DOI: 10.7860/JCDR/2026/82789.22726

Review Article

Community Section

Clinical Features and Pathogenesis of Noma: A Narrative Review Highlighting the Emerging Strategies for Prevention and Management

BHAGYESH SAPKALE[1], SONALI CHOUDHARI[2]

ABSTRACT

Noma, also known as cancrum oris, is a rapidly progressive gangrenous infection that affects the oral and facial tissues of malnourished children in impoverished regions, particularly in sub-Saharan Africa. The disease typically begins as acute necrotising gingivitis and rapidly progresses to extensive tissue destruction, facial disfigurement, and high mortality if left untreated. This narrative review explores the clinical progression of Noma, its World Health Orgnisation (WHO) classification and simplified staging systems, the pathogenesis involving malnutrition, immunosuppression, and microbial dysbiosis, as well as the polymicrobial nature of the disease. Diagnosis of Noma is primarily clinical, although emerging microbiome-based diagnostic techniques show promise for early detection. Management requires a comprehensive approach that combines early antibiotic therapy, nutritional rehabilitation, wound care, and delayed reconstructive surgery. Preventive strategies include adequate vaccination (particularly against measles), community-based oral health education, and improvements in Water, Sanitation, and Hygiene (WASH) initiatives. Strengthening local healthcare infrastructure and establishing sustainable surgical programmes are essential for long-term control and rehabilitation of Noma. Addressing the socio-economic determinants of Noma through holistic public health efforts remains crucial for reducing its burden and achieving global eradication.

Keywords: Anaerobic bacteria, Facial disfigurement, Malnutrition, Microbiome profiling, Neglected tropical diseases

INTRODUCTION

Noma is a rapidly progressive, gangrenous infection of the mouth and face that primarily affects malnourished children living in conditions of extreme poverty [1]. It typically begins as a minor gingival ulcer but can swiftly devastate oral and facial tissues, including bone, if left untreated [1,2]. In the absence of prompt antibiotic therapy and nutritional support, Noma is associated with high mortality rates. Early intervention significantly reduces fatality and improves outcomes through reconstructive surgery and comprehensive care [3].

The earliest descriptions of Noma date back to Hippocrates and Galen, with the term cancrum oris, a Latinised misnomer, appearing in mid-17th century British medical literature [4,5]. Today, the term Noma is preferred; it is derived from the Greek word meaning "to devour," reflecting the destructive nature of the disease [4]. During the 19th century, Noma was commonly observed in Europe. However, improvements in living conditions during the 20th century led to its near disappearance in developed regions, with outbreaks largely confined to war-torn settings, such as concentration camps during World War II [6]. In contrast, the disease continues to persist in resource-limited settings, particularly within the "Noma belt" of sub-Saharan Africa, where deep-rooted poverty, recurrent famine, and inadequate sanitation create conditions conducive to its occurrence [7,8].

Symptoms of Noma: From Gingival Necrosis to Facial Disfigurement

Noma (cancrum oris) predominantly affects vulnerable, malnourished children and often follows a predisposing illness such as measles, malaria, or kwashiorkor [9]. The initial manifestation is acute necrotising gingivitis, characterised by painful, bleeding gums, ulceration of the interdental papillae, and a foul-smelling oral cavity [9,10]. As the disease progresses to the oedematous stage, pronounced facial swelling becomes evident, particularly involving the cheeks, lips, and perioral regions [11]. Additional clinical features include halitosis, excessive salivation, difficulty in eating, and systemic symptoms such as fever and lymphadenopathy [12,13].

Noma advances with alarming rapidity, with necrosis extending to soft tissues and underlying bone. This process results in sharply demarcated, greyish-black lesions that subsequently slough off, leaving deep, often cone-shaped defects that may expose bone and teeth [14,15]. Affected children may experience severe dehydration, anaemia, lymphocytopenia, hypotension, and systemic deterioration, with mortality rates remaining high in untreated cases [16]. Survivors frequently suffer from long-term sequelae, including extensive facial disfigurement that impairs speech, mastication, respiration, and social integration [1,16].

