CASE STUDY

Cancrum Oris (Noma): The Role of Nutrition in Management

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Abstract Cancrum oris (noma) is caused by the spirochete *Borrelia vincenti* in association with anaerobic bacteria, commonly a member of the fusobacteria. The disease occurs in deprived and undernourished individuals with poor hygiene. We present a case of clinically diagnosed cancrum oris in a 2-year old child in sub-saharan Africa that was conservatively managed by wound care and hyperalimentation. As the underlying factor and cause of mortality from noma is the immune depression of malnutrition, the role of active nutrition or hyperalimentation in the management of noma is extremely important. © 2016 Elsevier Inc. All rights reserved.

Main lesson: The role of active nutrition or hyperalimentation in the management of cancrum oris (noma) is extremely important.

Case Report

A 2-year old African girl presented with a painful swelling in the submental region of 4 days duration. This developed from an ulcer at the corner of her right upper lip that had occurred a week earlier. The swelling progressed rapidly and was associated with intermittent fever. On

examination the patient was acutely malnourished and weighed 10 kg. She was pale, pyrexia (temperature -39°C), tachycardic and tachypnoeic. Extraoral examination revealed a tender 4 cm submental swelling, which was fluctuant warm with an erythematous base. There was mild crusting of the overlying smooth and shiny skin and a central punctum (Fig. 1). There were no skin lesions involving the palms and soles suggestive of yaws. The initial presentation was suggestive of a submental abscess with a differential diagnosis of a cellulitis. Investigations showed anemia (hemoglobin level of 7.6 g/dl), a leukocytosis ($\text{WBC } 22 \times 10^9$), a raised erythrocyte sedimentation rate (ESR 35 mm/h) and her HIV serology was negative. Following incision and drainage of the abscess and parenteral amoxicillin-clavulanic acid 80 mg/kg/24 h in 2 divided doses, the ulcer rapidly extended to the submandibular region, the corner of the mouth and to the preauricular region. This was managed from the second day of admission, with daily wound debridement, irrigation with hydrogen peroxide, saline and 0.2% chlorhexidine, parenteral antibiotherapy (amoxicillin-clavulanic acid 80 mg/kg/24 h in 2 divided doses, metronidazole 7.5 mg/kg every 8 h s for 7 days, and optimized treatment of acute malnutrition with starter (F75) and catch-up feed (F100)

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Figure 1 Cancrum oris (noma).

(Table 1). The feed were locally made with the contents as shown in the table. After 1 week of treatment the evolution was remarkable and the wound healed by the end of the second week (Fig. 2).

Discussion

We describe an acutely ill 2-year old African girl with severe protein deficiency that preceded a clinically diagnosed Cancrum oris (noma). ‘Noma’ from the Greek word ‘nomien’ means to devour. Cancrum oris (noma) affects children between 2 and 16 years of age with a peak incidence at ages 1–4 years after cessation of breast feeding. The global incidence is quite high, at 140,000 new cases per year but has disappeared in the developed world. It usually occurs in children who are recovering from a debilitating illness, commonly measles and may coincide with the period of linear growth retardation in deprived children.^{1–7} It is caused by the sprochaete *Borrelia vincenti* in association with anaerobic bacteria commonly a member of the fusobacteria. Rapidly progressive

Figure 2 Wound healed by week 2 following conservative treatment.

gangrenous destruction of the inner aspect of the mouth and cheek occurs, which may continue to progress even after treatment with penicillin.^{1,8} The impaired feeding in the sick child with the oral lesion and deformity will further exacerbate the malnourished condition. The mortality rate from the complications, septicemia, severe dehydration and rarely hemorrhage is as high as 70–90% if not promptly treated, and probably reflects the underlying poor physical state of the patient.^{3–6} Malnutrition leads to dysregulated immunity with the release of cytokines. Vitamin A and zinc deficiencies are characterized by diminished cell-mediated immunity, early breakdown in the integrity of epithelial tissue and pathologic alterations of the oral mucosa. These predispose *B. vincenti* infection in association with anaerobic bacteria. The massive release of anaerobic bacterial proteolytic enzymes causes a necrotizing fasciitis.^{1,8} The etiology is interestingly similar to ‘tropical ulcer’ usually due to fusiforms and *B. vincenti* following minor trauma, especially in malnourished young people in the tropics.⁹ The differential diagnosis include spirochetal infections due to *Treponema* (syphilis, bejel, yaws and pinta) and *Borrelia* (the relapsing fevers, tropical ulcer) (Tables 1 & 2)⁹ Yaws caused by *Treponema pertenue* spreads by direct contact, usually in children where the organism again enters through damaged skin. However, dissemination of the organism leads to papular skin lesions containing treponema involving the palms and soles. The gross destruction and disfigurement particularly of the skull and facial bones occur from late yaws bony lesions.⁹ The other *Borrelia* infections are characterized by a relapsing fever with the spirochetes demonstrated microscopically in the blood during the febrile illnesses (Tables 1 & 2).

Plasma-free aminoacid profiles in noma patients were consistent with the syndrome of protein-energy malnutrition (PEM).^{1,2,6,7} Most of the children with noma showed very marked depletion of plasma retinol, zinc and ascorbate, and, hence the need for their supplementation.¹ The

Table 1 Nutrition recipe.	
Initial feeding with locally made F75 (cereal-based starter)	Catch-up feeding with locally made F100.
1000 ml water	1000 ml of water
1 egg	80 g of dried skimmed milk
25 g dried skimmed milk	60 g vegetable oil
70 g sugar	50 g sugar
125 g cereal	1 amp KCl
27 g vegetable oil	1 amp NaCl
1 amp KCl	
1 amp NaCl	

Table 2 Spirochatal infections.⁹

Disease	Organism
Syphilis	<i>Treponema pallidum</i>
Bejel (endemic non-venereal syphilis)	<i>Treponema pallidum</i> variant
Yaws	<i>Treponema pertenue</i>
Pinta	<i>Treponema carateum</i>
Leptospirosis (Weil's disease)	<i>Leptospira icterohaemorrhagiae</i>
Canicola fever	<i>Leptospira canicola</i>
Louse-borne relapsing fever	<i>Borrelia recurrentis</i>
Tick-borne relapsing fever	<i>Borrelia duttonii</i>
Cancrum oris	<i>Borrelia vincenti</i>
Lyme disease	<i>Borrelia burgdorferi</i>
Rat-bite fever	<i>Spirillum minus</i>
	<i>Streptobacillus moniliformis</i>

management of noma requires a multidisciplinary team approach. In the acute stage of cancrum oris (noma) the role of surgery is a minor one: wound care. Wound care entails topical irrigation with hydrogen peroxide, saline and 0.2% chlorhexidine to deslough the necrotic tissue. Adequate hydration, correction of electrolytes and vitamin deficiencies with provision of sufficient nutritional support is essential. Antibiotics will arrest or retard the progression into septicemia.^{1,3,7} Late stage treatment may require plastic/reconstructive surgery of the extensive facial ulceration, but this can be avoided with adequate nutrition therapy as in this case.¹⁰ In patients who survive noma, and develop a mutilated and disabled face (trismus, leakage of saliva, impaired speech), reconstructive surgery may improve their fate significantly. Treatment consists of excision of all scar

tissue, correction of the trismus and closure of the tissue defects with local, pedicled or free flaps.¹¹

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