Original Article

# Nutritional Status and Anemia in Persons With Cancrum Oris

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## Abstract

**Background:** Cancrum oris, also known as Noma, is a devastating infectious disease that destroys the soft and hard tissues of the face and peri-oral tissues. The etiology of noma is still unknown, but several risk factors have been identified such as malnutrition, poverty, poor oral and general hygiene. **Aim:** The aim of this study was to assess the nutritional status (using body mass index) and hemoglobin levels of patients managed with Cancrum oris at the NOMA Children's hospital (NCH) in Sokoto Nigeria. **Materials and Methods:** This was a 4 year' retrospective study of patients managed with NOMA at the NCH from 2016 to 2019 using the hospital records. **Results:** One hundred and ninety-five (195) patients were seen within this period. The mean BMI was 16.768 standard deviation 4.59 (Underweight) with about 49% of the total patients seen being underweight. Anemia was observed in 60% of the patients. **Conclusions:** Malnutrition features are common among noma patients, observed in 49% of noma patients in Sokoto. This is in line with several other reports. However, this study raises questions on a possible relationship of noma with anemia beyond its coexistence with malnutrition.

**Keywords:** Anemia, cancrum oris, nutritional status

## Introduction

Cancrum oris (Noma) is a severe bacterial infection characterized by progressive necrotic ulceration and destruction of the oral and perioral soft and hard tissues. It is a poorly understood disease with mortality rates as high as 90%[1,2] in its acute phase due to associated septicemia.[1,3] The disease thereafter progresses to a quiescent stage characterized by facial and dental distortion arising from scarification of healing ulcers. Noma is an aggressive and devastating disease that is associated with poverty and malnutrition and is seen predominantly in children of ages 2–6 years[3,4] in some developing and under-developed regions in Africa and Asia.[3]

The word "noma" was derived from a Greek word that means "to graze" or "to devour"[5] in a bid to describe the necrotic destruction that characterizes the disease. Noma begins as gingival ulceration and progresses to tender swelling of the cheek, fever, cervical lymphadenopathy, and fetid breath. Without prompt intervention, this condition is followed by necrosis and then rapid destruction of the gingiva, oral mucosa, skin and bones of the face,[3] and associated life-threatening septicemia.[5]

The etiology of noma is thought to be multifactorial,[6] with socioeconomic, immunological, and microbial factors being implicated. *Fusobacterium necrophorum* and *Prevotella intermedia* have been identified, though not consistently, in the pathogenesis of noma. These organisms, particularly *F. necrophorum* has been identified as a commensal in the gut of herbivores and a cause of necrotizing infections in animals, suggesting cross-contamination by livestock.[6]

Immunodeficiency forms the foundation on which noma disease develops and progresses. Therefore, the risk factors associated with a reduced immune status such as malnutrition, measles, and malaria have also been implicated in noma.[7] Poverty and malnutrition have been remarkably highlighted by several authors as a risk factor in the occurrence of Noma.[1,6,8,9] However, noma has not been reported in certain other areas with poverty and malnutrition, such as in India,[1] and this further reinforces the notion of a complex interplay of multiple factors. Other reported risk factors include poor oral and general hygiene, measles comorbidity,[2] and communal living with farm animals.

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With an improved understanding of the aetiopathogenesis of noma, more effective preventive measures can be instituted. This study aims to describe the nutritional status using the body mass index (BMI) and the hemoglobin levels of patients presenting with any stage of noma.

## Materials and Methods

### Ethical consideration

Ethical clearance was obtained from the State Health Research Ethics Committee of the Sokoto State Ministry of Health with reference number SMH/1580/V.IV.

### Study design

This was a retrospective descriptive study.

### Study population

Patients of all ages presenting with any stage of noma at the Noma Children Hospital (NCH) Sokoto.

### Selection criteria

Patients with a diagnosis of Noma presenting between October 2016 and October 2019 and with complete records.

The study was a review of the records of new patients with the diagnosis of noma that presented at the Noma Children Hospital, Sokoto, between October 2016 and October 2019. A diagnosis was made clinically based on the presence of acute necrotizing stomatitis with exposure of underlying bone, edema, or initial facial necrosis. Data collected comprised the patients' weight (kg), height (cm), age (years), and hemoglobin concentration (g/dL).