WHO Classification and Emerging Simplified Models for Noma

The WHO has outlined a six-stage clinical classification for Noma, which describes disease progression from early gingival inflammation to severe facial disfigurement [17]. The stages include: Stage 0 (simple gingivitis), Stage I (acute necrotising gingivitis), Stage II (facial oedema), Stage III (gangrene with tissue necrosis), Stage IV (healing with fibrosis and trismus), and Stage V (sequelae with long-term functional and aesthetic impairments) [17]. This structured staging system facilitates early diagnosis and timely intervention, particularly during the initial gingival and oedematous stages, thereby potentially preventing the extensive tissue destruction and long-term disabilities characteristic of advanced Noma [17].

Recent studies advocate a more clinically intuitive two-stage model for Noma: acute Noma and arrested Noma [1]. Acute Noma refers to the rapidly necrotising phase, characterised by extensive soft-tissue destruction, malnutrition, sepsis, and high mortality if left untreated [1]. Arrested Noma describes the chronic, postinfectious stage observed in survivors, characterised by scarring, trismus,

and oral and facial deformities [1]. This simplified classification reflects the functional burden of the disease and facilitates easier data collection, public health planning, and rehabilitation services, particularly in low-resource settings [1]. The clinical staging and classification of Noma are depicted in [Table/Fig-1] [1,17].

Model	Stage	Description	Key features	References
WHO Six-Stage Model	Stage 0	Simple gingivitis	Mild gum inflammation; reversible if treated early	[17]
	Stage I	Acute necrotising gingivitis	Painful gingival ulcers with foul odour, necrosis begins	[17]
	Stage II	Facial oedema	Rapid facial swelling, especially around cheeks and lips	[17]
	Stage III	Gangrene and tissue necrosis	Extensive soft-tissue destruction, bone exposure possible	[17]
	Stage IV	Healing with fibrosis and trismus	Inflammation subsides, fibrotic healing leads to jaw stiffness (trismus)	[17]
	Stage V	Sequelae: functional and aesthetic impairments	Facial disfigurement, difficulty in eating/speaking; long-term disability	[17]
Two-stage model	Acute Noma	Necrotising phase	Sudden onset, rapid progression, soft-tissue necrosis, sepsis, malnutrition, high mortality	[1]
	Arrested Noma	Postinfectious sequelae	Chronic scarring, trismus, facial deformities; requires reconstructive and rehabilitative care	[1]

[Table/Fig-1]: Clinical staging and classification of noma [1,17].

Clinical and Emerging Diagnostic Strategies for Noma

The diagnosis of Noma is primarily clinical and relies on the rapid progression of oral and facial manifestations observed in vulnerable children, who are typically malnourished and immunocompromised [18]. Noma progresses through various stages, each characterised by hallmark features like gingival ulceration, facial swelling, foul odour, and black necrotic lesions involving both soft and bony tissues [8,19]. A thorough medical history—including recent illnesses (e.g., measles, malaria), nutritional status, and immune compromise—is essential for accurate staging and differential diagnosis. This helps distinguish Noma from other conditions such as oral cancer, leprosy, or necrotising fasciitis [20,21].

Laboratory investigations may help support clinical suspicion, although no point-of-care diagnostic test currently exists [22]. In acute Noma, commonly observed laboratory findings include elevated white blood cell counts (13,500-14,500 cells/μL) ((normal: 4,000-11,000 cells/μL)), increased C-Reactive Protein (CRP) levels (107-148 mg/L) (normal: <5 mg/L), elevated erythrocyte sedimentation rate (65-90 mm/hour) (normal-0-20 mm/hour), and markedly raised procalcitonin levels (>85 ng/mL) (normal-<0.05 ng/mL) [23]. Imaging studies such as plain radiography, Computed Tomography (CT), and Magnetic Resonance Imaging (MRI) can assist in assessing the extent of soft-tissue destruction and the degree of bone involvement [22,24,25].

Emerging diagnostic approaches increasingly focus on molecular and microbiome-based techniques to complement clinical diagnosis [14]. Oral microbiota dysbiosis has been shown to be useful in identifying shifts in microbial communities, particularly increases in anaerobic bacteria such as *Fusobacterium necrophorum*, *Prevotella intermedia*, and various spirochetes, which may serve as potential biomarkers for early detection of Noma [26].

