

# Human immunodeficiency virus and invasive external otitis - A case report

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## Summary

Acute invasive external otitis is an uncommon life-threatening infection of the external auditory canal (EAC), most often affecting the elderly diabetic patients. Although few reports have been made in HIV-positive/Aids patients among the caucasians.

We present here a 25 year old nursing mother with a month history of fever, persistent otalgia with acutely inflamed EAC, gross facial cellulitis, mastoid abscess and facial paresis, following a minor left ear trauma with a matchstick. This unusual course of ear infection in an otherwise healthy young adult prompts a search for an immunodepressing factor which was confirmed to be Human Immunodeficiency Virus (HIV).

This article highlights the clinical peculiarities and the management of invasive external otitis in an HIV-positive patient.

**Keywords:** Human immunodeficiency virus, Invasive external otitis, Wound sepsis.

## Résumé

Otitis externe invasive aigüe est une infection du conduit auditif externe (EAC), qui menace la vie mais qui n'est pas fréquente, qui le plus souvent atteint des vieux patients diabétiques. Quoiqu'on a recensé peu des rapports en ce qui concerne des patients atteints de VIH positif/SIDA parmi des caucasiens.

Ici, nous présentons un cas d'une mère qui allaite âgée de 25 ans atteinte de la fièvre pendant un mois, otalgie incessante avec un EAC enflammé très intense, la cellulite faciale brute, la mastoïdite abcès et paresthésie faciale, suite à un traumatisme inférieur dans l'oreille gauche causé par une allumette. Cette cause peu commune d'infection d'oreille chez un jeune adulte qui était autrement en bonne santé a provoqué la recherche d'un facteur immuno déprimant qu'on a confirmé d'être virus de l'immunodéficience humaine (VIH). Cet article met en relief (ces deux cas), les particularités cliniques et la prise en charge d'otite externe invasive chez un patient atteint du VIH positif.

## Introduction

Invasive external otitis is a severe infection of the external ear canal which may spread to the mastoid and the petrous part of the temporal bone and the soft tissues of the skull base in an immunosuppressed individual<sup>1-3</sup>. It is also referred to as malignant or necrotising otitis externa because of the rapid progression of the disease<sup>2-4</sup>. It is often found in the elderly diabetic patients and other immunosuppressive conditions such as chronic renal failure, malnutrition and patients on prolonged steroid therapy among others<sup>3-7</sup>. A few studies have reported cases of invasive external otitis in HIV positive/Aids patients<sup>8-10</sup>.

The aetiological agent is usually *Pseudomonas aeruginosa*, though in the HIV/AIDS patient *Aspergillus fumigatus*, *Proteus*

*mirabilis*, *Streptococcus epidermidis*, *Candida albicans* were reported<sup>6-9</sup>.

The common clinical presentation include otalgia, purulent otorrhoea and granulation tissues in the external auditory canal 2-5. The diagnosis is usually made by a high index of clinical suspicion though accuracy of the diagnosis and the monitoring of the response to treatment is aided by the use of Computerised Tomography (CT) Scanning, Magnetic Resonance Imaging (MRI) and Gallium-67 Single-photon Emission Tomography (SPET)<sup>3-8</sup>.

This case report is prompted by the peculiar presentation of the disease in an HIV positive individual and highlights the problems in the management of the patient.

## Case report

E. M. is a 25 year old trader who presented to the Otorhinolaryngology clinic of the hospital, on referral from a General Hospital with a 3-week history of accidental left ear trauma with a matchstick while pricking the ear. This is followed by persistent left otalgia, tinnitus, headache, fever, chills and rigors which was treated with the common analgesics and antipyretics following which she noticed slight improvement. About one week to presentation, there was associated blood stained, purulent otorrhoea and hearing loss.

The examination of the patient showed that she was acutely ill, febrile with left facial swelling involving the left lower eyelid, zygomatic and the left preauricular area. The left ear appeared prominent with obliteration of the postauricular sulcus and diffuse hyperaemia, warmth and tenderness in the mastoid area with a left facial nerve (lower motor neurone) palsy.



Fig. 1 Lateral view of the photograph of the face at presentation showing grossly oedematous and prominent left auricle, stenosed external auditory meatus and facial swelling and paresis.

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Fig. 2 Plain radiograph of the left mastoid showing cellular mastoid with multiple pus-filled cavities in the mastoid antrum and opacity in the external auditory meatus resulting in stenosis.

sis (Legend 1).

The left external auditory meatus was grossly oedematous and stenotic with copious, creamy foul smelling and cheesy secretion. The submandibular, postauricular and the upper group of the deep cervical lymph nodes were palpable, (0.5–1 cm in diameter) discrete, mobile and tender. The right ear, nose and the throat were grossly normal.

The diagnosis of left invasive external otitis was made to



Fig. 3 Lateral view of the photograph of the face after surgery and ear dressing showing scarring of the post-auricular incision wound and resolution of the oedema of the left auricle and the facial paresis.

keep in view an associated immunosuppression. The patient was admitted for full sepsis work-up (including Retroviral Screening) and Emergency Exploratory Mastoidectomy. The plain radiograph of the mastoid (Townes and Owens Views) showed a cellular left mastoid with multiple pus-filled cavities

but the tegmen tympani was intact. The right mastoid had a normal Honey-Comb appearance (Legend 2.)

There was mild anaemia (hematocrit was 29%) and elevated Erythrocyte Sedimentation Rate (ESR) 105mm/hr (normal is 0–10mm/hr using Westergren method).