Height was measured using a measuring tape and height board for persons aged ≤5 years and >5 years, respectively. A manual floor scale was used for weighing all participants. Age estimated to the closest year was self-reported by either the patient (if ≥18 years) or by their caregiver.

BMI value was then used to assess their nutritional status by anthropometric indicator estimates proposed by the World Health Organization (WHO) and the US Centers for Diseases Control and Prevention.[10] The WHO guideline[11] for the diagnosis and assessment of the severity of anemia was used to categorize the patients as anemic or healthy [Table 1].

For children aged 2–15 years, BMI was calculated and categorized accordingly using the gender-specific WHO BMI charts. Children were classified as either underweight (BMI <5$^{th}$ percentile), normal (6$^{th}$–84$^{th}$ percentile), overweight (85$^{th}$–95$^{th}$ percentile), or obese (>95$^{th}$ percentile).[10]

For persons aged ≥16 years, weight and height were used to classify their BMI range as either underweight <18.5 kg/m$^2$, normal 18.5–25 kg/m$^2$, overweight 25–30 kg/m$^2$, or obese >30 kg/m$^2$.

### Data analysis

A descriptive analysis was done. Median and interquartile range (IQR) were reported for non-normally distributed continuous values while means and standard deviations (SD) were reported for parametric values. Data were analyzed using STATA14 and significance levels of the variables were set ($P \leq 0.05$).

## Results

A total of 195 patients were seen within this study period. There were 103 (52.8%) males and 92 (47.2%) females (M:F = 1.12:1). The age ranged from 7 months to 72 years with a median age of 10 years (IQR = 21) and modal age group of the first decade of life [Figure 1]. The BMI ranged from 5.18 to 36.22 with a mean of 16.768 and SD ± 4.59 while the hemoglobin concentration also ranged from 4.5g/dL to 16.5 g/dL with a mean of 11.28g/dl (SD ± 2.42) [Tables 1 and 2].

## Discussion

The result of this study showed no difference in gender affectation of Noma, with a M:F ratio of 1.12:1. This is comparable to the result of a study by Farley who reported a M:F ratio of 1.3:1.[8] Another study by Baratti-Mayer *et al*. reported a M:F ratio of 1.05:1.[6]

**Table 1: Hemoglobin levels to diagnose anemia (g/l)**

| Population | Non-anemia | Anemia | | |
|---|---|---|---|---|
| | | Mild | Moderate | Severe |
| Children 6-59 months of age | 110 or higher | 100-109 | 70-99 | Lower than 70 |
| Children 5-11years of age | 115 or higher | 110-114 | 80-109 | Lower than 80 |
| Children 12-14years of age | 120 or higher | 110-119 | 80-109 | Lower than 80 |
| Non-pregnant women (15years of age and above) | 120 or higher | 110-119 | 80-109 | Lower than 80 |
| Pregnant women | 110 or higher | 100-109 | 70-99 | Lower than 70 |
| Men (15years of age and above) | 130 or higher | 110-129 | 80-109 | Lower than 80 |