Recent studies using shotgun metagenomic sequencing have characterised the oral microbial profile of acute Noma. These studies demonstrated not only changes in microbial composition, including increased abundance of *Treponema*, *Porphyromonas*, and *Bacteroides*, but also identified novel species (e.g., *Treponema* sp. A) that were associated with disease but absent in healthy controls, along with distinct antibiotic resistance gene profiles [27]. Multiplex Polymerase Chain Reaction (PCR) panels targeting major anaerobic genera (e.g., *Fusobacterium*, *Prevotella*, *Veillonella*, *Clostridium*, and *Bacteroides*) have also been developed, allowing rapid genus-level detection of anaerobes in clinical samples, with significantly faster turnaround times than conventional culture methods [28].

These findings suggest that microbiome profiling using culture-independent techniques—such as 16S rRNA gene sequencing and shotgun metagenomic sequencing—may aid in differentiating pre-Noma stages and detecting early lesions before overt clinical signs become apparent [29,30]. Various diagnostic methods for Noma are summarised in [Table/Fig-2] [8,14,18-30].

Diagnostic aspect	Details	References
Primary mode of diagnosis	Clinical diagnosis based on rapid progression of oral and facial signs in vulnerable children (malnourished, immunocompromised).	[18]
Characteristic clinical signs	- Gingival ulceration - Facial swelling - Foul odour - Black necrotic lesions penetrating soft and bony tissues	[8,19]
Patient history	- Recent illnesses: Measles, malaria - Nutritional status - Immune compromise	[20,21]
Laboratory investigations	No point-of-care test available Common markers in Acute Noma: - WBC: 13,500-14,500 cells/μL - CRP: 107-148 mg/L - ESR: 65-90 mm/hr - Procalcitonin: >85 ng/mL	[22,23]
Imaging studies	X-ray, CT, MRI useful to assess extent of: - Tissue destruction - Bone involvement	[22,24,25]
Emerging diagnostic methods	Molecular and microbiome-based diagnostics being explored	[14,26]
Microbiome profiling	- Oral microbiota dysbiosis observed - Increase in anaerobic bacteria: • *Fusobacterium necrophorum* • *Prevotella intermedia* • Spirochetes	[26,27]
Molecular techniques	- 16S rRNA sequencing and shotgun metagenomic sequencing enable detection of microbial shifts and pre-Noma stages before overt clinical signs - Multiplex PCR panels for major anaerobic genera (*Fusobacterium*, *Prevotella*, *Veillonella*, *Clostridium*, *Bacteroides*) allow rapid genus-level detection	[27-30]

[Table/Fig-2]: Various diagnostic methods for Noma [8,14,18-30].

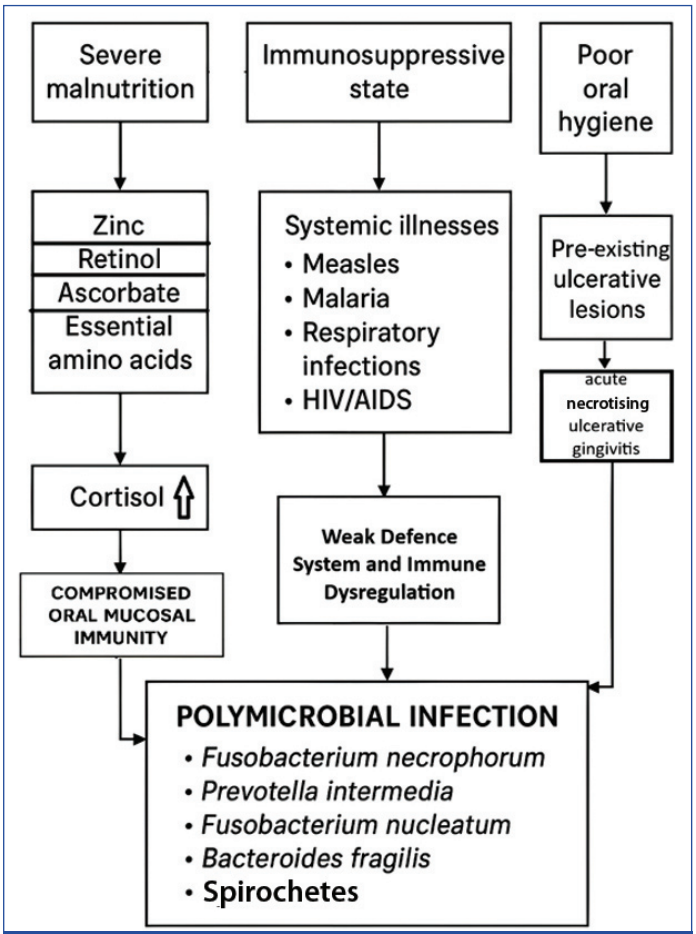
Pathogenesis of Noma: Malnutrition, Immunosuppression, and Microbial Dysbiosis

