Deformity mandibular region of face Hyperaesthesia L hemiface			*Pseudomonas aeruginosa*	Soft tissue fullness of the ear canal and mastoid Thinning of bone of external auditory meatus (EAM) but no bony erosion	Ampicillin Gentamicin systemic + local followed by Ciprofloxacin	Cured at 3 weeks No recurrence at 6 months
McElroy *et al.* (1991)	35	M	R otalgia and otorrhoea for 2 weeks	HIV positive for 4 years Idiopathic nephotic syndrome AIDS Oesophageal candidasis *Pneumocystis carinii* pneumonia	Distension of R side of face and forehead Indurated and occluded R external auditory canal R facial nerve palsy		500/mm^3 19% neutrophils 39% lymphocytes	*Pseudomonas aeruginosa*	Marked soft tissue swelling with obliteration of the EAM but no bony erosion	Ceftazidime Imipenem Cilastatin Amikacin	Death (3 days later) due to overwhelming infection
Reiss *et al.* (1991)	(1) 42	M	Previous L mastoidectomy Recurrent L otorrhoea for many years L facial nerve palsy	AIDS for 1 year following Cutaneous Kaposi's sarcoma *Candida* oesophagitis	L facial nerve palsy Exposed temporal bone	60/mm^3		*Aspergillus fumigatus*		Amphotericin B Itraconazole (oral)	Cured (7 months later)
	(2) 30	M	Previous L mastoidectomy R sided hearing loss R otalgia and otorrhoea	AIDS for 2 years following *Candida* oesophagitis	Exposed temporal bone	30/mm^3		*Aspergillus fumigatus*		Amphotericin B Itraconazole (oral)	Death (7 months later) due to cerebral toxoplasmosis and *pseudomonas* septicaemia
Kielhofner *et al.* (1992)	38	M	L otalgia and otorrhoea L auricle inflammation for 3 weeks	AIDS *Pneumocystis carinii* pneumonia	L auricle and ear canal red and markedly swollen Granulation tissue in ear canal	22/mm^3	3500/mm^3	*Pseudomonas aeruginosa*		Ticarcillin Tobramycin – systemic + local	Cure (at time of publication)
Daniels *et al.* (1992)	44	M	R otalgia and hearing loss for 1 week followed by severe R otalgia and otorrhoea R facial nerve palsy for 4 days	AIDS for 1 year *Pneumocystis carinii* pneumonia	Complete R facial palsy Total R sided deafness Purulent discharge in ear canal			*Pseudomonas aeruginosa*	Soft tissue infiltration in the right auditory canal extending to the middle ear No evidence of bone destruction	Ceftazidime Metronidazole	Resolution of otalgia Partial recovery of facial nerve Death (3 months later) due to cryptosporidial diarrhoea
Loranzo *et al.* (1992)	28					55/mm^3	'Neutropenic'	*Pseudomonas aeruginosa*			Cured (at time of publication)
Weinroth *et al.* (1994)	32	M	R otorrhoea and minimal otalgia for several months followed by R temporal headache fever and otalgia	AIDS for 1 year *Pneumocystic carinii* pneumonia Kaposi's sarcoma Rectal herpes Oral candidiasis	Inflamed and swollen R ear canal Exposed bone in ear canal	3/mm^3	1520/mm^3 25 neutrophils 47% lymphocytes	*Pseudomonas aeruginosa*	Soft tissue fullness of the external ear canal Bony erosion of the postero-inferior wall of ear canal	Ciprofloxacin (oral) followed by Ceftazidime Tobramycin Corticosporin (local)	Cured (at time of publication)
Mendelson *et al.* (1994)	(1)			AIDS				*Pseudomonas aeruginosa*		Ceftazidime	
	(2)			AIDS				*Pseudomonas aeruginosa*			