There was gross reduction of the Immunoglobulin (Ig):

IgG = 900mg/dl (normal = 1272 – 2713

ig M = 14mg/dl (normal = 20 – 405)

Ig A = 12mg/dl (normal = 0-430).

The patient had a modified left radical mastoidectomy (Canal wall down) with removal of pus and necrotic bone chips via a postauricular incision. Osteotomy of the posterior wall of the external auditory canal (EAC) was also done to create a communication between the EAC and the mastoidectomy cavity (Canal wall down procedure). This surgical option was offered because the infection has spread to the preauricular soft tissues and to create a drainage for mastoid antrum.

The intraoperative findings included left mastoid air cells replaced by multiple cavities containing purulent, cheesy materials; dead bone chips and the antrum about 2cm deep containing necrotic tissues. The needle aspiration of the preauricular area revealed nothing (i.e. no material).

Postoperatively, the patient was continued on high-dose parenteral antibiotics: clavularic acid-potentiated Amoxycillin and Metronidazole which had been commenced preoperatively, and analgesics. The patient had the antibiotics for 6 weeks. The retroviral screening result confirmed that the patient was HIV positive by the Western Blot technique.

Postoperatively, she developed postauricular wound sepsis with breakdown of the inferior aspect of the incision wound and persistently discharging mastoidectomy cavity. However, the patient was stable with remission of the fever, the left facial cellulitis and the facial paresis. The culture of the preoperative left ear swab and the postauricular wound swab yielded heavy growth of *staphylococcus aureus* sensitive to Cefuroxime, Ceftriaxone and Cephalexine and resistant to Penicillin, Ampicillin, Erythromycin and Chloramphenicol.

The postauricular wound sepsis was treated conservatively. The left external auditory canal dressing was commenced on the 4th postoperative day using otomed® and postauricular wound dressing using Honey. She was discharged on the 8th postoperative day to the clinic to continue this treatment.

There was complete healing of the postauricular wound and a dry mastoidectomy cavity after about 3 weeks of the dressing (Legend 3).

## Discussion

The occurrence of a widespread infection of the external ear with the involvement of the mastoid and the parotid area following a common mild ear trauma as presented in this patient is an uncommon finding in this era of antimicrobial medication<sup>10</sup>. This thus prompts a clinical suspicion of a severe invasive ear infection and to search for the immunosuppressing factor which was confirmed to be HIV.

Severe invasive external otitis is an uncommon condition in HIV subjects though it has been reported by others<sup>7-9</sup>.

The typical patient is an elderly diabetic with persistent aural granulation<sup>2-3</sup>, as opposed to this patient who is young and presented with copious purulent secretion without an aural granulation in the EAC. This is similar to the finding of Munos *et al*<sup>7</sup> whose patient presented in this peculiar way like our patient. This presentation is perhaps due to the fact that immunosuppression is more severe in the HIV/AIDS subjects

as shown by the gross reduction of the immunoglobulins in this patient.

In contrast to the typical case which is most commonly due to *Pseudomonas aeruginosa*<sup>2,6</sup>, the aetiologic agent in our patient is *staphylococcus aureus*. This was also found by Yu et al<sup>4</sup> in the cases reported in 4 non-HIV patients. Although *staphylococcus aureus* has not been reported in the HIV patients in this regard. Complete surgical excision as in the management of this patient is advocated by most authors combined with the correction of the predisposing factor where it is possible though Hannaya et al reported success with prolonged conservative treatment<sup>4,8,10</sup>.

In conclusion, with the increasing scourge of HIV, this otherwise rare clinical condition may become common. The practitioners should be sensitized by a high index of clinical suspicion followed by prompt institution of the management. This will help in reducing the mortality of the condition and curbing the spread of Aids in surgical practice.

#### References

1. Amorosa L, Modugno GC and Pirodda A. Malignant External Otitis: Review and personal experience. Acta Oto laryngol. Suppl. 1996; Suppl 521: 3 – 16.
2. Rubin J and Yu VL: Malignant External Otitis: Insight into Pathogenesis, Clinical Manifestations, Diagnosis and Therapy. Am J Med 1998; 85: 391 – 398.
3. Midwinter KI, Gill KS, Spencer JA and Fraser ID. Osteomyelitis of the temporomandibular joint in patients with malignant otitis externa. JLO. 1999, 113(5): 451 – 453.
4. Yu LH, Shu CH, Tu TY, Shiao AS and Lien CF. Malignant otitis externa. Chinese Med J 1999; 62 (6): 362 – 368.
5. Stokkei MP, Take RP, Van Eck-Smir BL and Baatenburg de Jong RJ. The value of gallium-67 single-photon emission tomography in the clinical management of malignant otitis externa; European J Clin Med 1997; 24(11): 1429 – 1432.
6. Garty I, Rosen G and Holdstein Y. The radionuclide diagnosis, evaluation and follow-up of malignant external otitis (MEO); JLO 1985; 99: 109 – 115.
7. Munos A and Martinez-Chamorro E. Necrotising external otitis caused by *Apergillus fumigatus*: computed tomography and high resolution magnetic resonance imaging an AIDs patient. JLO 1998; 112(1): 98 – 102.
8. Hern JD, Almeyda J, Thomas DM, Main J and Patel KS. Malignant otitis externa in HIV and Aids: JLO 1996; 110(8): 770 – 775.
9. Ress BD, Luntz M, Telischi FF, Balkany TJ and Whiteman ML. Necrotising external otitis in patients with Aids. laryngoscope. 1997; 107 (4): 456 – 460.
10. Hannanya S and Horowitz Y. Gradenigo syndrome and cavernous sinus thrombosis in fusobacterial acute otitis media. harefu. 1997; 133 (7–8): 284 – 286.