The pathogenesis of Noma involves a complex interplay of factors, such as severe malnutrition, immunosuppression, poor oral hygiene, and pre-existing ulcerative lesions like acute necrotising ulcerative gingivitis [31,32]. Nutrient deficiencies—particularly zinc, retinol, ascorbate, and essential amino acids—along with elevated cortisol levels, further compromise oral mucosal immunity, creating a permissive environment for disease progression [14,33]. Systemic illnesses such as measles, malaria, respiratory infections, and Human Immunodeficiency Viruses/Acquired Immunodeficiency Syndrome (HIV/AIDS) further weaken host defence mechanisms. HIV infection plays a particularly important role in regions such as southern Africa, where it contributes to immune dysregulation and increased susceptibility to Noma [34,35].

The microbial trigger of Noma remains incompletely understood, as no single pathogen has been definitively identified [36]. Instead, Noma

is considered a polymicrobial infection involving various anaerobic and commensal bacteria, including *Fusobacterium necrophorum*, *Prevotella intermedia*, *Fusobacterium nucleatum*, *Bacteroides fragilis*, and Spirochetes [26,36]. However, findings across studies have been inconsistent. A large case-control study conducted in Niger reported reduced proportions of *Fusobacterium*, *Capnocytophaga*, *Neisseria*, and Spirochetes, alongside an increased abundance of *Prevotella*, highlighting the role of oral microbial dysbiosis rather than a single causative organism [31,37,38].

Spirochetes and *P. intermedia* are thought to act as early triggers during the initial dysbiotic phase, whereas *F. nucleatum* may contribute to later stages through biofilm formation and tissue invasion [26,36,37]. The proposed pathogenesis of Noma is illustrated in [Table/Fig-3].



[Table/Fig-3]: Pathogenesis of Noma.
Image created by authors

Integrated Medical, Nutritional, and Surgical Care for Managing Noma

Management of Noma primarily focuses on early detection and prompt medical intervention [39]. In its acute phase, Noma often begins with necrotising gingivitis, which requires treatment involving a combination of antibiotics (typically metronidazole paired with amoxicillin or penicillin), nutritional rehabilitation, and meticulous wound care, including antiseptic rinses or debridement [40]. Timely implementation of such medical interventions can dramatically reduce the historically high mortality rates [40]. Supportive care includes fluid and electrolyte replacement, multivitamin supplementation, and management of concurrent infections (e.g., malaria or measles) to improve the patient's overall condition [41]. The WHO also emphasises adjunctive measures like improving oral hygiene, using disinfectant mouthwashes (e.g., chlorhexidine or saline), and providing nutritional support to promote healing and prevent disease progression [39,42].

Survivors of the acute phase often suffer severe sequelae, including facial disfigurement, trismus, and difficulties with speech and eating, necessitating extensive reconstructive surgery [43]. Surgical intervention, typically delayed for 6-18 months to allow disease stabilisation, involves excising fibrotic or scar tissue, correcting trismus, and repairing defects using local, pedicled, or free tissue flaps. Common options include radial forearm, scapular, anterolateral thigh, or supraclavicular flaps [43,44]. Although these procedures can restore form and function, complications remain significant, and improvements such as increased mouth opening (mean gain ~20 mm) may diminish over time without sustained follow-up [44]. Hence, establishing sustainable surgical programmes with strong local capacity and long-term postoperative care is essential to improve outcomes for Noma survivors in resource-limited settings [45].

Vaccination programmes, particularly against measles, are crucial because measles-induced immunosuppression can predispose children to Noma [46]. Additionally, community-based oral health education and training of community health workers can facilitate early detection and timely treatment of oral diseases, including Noma [25,42]. WASH (Water, Sanitation, and Hygiene) initiatives also play a key supportive role in preventing neglected tropical diseases and should be incorporated into comprehensive Noma prevention strategies [33,47]. Comprehensive approaches for the management of Noma are illustrated in [Table/Fig-4] [25,33,39-47].