chemotherapy for severe infections caused by *Pseudomonas aeruginosa.*

Case reports

Case 1

A 33-year-old male homosexual was diagnosed HIV positive in November 1990 and as having AIDS in January 1991, following AIDS-defining diagnoses of cerebral toxoplasmosis and cryptosporidiosis of the bowel. He was successfully treated with clindamycin and pyrimethamine and commenced on zidovudine (AZT) 250 mg twice daily. His full blood count at this time revealed a white cell count of 1.9×10^9 (neutrophils 0.9; lymphocytes 0.5) and his CD4 count was $50/mm^3$. In January of 1992 he was admitted to hospital with a four-week history of progressive otalgia and purulent otorrhoea of the left ear. The left auricle and external ear canal were inflamed, with purulent material and granulation tissue obscuring the tympanic membrane. After aural toilet, an ulcer with exposed bone and cartilage was revealed on the postero-inferior wall of the external ear canal at the junction of the bony and cartilaginous parts. Facial nerve function was intact. Examination of the mouth revealed hairy oral leukoplakia and a Kaposi's sarcoma on the palate. Microbiological swabs were taken for culture and biopsies were taken of the granulation tissue and the ulcer for histology. A full blood count at this time revealed a white cell count of 1.1×10^9 (neutrophils 0.6; lymphocytes 0.3) and his CD4 count was $60/mm^3$. Culture of the purulent exudate revealed *Pseudomonas aeruginosa* sensitive to ciprofloxacin, gentamicin and polymyxin. Histology revealed non-specific inflammatory changes. The patient was commenced on intravenous ciprofloxacin 500 mg twice daily and his condition markedly improved over 48 hours. This treatment was continued for a further three weeks with continued resolution at which point he was converted to oral ciprofloxacin for a further four weeks and discharged home. In addition to this, he underwent regular aural toilet and received gentisone HC ear drops. The ulcer healed slowly over a period of five weeks and at three months there was no evidence of recurrent otitis.

Case 2

A 59-year-old male homosexual was diagnosed HIV positive in February 1990 and as having AIDS in February 1993, following the AIDS-defining diagnosis of Kaposi's sarcoma of the foot. At this time his full blood count revealed a white cell count of 2.0×10^9 (neutrophils 0.8; lymphocytes 0.5) and his CD4 count was $100/mm^3$. In July of 1993 he was referred to the ENT department with a two-week history of severe otalgia, purulent otorrhoea and hearing loss in the left ear associated with trismus. He had initially been treated with amoxycillin with little improvement. On examination there was occlusion of the left external ear canal due to oedema and a purulent exudate was present. Facial nerve function was intact. Microbiological swabs were taken for culture but due to severe pain only limited aural toilet was performed. The canal was packed with glycerine and icthammol ribbon gauze. The patient was commenced on oral ciprofloxacin 500 mg twice daily and reviewed four days later. At this stage the otalgia improved and the gauze was removed from the external ear canal. This revealed granulation tissue present deep in the canal antero-inferiorly. Culture of the swab revealed *Pseudomonas aeruginosa* sensitive to ciprofloxacin. In-patient admission for intravenous ciprofloxacin and regular aural toilet was declined and so the patient was continued on oral ciprofloxacin and instructed to attend out-patients on a twice weekly basis for regular review. At initial follow-up a satisfactory response to treatment was recorded. However, after a period of non-attendance and voluntary stopping of the antibiotics, he returned eight weeks later with left-sided otalgia, profuse otorrhoea, pre- and post-auricular swellings with a post-auricular fistula. He also complained of tender swellings in the left axilla and the left groin. Examination of the ear canal and tympanic membrane were impossible due to intense pain. General examination revealed large fluctuant abscesses in the left axilla and the left groin, and so the patient was

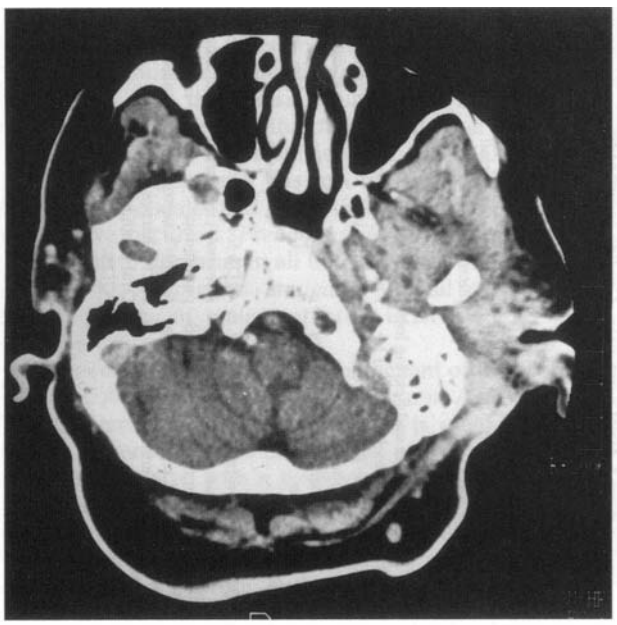

FIG. 1

Computerised tomography (CT) scan showing marked swelling of the tissues around the base of the left pinna and soft tissues overlying the entire temporal fossa.

