



CLINICAL IMAGES

Malignant otitis externa with ecthyma gangrenosum and pneumonia in an infant

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Our patient, a previously healthy 5-month-old infant, presented with malignant otitis externa, ecthyma gangrenosum and pneumonia, but without bacteraemia.

Background

Necrotising (malignant) external otitis (MEO), an infection involving the temporal and adjacent bones, is a rare complication of external otitis.¹⁻³ It is characterised by unremitting pain and purulent discharge, and tends to invade cartilage, bone, nerve and adjacent soft tissues. It primarily affects elderly diabetics. The causative agent is almost uniformly *Pseudomonas aeruginosa* and may progress to involve the base of the skull, with multiple cranial nerve palsies and meningitis, leading to death in 30 - 80% of cases.⁴ Blood cultures can be sterile.⁵ Ecthyma gangrenosum (EG) is a rare and invasive cutaneous infection caused by *Pseudomonas* in the majority of cases.⁶⁻⁸ Pseudomonal pneumonia typically occurs in a patient who is immunocompromised; it is the most common cause of hospital-acquired pneumonia, sometimes in the absence of bacteraemia.^{9,10}

Case report

An infant who is immunocompromised can develop all three of these conditions, as in our patient, a 5-month-old previously healthy infant, who developed transient neutropenia and presented with these manifestations of *P. aeruginosa* infection and without bacteraemia. His ear commenced a slight, yellowish discharge which became profuse, greenish and mixed with blood. There was blackening and ulceration of the skin of the right ear (Fig. 1) and facial deviation; one black spot over the neck was followed by two other spots on the scalp and



Fig. 1. Malignant otitis externa with ecthyma gangrenosum patch over scalp (arrow).

forehead which progressed. The infant had a cough, difficulty in breathing and refused to feed. He was well-nourished, pale, restless and irritable. His heart rate was 156, respiratory rate 60, perfusion <3 seconds, temperature 102°F, and SpO₂ 89% without oxygen. There was facial weakness and deviation of the mouth to the left on crying (Fig. 2). There was intercostal and subcostal recession, accessory muscles were working, and bilateral decreased air entry and bilateral crepitus were present.

There was ulceration, necrosis and blackening of the right external auditory canal and part of the pinna (Fig. 1). The external auditory canal was filled with reddish soft tissue and greenish pus along with three blackish eschars over the scalp (Fig. 1), neck and forehead (Fig. 2).

Haemoglobin was 10 gm/dl, neutrophils 8%, absolute neutrophil count $0.5 \times 10^9/l$; ESR 110 mm/h; HIV ELISA and VDRL were negative; and serum immunoglobulin levels were normal.

Pus from the external auditory canal revealed *P. aeruginosa* sensitive to ceftazidime, amikacin and piperacillin and resistant to cefeprozone, ceftriaxone and amoxicillin. Blood cultures on admission 12 hours apart revealed no growth. The eschar revealed Gram-negative rods and on culture revealed *Pseudomonas* growth. Histology of the mass in the auditory canal showed granulation tissue. Chest X-ray showed bilateral pneumonic patches. Contrast-enhanced computed tomography (CECT) showed severe swelling of soft tissues in the right

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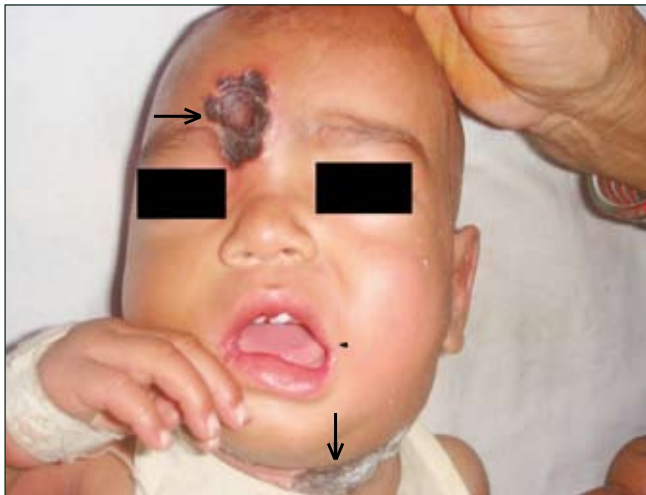


Fig. 2. Ecthyma gangrenosum patches over forehead and neck (arrows) along with right facial palsy.

external canal with a soft-tissue mass in the middle ear and partial erosion of the posterior wall of the bony canal.

The patient responded well to treatment with piperacillin and amikacin, local debridement of EG lesions and necrotic lesions of the ear, and regular dressings. A deformed right ear with loss of tympanic membrane and ossicles and partial facial paralysis remained.

Discussion

Few cases of MEO have been described in the paediatric age group and in infants.¹¹ We have not found another case in the literature with this combination of MEO with EG and pneumonia.

Paediatric case reports stress anaemia, poor general condition, and concurrent systemic disease as predisposing factors. All paediatric patients with MEO are

immunocompromised, which permits this opportunistic infection to be progressive and virulent. The most frequently cultured pathogen is *P. aeruginosa*; others include *Staphylococcus epidermidis*, Gram-negative bacteria, and fungi. Diagnosis requires culture of ear secretions and pathological examination of granulation tissue from the infection site to exclude malignancy. The characteristic lesions of EG are haemorrhagic pustules or infarcted-appearing areas with surrounding erythema that evolve into necrotic ulcers surrounded by erythematous halo. EG may appear in any location on the body; however, it predominantly affects the anogenital and axillary areas. Our patient had lesions over the scalp, forehead, and neck.

Patients at high risk for EG include those with malignancies, hypogammaglobulinaemia, patients on steroid therapy and those who are immunodeficient. Our patient also had pneumonia which, although not proved to be of pseudomonal origin, responded to antipseudomonal antibiotics.

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