Category	Key interventions	References
Early detection and acute medical management	- Early identification of necrotising gingivitis (early stage of Noma) - Prompt initiation of antibiotics (Metronidazole + Amoxicillin/Penicillin) - Nutritional rehabilitation - Wound care (antiseptic rinses, debridement)	[39,40]
Supportive care	- Fluid and electrolyte replacement - Multivitamin supplementation - Management of concurrent infections (e.g., malaria, measles)	[41]
WHO adjunctive measures	- Emphasis on oral hygiene - Use of disinfectant mouthwashes (chlorhexidine or saline) - Nutritional support	[39,42]
Surgical management	- Performed after 6-18 months for disease stabilisation - Excision of fibrotic/scar tissue - Correction of trismus - Reconstruction using local, pedicled, or free flaps (e.g., radial forearm, scapular, anterolateral thigh, supraclavicular)	[43,44]
Postoperative care	- Long-term follow-up to maintain surgical outcomes (e.g., prevent loss of mouth opening gain) - Establishment of sustainable surgical programmes in resource-limited settings	[44,45]
Vaccination programmes	- Measles vaccination to prevent immunosuppression-associated Noma	[46]
Community-based interventions	- Oral health education - Training of community health workers for early detection and treatment	[25,42]
WASH initiatives	- Water, Sanitation, and Hygiene (WASH) programmes integrated into Noma prevention strategies	[33,47]

[Table/Fig-4]: Comprehensive approaches for management of Noma [25,33,39-47].

CONCLUSION(S)

Noma remains a devastating yet preventable disease rooted in poverty, malnutrition, and poor hygiene. Early detection and intervention through medical, nutritional, and surgical strategies can dramatically reduce mortality and long-term complications. Simplified staging and emerging diagnostic techniques, such as microbiome profiling can facilitate timely diagnosis and treatment. Holistic public health measures—including vaccination, community education, and WASH initiatives—are vital for prevention. Strengthening local healthcare systems is essential for providing sustainable care to survivors and ultimately eliminating this neglected tropical disease.