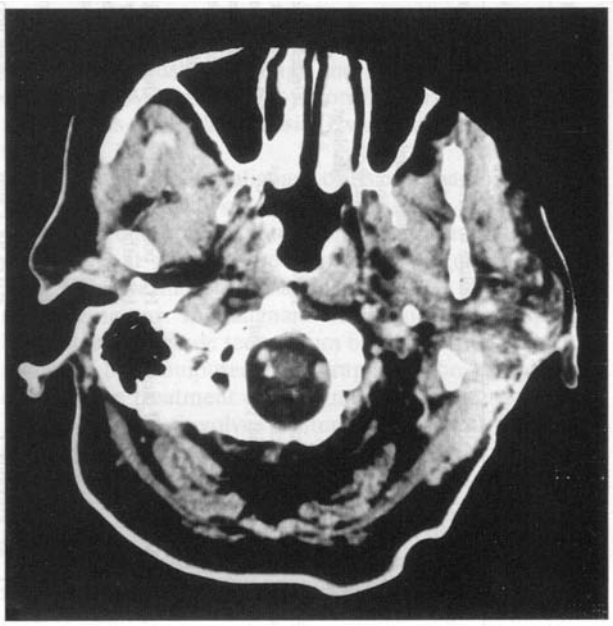

FIG. 2

Computerised tomography scan showing destruction of the left temporal bone and skull base.

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admitted. His full blood count revealed a white cell count of 1.4×10^9 (neutrophils 0.7; lymphocytes 0.5) and his CD4 count was 150/mm^3. Further culture of the discharge from the ear revealed *Pseudomonas aeruginosa* sensitive to ciprofloxacin. A computerised tomography scan revealed marked swelling of the tissues around the base of the left pinna and soft tissues overlying the entire infra temporal fossa. In addition bony destruction was present near the base of the squamous temporal bone and involving the external auditory canal and skull base (Figures 1 and 2). The patient was taken to the operating theatre for incision and drainage of the abscesses and examination of the ear under general anaesthesia. This revealed extensive granulation tissue in the ear canal which was eroded posteroinferiorly revealing exposed bone and cartilage. In one area the exposed bone was necrotic and a cavity had developed which communicated with the post-auricular skin, forming the post-auricular fistula. This area was debrided and post-operatively further anti-*Pseudomonas* antibiotics were administered. Initially there was an overall improvement in the clinical condition. However, after one month of treatment in hospital the patient developed a *Pseudomonas* septicaemia, possibly secondary to a brain abscess. In accordance with the patient's wishes further active treatment including a proposed repeat CT scan of the brain was not performed, and the patient died soon after.

Discussion

The first case of a progressive osteomyelitis of the temporal bone was described by Meltzer and Keleman in 1959. However the term malignant otitis externa was actually coined by Chandler in 1968 but debate still exists today as to the most appropriate terminology. Other suggested descriptions include 'necrotising' and 'invasive' otitis externa. The infection is almost exclusively caused by *Pseudomonas aeruginosa*, although other pathogens have been reported including *Staphylococcus aureus*, (Bayardelle *et al.*, 1982) *Proteus mirabilis* (Coser *et al.*, 1980) and *Apergillus fumigatus.* (Cunningham *et al.*, 1988). *Pseudomonas aeruginosa* is often considered to be an opportunistic pathogen as it seldom causes infection in healthy patients. Conditions predisposing to *Pseudomonas* infections include diabetes mellitus, intravenous drug use, cystic fibrosis, burns, chronic ambulatory peritoneal dialysis, neutropenia, corticosteroid administration and other forms of immunosuppression including AIDS (Kielhofner *et al.*, 1992). Infection usually commences after disruption of a natural body barrier, for example with in-dwelling intra-vascular cannulae, urinary catheters and endotracheal tubes. *Pseudomonas* infection occurs especially after recent antibiotic use which affects the normal bacterial flora. For these reasons, many *Pseudomonas* infections are hospital-acquired, although recent studies have indicated a higher rate of community-acquired infection in AIDS patients (Mendelson *et al.*, 1994). *Pseudomonas aeruginosa* has a predilection for a moist environment, which facilitates growth of the organism prior to causing invasive disease.

Malignant otitis externa develops after an initial dermatitis of the external ear canal, which compromises the natural barrier of the skin. *Pseudomonas aeruginosa* may colonise the moist environment of the external ear canal, especially if previous systemic or local antibiotic therapy has been administered. Patients with AIDS may be on many different chemotherapeutic agents which alter the local microbiological flora. In particular, co-trimoxazole is administered for prophylaxis of *Pneumocystis carinii* pneumonia. This antibiotic can lead to a 50 per cent decrease in the frequency of serious bacterial infections (Hardy *et al.*, 1992). However co-trimoxazole may predispose to *Pseudomonas* infections as it is not active against *Pseudomonas aeruginosa* (Dropulic *et al.*, 1995). Recent ear syringing also appears to be an aetiological factor and it is possible that the jet of water may damage the skin of the external ear canal or the reservoir of the apparatus is already colonized by *Pseudomonas* species. After the natural skin barrier has been broken down and the external ear canal has been colonized with *Pseudomonas*, external otitis may progress to the more invasive disease process, particularly if the host is immunosuppressed.

