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Review Article

Cancrum oris

Shad Muhammad Wazir, Sami Ullah Khan

Department of Dermatology, Postgraduate/Lady Reading Hospital, Peshawar

Abstract

Cancrum oris (noma) is an orofacial gangrene, which causes progressive and mutilating destruction of the infected tissues. The disease mainly occurs in children having malnutrition, poor oral hygiene and debilitating illness. It is well-documented in literature. In the international statistical classification of WHO, it is mentioned as necrotizing ulcerative stomatitis of children. Noma has disappeared from industrialized countries since the 20th century, but still common in third world countries especially in Africa.

Key words
Noma, cancrum oris, necotizing ulcerative stomatitis.

Introduction

Cancrum oris or noma (from the Greek nomein "to devour")[1] is a gangrenous stomatitis of the mouth, soft and hard tissues of the face especially in children between 2 to 16 years of age. Cases have been reported even up to the age of 26 years. If not treated, noma is always quickly fatal.[2]

Epidemiology

The epidemiology of noma has not changed much over the years, except that there has been a reduction in the mortality rate from 90% to about 8% to 10%, mainly because of modern antibiotics. The WHO estimates that 500,000 people are affected with 100,000 new cases each year. Noma has disappeared from the industrialized countries since the 20th century, but is common in the third world especially in Africa.[3] It is still a rare disease in Pakistan.

Address for correspondence
Dr. Shad Muhammad Wazir,
Senior Registrar,
Department of Dermatology,
PGMI/LRH, Peshawar.

Etiology

The exact cause of the disease is still not known but bad oral hygiene in children, oral nutrition, weak immune system, past history of measles, scarlet fever, typhoid, bacillary dysentery, malaria, whooping cough, tuberculosis, malignancy and HIV are predisposing factors of noma.[4,5,6] Its main cause may be bacteria, although the disease is not communicable.

It is postulated that the disease is triggered by a consortium of micro organism of which *Fusobacterium necrophorum* is a key component.[10,11] *F. necrophorum* elaborates several dermonecrotic toxic metabolites and is acquired by the impoverished children via fecal contamination, resulting from shared residential facilities with animals and very poor environmental sanitation. Other common pathogens found in the lesions of noma are *Prevotella intermedia* and *Borrelia vincentii.* Symbiotic relationship between fusiform bacilli and non-hemolytic streptococci and staphylococci has been considered a significant factor in the development of noma. *B. vincenti* and

fusiform bacilli can be cultured in most cases. Anaerobic bacteria may be present in rapidly progressing disease.[12,13,14]

Clinical features

The mucous membranes i.e. gums, lining of cheeks) become inflamed and develop an ulcer. The infection spreads from the mucous membranes to the skin thus causing necrosis of the tissues of lips and cheeks.[8] There is sore mouth with focal edema having fetid odour and taste. Foul smelling, purulent oral discharge is associated with profuse salivation, anorexia and palpable cervical lymphadenopathy. First there appears a tender, small purplish red spot on the gingiva, which quickly becomes indurated, ulcerated and then becomes necrotic with associated edema. It forms a bluish black necrotic cone-shaped mass with intra-oral base. There is rapid progression to gangrene during the next 4 to 72 hours. The involvement can be uni- or bilateral and may affect any part of the face including upper/lower jaw. It may produce extensive facial mutilation with loss of intraoral structures and functions.

Rapid, painless tissue break down continues and this gangrenous process can destroy the soft tissues and even the bone. The disease degrades the face of children within days (**Figure 1**). Noma causes sudden, rapidly progressive tissue destruction. Occasionally, genitals are involved, called noma pudenda.[7]

Systemically, the patient suffers from fever, tachycardia, tachypnea and anorexia. If untreated, the disease is rapidly fatal. In one of the study, in impoverished African children at risk for cancrum oris (noma) had

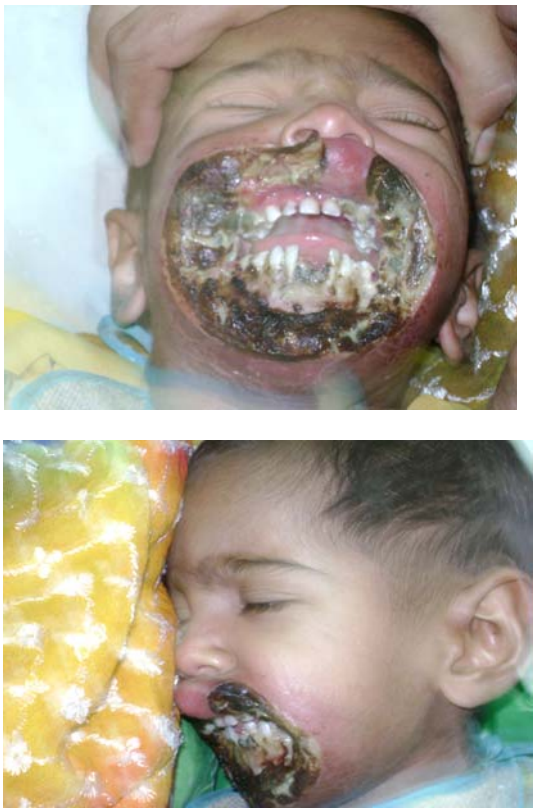

Figure 1 Extensive necrosis affecting soft tissues, bones and teeth in noma.

significantly reduced plasma concentration of zinc (<10.8 micromole/l), retinol (<1.05 micromole/l), ascorbate (<11 micromole/l), and essential amino acids, with prominently increased plasma and saliva levels of free cortisol, compared with their healthy counterparts.[9]

Management

The management of noma requires a multidisciplinary team approach.[15] In the early stages the child will need oral irrigation with hydrogen peroxide, saline and 0.2% chlorhexidine, thus helps to slough the necrotic tissue. Adequate hydration, correction of electrolytes and vitamin deficiencies with provision of sufficient nutritional support is essential, even through nasogastic tube, if necessary. As there is no

clear concensus but most authors recommend penicillin plus metronidazole to cover predominant organisms. Medication needs to be continued for at least 14 days. The preferred antibiotic is penicillin G 2.4 million U intravenously qid + metronidazole 500mg IV 8 hourly. The other alternative is ampicillin/sulbactam 3.0 g IV 6 hourly. The use of antibiotic may cause candida overgrowth, thus requires antifungal coverage with nystatin rinses 5ml q.i.d. or fluconazole 200mg orally once daily. Late stage treatment requires plastic/reconstruction surgery for correction of extensive facial mutilation. To prevent noma, measures to improve nutrition, cleanliness and sanitation and early vaccination is required.

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