REFERENCES

[1] Feller L, Lemmer J, Khammissa RAG. Is noma a neglected/overlooked tropical disease? Trans R Soc Trop Med Hyg. 2022;116(10):884-88.
[2] Srour ML, Marck K, Baratti-Mayer D. Noma: Overview of a neglected disease and human rights violation. Am J Trop Med Hyg. 2017;96(2):268-74.
[3] Ainsworth S. Noma finally recognised as a neglected tropical disease. PLoS Negl Trop Dis. 2024;18(5):e0012177.
[4] Farley E, Mehta U, Srour ML, Lenglet A. Noma (cancrum oris): A scoping literature review of a neglected disease (1843 to 2021). PLoS Negl Trop Dis. 2021;15(12):e0009844.
[5] Romero-Maroto M, Sáez-Gómez JM. Mouth ulcers: A deadly disease for children from the sixteenth to eighteenth centuries. Ir J Med Sci. 2013;182(2):297-300.
[6] Mascitti M, Barlattani A, Togni L, Sampalmieri F, Favia G, Lo Muzio L, et al. Noma: A reappraisal in Western countries - Are HIV-negative immunocompetent adult patients safe? J Biol Regul Homeost Agents. 2019;33(3):957-61.
[7] Onu JU, Aluh DO, Ononiwu CN. Psychosocial aspects of Noma (cancrum oris) in sub-Saharan Africa: A scoping review. Trop Doct. 2023;53(4):470-74.
[8] Farley E, Ariti C, Amirtharajah M, Kamu C, Oluyide B, Shoaib M, et al. Noma, a neglected disease: A viewpoint article. PLoS Negl Trop Dis. 2021;15(6):e0009437.
[9] Feller L, Khammissa RAG, Altini M, Lemmer J. Noma (cancrum oris): An unresolved global challenge. Periodontol 2000. 2019;80(1):189-99.
[10] Zwetyenga N, See LA, Szwebel J, Beuste M, Aragou M, Oeuvrard C, et al. [Noma]. Rev Stomatol Chir Maxillo-Faciale Chir Orale. 2015;116(4):261-79.
[11] Dominic C, Farley E, Elkheir N. More than 100 years of neglect: A bibliometric analysis of global research on Noma (cancrum oris). Trans R Soc Trop Med Hyg. 2022;116(5):479-86.
[12] Ashok N, Tarakji B, Darwish S, Rodrigues JC, Altamimi MA. A review on Noma: A recent update. Glob J Health Sci. 2015;8(4):53-59.
[13] The Lancet Global Health null. Noma: Neglected no more? Lancet Glob Health. 2024;12(2):e170.
[14] Gezimu W, Demeke A, Duguma A. Noma - A neglected disease of malnutrition and poor oral hygiene: A mini-review. SAGE Open Med. 2022;10:20503121221098110.
[15] Khammissa RAG, Lemmer J, Feller L. Noma staging: A review. Trop Med Health. 2022;50(1):40.
[16] Galli A, Brugger C, Fürst T, Monnier N, Winkler MS, Steinmann P. Prevalence, incidence, and reported global distribution of noma: A systematic literature review. Lancet Infect Dis. 2022;22(8):e221-e30.
[17] WHO. Information Brochure for Early Detection and Management of Noma. Regional Office for Africa [Internet]. 2025. [cited 2025 Oct 16]. Available from: https://www.afro.who.int/publications/information-brochure-early-detection-and-management-noma.
[18] Tonna JE, Lewin MR, Mensh B. A case and review of noma. PLoS Negl Trop Dis. 2010;4(12):e869.
[19] Srour ML, Farley E, Mpinga EK. Lao Noma survivors: A case series, 2002-2020. Am J Trop Med Hyg. 2022;106(4):1269-74.
[20] Gebretsadik HG. Estimation of the prevalence of Noma in Ethiopia, 2007-2019: A retrospective study. Am J Trop Med Hyg. 2023;109(6):1388-92.
[21] Miller LE, Shaye DA. Noma and necrotizing fasciitis of the face and neck. Facial Plast Surg FPS. 2021;37(4):439-45.
[22] Braimah RO, Adeoye J, Taiwo AO, Bello S, Bala M, Butali A, et al. Estimated incidence and clinical presentation of Noma in Northern Nigeria (1999-2024). PLoS Negl Trop Dis. 2025;19(5):e0012818.
[23] Gebretsadik HG, de Kiev LC. A retrospective clinical, multi-center cross-sectional study to assess the severity and sequela of Noma/Cancrum oris in Ethiopia. PLoS Negl Trop Dis. 2022;16(9):e0010372.
[24] Gebretsadik HG. Clinical diagnosis of acute noma: Essential infection markers and clinical presentations. Am J Trop Med Hyg. 2025;112(3):528-32.
[25] Farley E, Amirtharajah M, Shaye DA. Noma, a neglected disease: Prevention is better than cure. Curr Opin Otolaryngol Head Neck Surg. 2022;30(4):219-25.
[26] Uzochukwu I, Moyes D, Proctor G, Ide M. The key players of dysbiosis in Noma disease: A systematic review of etiological studies. Front Oral Health. 2023;4:1095858.
[27] Olaleye M, O'Ferrall AM, Goodman RN, Kabila DW, Peters M, Falq G, et al. Shotgun metagenomic analysis of the oral microbiomes of children with noma reveals a novel disease-associated organism. bioRxiv; 2025:01-54. [cited 2025 Oct 16]. p. 2025.06.24.661267. Available from: https://www.biorxiv.org/content/10.1101/2025.06.24.661267v1
[28] Olcu M, Atalay MA, Percin Renders D. Development of multiplex PCR panel for detection of anaerobic bacteria in clinical samples. Anaerobe. 2022;76:102611.