In HIV and AIDS, there is a defect of cells bearing the CD4 marker, which is the cellular receptor for the virus and this leads to CD4 lymphopenia. There are also quantitative and qualitative abnormalities in neutrophils and monocytes/macrophages. The defect in CD4 cells leads to depression of cell-mediated immunity. In a study by Dropulic *et al.* 1995, a majority of cases with *Pseudomonas aeruginosa* infections had a CD4 count of <100 cells/mm^3. The low CD4 count may lead to impaired humoral immunity with loss of modulation of the antibody response. The protective role of humoral immunity against *Pseudomonas* infections is due to antibody to exotoxin A and to type-specific polysaccharide, the levels of which correlate with patient survival in *Pseudomonas aeruginosa* septicaemia (Pennington, 1990). However, in HIV and AIDS there may also be an additional primary humoral defect (Lane *et al.*, 1983) and further specific involvement of cell-mediated immunity in *Pseudomonas* infections (Pier and Markham; 1982; Markham *et al.*, 1985; Powderly *et al.*, 1988; Markham *et al.*, 1991). The role of neutropenia in the development of *Pseudomonas* infections is well documented in non-AIDS patients (Bodey *et al.*, 1985). In HIV and AIDS neutropenia may be a less significant factor. In a study by Mendelson *et al.* 1994, only seven of 26 AIDS patients with *Pseudomonas* bacteraemia had a neutrophil count $<1.0 \times 10^3$/mm^3. However, both quantitative and, perhaps more importantly, qualitative defects involving impaired chemotaxis and degranulation of neutrophils have been noted in AIDS patients (Lane *et al.*, 1983; Ellis *et al.*, 1988; Schrager, 1988). Factors contributing to the neutropenia include the effect of the HIV infection itself, administration of zidovudine, toxic effects of other drugs and bone marrow suppression secondary to opportunistic infections and neoplasia (Kielhofner *et al.*, 1992). Another factor in the susceptibility of HIV and AIDS patients to *Pseudomonas* infections is the effect of concurrent infection with cytomegalovirus (CMV). CMV itself may have immunosuppressive effects and enhance the ability of *Pseudomonas aeruginosa* to cause disease (Hamilton and Overall, 1978).

The immunosuppressive effects described may predispose patients with HIV and AIDS to the invasive osteomyelitis of malignant otitis externa. This must be treated aggressively to prevent *Pseudomonas* septicaemia occurring. Several reports have suggested the need for combination therapy in severe *Pseudomonas* infection. Mendelson *et al.* 1994, in a study of 27 episodes of *Pseudomonas* bacteraemia in AIDS patients, found that in an analysis of the effect of the number of agents used in the total intravenous course of treatment, mortality was 44.4 per cent for one agent and 26.7 per cent for two agents (p = 0.036). Thus the combination of two anti-microbial agents was found to be significantly more beneficial in treating *Pseudomonas* infection. In a similar series of cases reported by Hilf *et al.*, 1989, monotherapy was associated with a mortality of 47 per cent and combination therapy with a mortality of 27 per cent. Nelson *et al.* 1991, suggest that in seriously ill patients with *Pseudomonas* septicaemia,

particularly those with a CD4 count of <50/mm^3, treatment using two drugs with anti-*Pseudomonas* effects such as ciprofloxacin and ceftazidime should be considered as it has been shown that dual therapy results in a significantly lower mortality. For this reason we now suggest using combination anti-Pseudomonas therapy in the treatment of malignant otitis externa in AIDS patients, although a prospective trial is needed for definitive advice.

Conclusion

Factors implicated in malignant otitis externa include pre-existing dermatitis of the external ear canal, concurrent medication with antibiotics, recent ear syringing and immunosuppressive defects. Patients with HIV and AIDS are at risk because many take multiple chemotherapeutic medications including co-trimoxazole and have defects in both cell-mediated and humoral immunity. If a patient with HIV or AIDS presents with a history of intense otalgia and purulent otorrhoea, the diagnosis of malignant otitis externa should be considered and appropriate investigations undertaken. Treatment should include combination anti-*Pseudomonas* antibiotic therapy such as ciprofloxacin and ceftazidime.

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