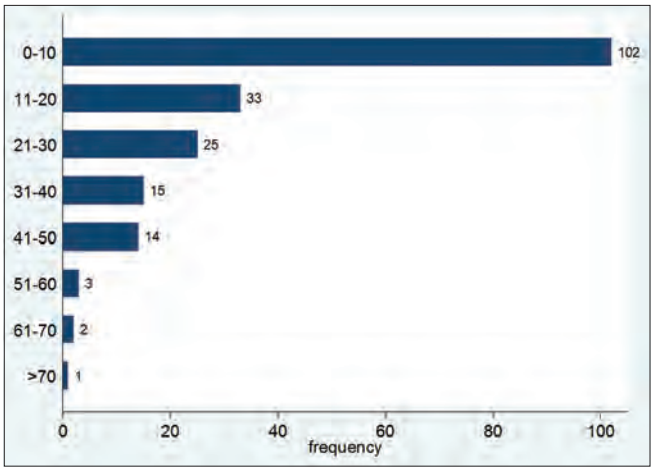


**Figure 1:** Age groups of patients with noma

The age of patients ranged from 7 months to 72 years, with peak occurrence in the first decade of life [Figure 1]. This shows that noma affected all age groups, mostly young children less than 10 years. These young children have less physiological reserve and are therefore more prone to the adverse effects of malnutrition and reduced immunity. The youngest age in this study was 7 months old in one subject. This coincides with the age some mothers commence weaning the child off breast milk. This may suggest the role of passive immunity in the prevention of noma. Passive immunity comprises of the antibodies transmitted from the mother to the child *in utero* in the third trimester, and then through the breast milk. These antibodies are capable of mounting some defense in the first months of life.[12] Waning levels of this passive immunity especially during the weaning period could herald the occurrence of infections such as noma in infants.

The incidence of noma has continued to increase in developing regions in sub-Saharan Africa. This is expected to even worsen due to the rising poverty and insecurity in the region made worse by the COVID-19-induced global austerity. Noma is regarded as an opportunistic infection, arising in the background of some degree of immunosuppression. When poverty prevails, there appears to be a synergistic relation between malnutrition, weakened immune functions, and increased susceptibility to infections.

Noma is thought to arise as a progression from necrotizing ulcerative gingivitis; to necrotizing ulcerative periodontitis and then to ulcerative stomatitis. This course of progression is still uncertain. However, noma is more commonly described as having five stages: a warning stage of gingivitis which is a nonspecific gingival inflammation in response to poor oral hygiene; acute necrotizing gingivitis characterized by painful and foul-smelling ulceration of the gums and interdental papillae; then a facial edematous stage [Figures 2 and 3] having marked facial swelling and tenderness, pyrexia and dysphagia; the gangrenous stage [Figure 4] which is characterized by sepsis, bluish-black discoloration of overlying skin, rapid and extensive necrosis and then perforation of facial skin to expose the teeth and bone. This gangrenous stage requires emergency debridement, resuscitation and parenteral broad-spectrum antibiotics. This stage is potentially lethal and patients who survive this stage go on to the quiescent stage marked by progressive scarring of the healing wound [Figure 5]. Patients then move to the sequelae stage which is an inactive phase characterized by facial disfigurement, trismus, and severe psychosocial and function affectation [Figure 6].

The management of the incipient gingivitis stage of noma includes oral hygiene measures such as scaling and polishing, use of antimicrobial mouth rinses.[3] The use of antibiotics and wound debridement is used when the lesion extends beyond the gingiva tips and margin. Medical and nutritional support is also required in the management of the different stages of the active disease. Medical management of noma includes the treatment of existing diseases such as malaria, respiratory tract infection, measles, and correction of dehydration, electrolyte imbalance and anemia.[7] High protein diet and nutrient supplements – Vitamin A, C, Bco, folic acid, zinc, and iron are given[6,12,13] either through oral or nasogastric feeding.