[29] Bolivar I, Whiteson K, Stadelmann B, Baratti-Mayer D, Gizard Y, Mombelli A, et al. Bacterial diversity in oral samples of children in niger with acute noma, acute necrotizing gingivitis, and healthy controls. PLoS Negl Trop Dis. 2012;6(3):e1556.
[30] Whiteson KL, Lazarevic V, Tangomo-Bento M, Girard M, Maughan H, Pittet D, et al. Noma affected children from Niger have distinct oral microbial communities based on high-throughput sequencing of 16S rRNA gene fragments. PLoS Negl Trop Dis. 2014;8(12):e3240.
[31] Maguire BJ, Shrestha R, Dahal P, Ngu R, Nizigama L, Rashan S, et al. Systematic scoping review of the noma evidence landscape: Current knowledge and gaps. BMJ Glob Health. 2025;10(7):e018023.
[32] Ogbureke KUE. Noma: A neglected area for research. J Dent Res. 2022;101(12):1424-29.
[33] Enwonwu CO, Phillips RS, Ibrahim CD, Danfillo IS. Nutrition and oral health in Africa. Int Dent J. 2004;54(6 Suppl 1):344-51.
[34] Feller L, Altini M, Chandran R, Khammissa R a. G, Masipa JN, Mohamed A, et al. Noma (cancrum oris) in the South African context. J Oral Pathol Med Off Publ Int Assoc Oral Pathol Am Acad Oral Pathol. 2014;43(1):01-06.
[35] Prado-Calleros HM, Castillo-Ventura BB, Jiménez-Escobar I, Ramírez-Hinojosa JP, López-Gómez A, García-de-la-Cruz M, et al. Noma and Noma-like disease in HIV/AIDS patients, a comorbid interaction: A systematic review. J Infect Dev Ctries. 2018;12(2):89-96.
[36] García-Moro M, García-Merino E, Martín-Del-Rey A, García-Sánchez E, García-Sánchez JE. Noma/Cancrum oris: A neglected disease. Rev Esp Quimioter. 2015;28(5):225-34.
[37] Baratti-Mayer D, Gayet-Ageron A, Hugonnet S, François P, Pittet-Cuenod B, Huyghe A, et al. Risk factors for noma disease: A 6-year, prospective, matched case-control study in Niger. Lancet Glob Health. 2013;1(2):e87-e96.
[38] Abdullahi MAS, Balarabe MR, Tyndall JA, Alele FO, Habib AG, Adegboye OA. Noma disease among internally displaced persons in Northeast Nigeria: A retrospective descriptive study. Ther Adv Infect Dis. 2024;11:20499361241261269.
[39] Caulfield A, Alfvén T. Improving prevention, recognition and treatment of noma. Bull World Health Organ. 2020;98(5):365-66.
[40] Khammissa RAG, Lemmer J, Feller L. Noma: A neglected oro-facial childhood disease. Lancet Child Adolesc Health. 2021;5(10):685-86.
[41] Ahlgren M, Funk T, Marimo C, Ndiaye C, Alfvén T. Management of noma: Practice competence and knowledge among healthcare workers in a rural district of Zambia. Glob Health Action. 2017;10(1):1340253.
[42] Satapathy P, Rustagi S, Kumar P, Khatib MN, Gaidhane S, Zahiruddin QS, et al. Understanding noma: WHO's recognition and the path forward in global health. Trans R Soc Trop Med Hyg. 2024;118(9):625-28.
[43] Rakhorst HA, Gresnigt TM, van Kooten O, Nishikawa H, Fourie L, Mizen KD. Reconstruction of noma sequelae: A surgical treatment algorithm developed from lessons from 210 cases in Ethiopia. Plast Reconstr Surg Glob Open. 2023;11(3):e4844.
[44] Speiser S, Langridge B, Birkl MM, Kubiena H, Rodgers W. Update on Noma: Systematic review on classification, outcomes and follow-up of patients undergoing reconstructive surgery after Noma disease. BMJ Open. 2021;11(8):e046303.
[45] Isah S, Amirtharajah M, Farley E, Semiyu Adetunji A, Samuel J, Oluyide B, et al. Model of care, Noma Children's Hospital, northwest Nigeria. Trop Med Int Health TM IH. 2021;26(9):1088-97.
[46] Verma A, Zaheer A, Ahsan A, Anand A, Abu Serhan H, Nazli Khatib M, et al. Noma in the WHO's list of neglected tropical diseases: A review of its impact on undeveloped and developing tropical regions. Prev Med Rep. 2024;43:102764.
[47] Bowling T, Hall N. Improving rural public health through "best practice" water, sanitation and hygiene initiatives. Health Lond Engl 1997. 2019;23(2):197-214.

PARTICULARS OF CONTRIBUTORS:
1. Undergraduate Student, Department of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Wardha, Maharashtra, India.
2. Professor and Head, Department of Community Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Wardha, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Mr. Bhagyesh Sapkale,
Department of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Higher Education and Research, Wardha-442107, Maharashtra, India.
E-mail: bhagyeshsapkale@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Aug 26, 2025
- Manual Googling: Nov 20, 2025
- iThenticate Software: Nov 22, 2025 (4%)

ETYMOLOGY: Author Origin

EMENDATIONS: 5

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? NA
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Aug 16, 2025**
Date of Peer Review: **Oct 16, 2025**
Date of Acceptance: **Nov 25, 2025**
Date of Publishing: **Apr 01, 2026**