CASE STUDY

CANCRUM ORIS (NOMA) IN A MALNOURISHED HIV-POSITIVE CHILD FROM RURAL KWAZULU-NATAL

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Cancrum oris (noma – derived from the Greek *nomein*, 'to devour') is an infectious disease with a fulminating course that destroys the oro-facial tissues and other neighbouring structures.[1] Although cancrum oris can occur at any age, it is most commonly in malnourished children between the ages of 1 and 5 years whose general health has been further weakened by some infectious disease, usually measles but also tuberculosis, gastro-enteritis, typhoid, whooping cough, or malignant disease such as leukaemia. The possible relevance to HIV has not been fully investigated. This report details a case presenting to East Griqualand and Usher Memorial Hospital, Kokstad, KwaZulu-Natal.

CASE REPORT

A female toddler aged 2 years and 5 months from a poor rural area in the Eastern Cape, presented at East Griqualand and Usher Memorial Hospital in June 2002 for treatment of a lesion on the lower lip affecting the mandibular gingivae and bone associated with the lower incisors (Fig. 1). The mother gave a history of a small ulcer which began on the interdental papilla of the lower incisors and in the course of 6 weeks had spread causing huge tissue destruction.

The child had been born at term following a normal pregnancy and apart from a single admission for 1 week had been relatively well. Physical examination confirmed features of typical cancrum oris with severe destruction of superficial and deep tissues and accompanying disfigurement of the face. The attending physician prescribed augmentin, gentamycin, metronidazole and folate.

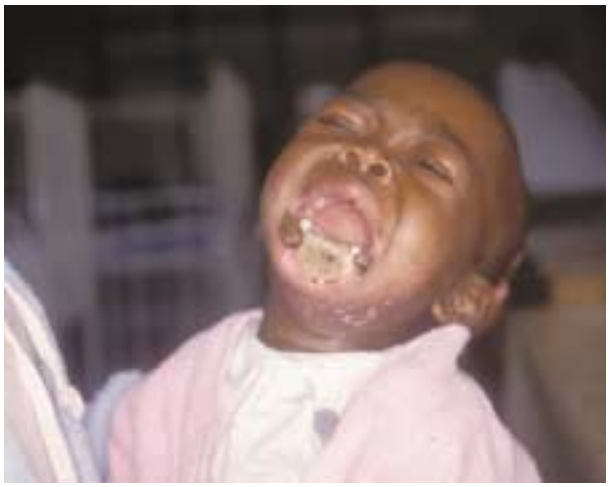

Fig. 1. Typical cancrum oris with severe destruction of superficial and deep tissues and accompanying disfigurement.

The dentist and oral hygienist gave oral hygiene instruction, provided a mouth care pack and irrigated the area using hydrogen peroxide 20 volume 5 ml in 250 ml warm water. The child was able to eat and drink well and appeared to be in very little pain. She was transferred to the regional referral centre for further management.

On admission the patient was afebrile and had generalised pallor, hypotonia and wasting. Her weight was 7.9 kg, 60% of her expected weight for age. The left half of the lower lip was absent and the gum covered by slough. Her skin was clear, she had no lymphadenopathy or hepatosplenomegaly, and the remaining systems were all normal.

Apart from a normochromic, normocytic anaemia the full blood count and urea and electrolyte levels were normal.

Fig. 2. The second case of noma, showing typical severe necrosis, rapid dissemination and tissue infiltration.

The serum total protein level was 75 g/l and the albumin level 26 g/l. Enzyme-linked immunosorbent assay (ELISA) for HIV was positive.

Nutritional support was started, treatment with augmentin, metronidazole and oral toilet continued, and fluconazole, topical anaesthetic and acyclovir added.

Despite an improvement in the patient's nutritional status and general wellbeing over the next month, during which her weight increased to 9.7 kg, the local lesion deteriorated with further involvement of the mandible leading to a fracture of the left ramus.

DISCUSSION

The early detection of this infection is of paramount importance. If the lesion is detected at the gingival stage, local disinfection, appropriate antibiotics and nutritional rehabilitation can prevent progression. Clinical diagnosis is imperative as laboratory analysis is inconclusive and early treatment and support is the only option for reducing mortality and morbidity rates.

The link with HIV needs to be explored.

The incorporation of oral examination and mouth care needs to be urgently revisited by the medical profession as multiple intra-oral lesions have a particular association with HIV and can be highly diagnostic. In their early stages they can be relatively easily managed.

Noma is a public health scourge of economically disadvantaged children particularly in sub-Saharan Africa.[2] The World Health Organisation (WHO) estimates that an annual total of 500 000 cases of noma occur worldwide, most in a belt from Senegal to Ethiopia, where the mortality rate associated with the condition is close to 90%.

Poverty is the most important risk factor for noma, with chronic malnutrition, very poor environmental sanitation, poor oral health, unsafe water supply, close residential proximity to livestock and increased exposure to viral and bacterial infections the major predisposing factors.

Recently a few atypical cases have been reported in adults, in whom the primary risk factor appears to have been immune deficiency.[3] This raises concern over a possible causal relationship between HIV infection and noma, as in the case reported above.

The global distribution pattern of noma cases shows a striking similarity to the worldwide distribution of malnutrition, particularly the vitamin A deficiency in children under 5 years of age, as compiled by the WHO Programme on Nutrition.[2] Perhaps of relevance is the reported observation that malnourished children with compromised vitamin A status are prone to develop deep erosive ulcers of the oral mucosa and eyes following measles. Emslie[4] has proposed the possibility that primary herpes simplex infection may be an initiating factor in noma and acute necrotising ulcerative gingivitis (ANUG). ANUG is generally considered a risk factor for noma.[5] Published reports indicate an escalation in the incidence of ANUG in poor African countries and this is independent of the current AIDS pandemic on the continent.

Since preparing this article a second case of noma has been identified at St Apollinaris Hospital, Centocow (Fig. 2). The child was transferred to the regional referral centre, but lived for only a few days.

In conclusion, these cases must alert us to the possibility of an increase in noma, for which we all need to be prepared.

REFERENCES

1. Tempest MN. Cancrum oris. *Br J Surg* 1966; **53:** 949-969.
2. Enwonwu CO, Falkler WA jun, Idigbe EO, Savage KO. Noma (cancrum oris): questions and answers. *Oral Diseases* 1999; **5:** 144-149.
3. Costini B, Larroque G, Duboscq JC, Montandon D. Noma or cancrum oris: etiopathogenic and nosologic aspects. *Med Trop* 1995; **55:** 263-273.
4. Emslie RD. Cancrum oris. *The Dental Practitioner* 1963; **13:** 481-495.
5. Falkler WA jun, Enwonwu CO, Idigbe EO. Microbiological understandings and mysteries of noma (cancrum oris). *Oral Diseases* 1999; **5:** 150-155.