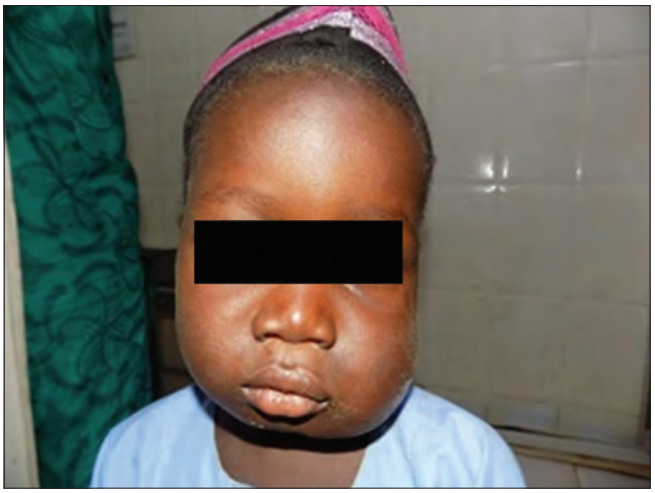

**Figure 2:** Facial edematous stage

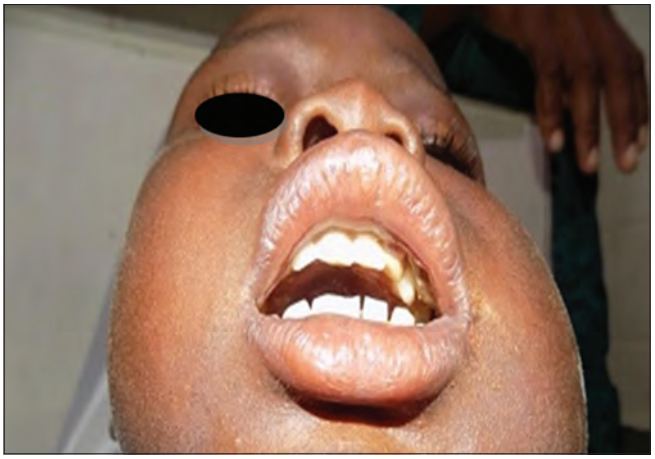

**Figure 3:** Necrotizing gingivitis

**Figure 4:** Gangrenous stage

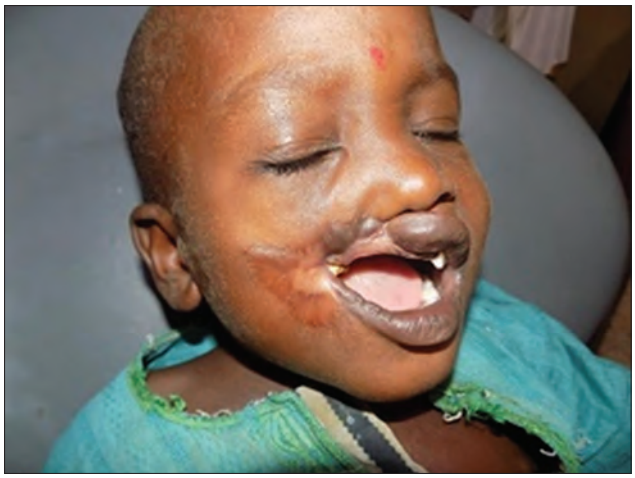

**Figure 5:** Scarification stage

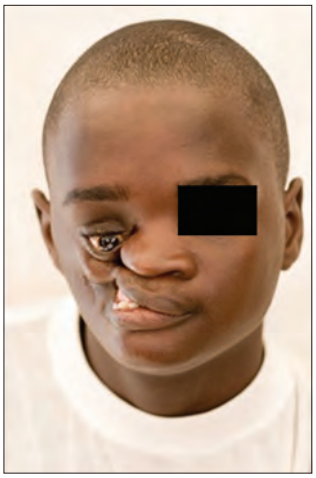

**Figure 6:** Severe facial disfigurement

BMI is an indicator of the nutritional status of an individual.[14] There are several pitfalls in using the BMI for the assessment of nutritional status due to demographic variations,[15] however, it remains one of the most widely used tools for nutritional status screening. The BMI for children aged 2–18 years has been adjusted for age and gender[16] using a percentile chart. Our results showed that 49% of the patients seen were underweight which is higher than the 2018 National Nutrition and Health Survey by the Federal Ministry of Health that reported 29.7% of children in the North-western region of Nigeria were underweight.[17] This may suggest a crucial role of malnutrition in the pathogenesis of noma. This finding is further buttressed by reports of other noma investigators.[2,5] Other reported risk factors[6,7,18] include recent respiratory or diarrhea syndrome, debilitating diseases such as measles and malaria, the number of previous pregnancies, altered oral microbiota compared with controls, and poverty-related factors such as the absence of chickens at home and overcrowded households. Baratti-Mayer *et al*.[6] postulated that frequent pregnancies often cause maternal malnutrition, resulting in an increased risk of low-birthweight babies. They implied that this resultant low birth weight was a risk factor for noma in infants.

**Table 2: Body mass index group of the study patients**

| | Frequency (%) |
|---|---|
| Underweight | 95 (48.7) |
| Normal | 80 (41) |
| Overweight | 10 (5.1) |
| Obese | 10 (5.1) |
| Total | 195 (100) |

**Table 3: Hemoglobin concentration group of the study participants**

| | Frequency (%) |
|---|---|
| Normal | 78 (40) |
| Anemic | 117 (60) |
| Total | 195 (100) |

Besides the BMI, other methods for assessment of nutritional status include[19] clinical methods – skin and hair texture and color, weight loss or inability to gain weight, bilateral pitting edema; anthropometric methods – mid-arm upper arm circumference, head circumference, skinfold thickness, and muscle mass; biochemical methods – serum levels of albumin, prealbumin, and transferrin, and more recently serum levels of insulin-like growth factor, serum cholesterol, urinary urea nitrogen, creatinine-height index have been employed; as well as the frequently overlooked neurological methods - functional capacity and muscle power (weak hand-grip strength and respiratory muscle strength).

Nutritional rehabilitation of a chronically malnourished patient should be done with caution, especially in those with diminished physiological reserves.[20] These groups of patients are a high risk for refeeding syndrome, which is a potentially life-threatening fluid and electrolyte imbalance that occurs in malnourished persons who receive rapid nutritional supplementation. Potential fatality is due to resultant multi-organ dysfunction.[21] The hallmark biochemical feature of refeeding syndrome is hypophosphatemia and may include abnormal sodium and fluid balance; changes in glucose, protein, and fat metabolism; thiamine deficiency; hypokalemia; and hypomagnesemia.[20] Nutritional replenishment of energy should be started slowly (less than 50% of daily requirement) and should be tailored to each patient. It can then be increased to meet or exceed full needs over 4–7 days.[20] Cardiac monitoring during the feeding period as well as supplementation of potassium, phosphate, calcium, magnesium, and vitamins have been recommended.[22]

Anemia refers to a low level of red blood cells or low level of hemoglobin in the red blood cells and reduced oxygen delivery to tissues, thereby resulting in an inability to meet the body's physiologic needs.[10] Demographic variations of normal levels of hemoglobin have been seen, with variations in age, gender, tribe, and geographical regions noted.[22]

Anemia is a common blood disorder, and it results from many conditions that interfere with the production of healthy red blood cells or with an increased rate of breakdown of the red blood cells.

In this study, we found that 60% of the patients were anemic at presentation [Table 3]. This is classified as a severe public health issue according to the WHO[11] and is significantly higher than a population-based study from this region[23] which reported 40.3%. In what way noma is related to the finding of anemia is yet to be described as there is scanty literature relating them.

The cause-effect role of anemia in the pathogenesis of noma has not been fully understood. The anemia seen in patients with noma could be a result of severe malnutrition, in which case there is a shortage of essential minerals and nutrients required for the production of red blood cells. Anemia could also be a result of an increased breakdown of red blood cells seen during sepsis, which is a feature of the acute gangrenous stage. On the other hand, in what may be a more complex interplay, the reduction in red blood cells and oxygen delivery featured in anemia could have a role in the initiation of noma. Reduced oxygen delivery to perioral tissues may play a role in the characteristic necrosis seen in the early stages of noma. If the anemia is linked to the aetiopathogenesis of noma, it could suggest the role of reduced oxygen delivery and tissue hypoxia in the formation of the initial necrotic ulcers in addition to the microbial factors. The face and perioral tissues are well vascularized and richly perfused in health. However, microbial factors and the inflammatory cascade result in small vessel thrombosis and tissue ischemia. In healthy patients, a compensatory increased perfusion during healing limits tissue destruction. However, in a pre-existing anemic state, in which there is an inability to meet the body's physiological needs, this compensatory flow is compromised and consequently, the rapid necrotizing destruction of oral and perioral tissues is seen. The understanding of this process could open new treatment modalities in the management of early noma. For example, the use of high oxygen therapies and hemorheologic agents may help improve tissue perfusion and reduce tissue destruction in the pre-necrotic stages of noma.

The type of anemia was also not investigated in this study. Knowledge of the type of anemia seen in these patients will elucidate a possible relationship between noma and anemia.

## Conclusions

Malnutrition plays an important role in the occurrence of cancrum oris, while anemia may play a significant role in the initiation of the disease. Further studies on the relationship between anemia and noma are recommended.

### Acknowledgment

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Nil.

### Conflicts of interest

There are no conflicts of interest.

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