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STAGE 1 ACUTE NECROTIZING ULCERATIVE GINGIVITIS TRIGGERED BY ACADEMIC STRESS AND SLEEP DEPRIVATION: A CASE REPORT

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Abstract

This case report highlights the successful step-by-step management of Stage 1 Acute Necrotizing Ulcerative Gingivitis (ANUG) and emphasizes the critical role of managing psychosomatic triggers.

A 19-year-old female university student presented with severe, painful, and generalized bleeding gums, high fever, and malaise developing over one week. The condition severely impaired her ability to brush her teeth, eat, and swallow. Social history revealed extreme academic stress and sleep deprivation due to upcoming examinations, leading to nutritional neglect and poor oral hygiene.

Intraoral examination showed generalized marginal gingivitis with characteristic crater-like ulcers covered by a grayish pseudomembrane along the palatal aspect of teeth 11 to 25. Random blood glucose testing ruled out underlying diabetes mellitus. A final diagnosis of Stage 1 ANUG was established based on Horning and Cohen's classification.

Initial intervention focused on non-invasive therapy to stabilize the patient without mechanical trauma. This comprised empirical pharmacological therapy with systemic amoxicillin (500 mg), paracetamol (500 mg), and a brief 3-day course of dexamethasone (0.5 mg) to control inflammation, combined with chemical plaque control using 0.12% chlorhexidine gluconate mouthwash. Definitive scaling and root planing (SRP) were deferred to a second phase after the acute symptoms subsided.

At the 7-day follow-up, the non-invasive approach achieved complete resolution of pain, extensive epithelialization of the ulcers, and a substantial reduction in gingival inflammation, allowing successful completion of mechanical therapy.

Stage 1 ANUG can be effectively managed through a non-surgical phased protocol. Early chemical plaque control and targeted antimicrobials yield rapid tissue resolution. Addressing psychological stressors and ensuring long-term maintenance are essential key management points to prevent recurrence.

Keywords: case report; clinical reasoning; diagnosis; management; outcome

Abstrak

Laporan kasus ini bertujuan untuk menyajikan tatalaksana bertahap yang berhasil pada pasien ANUG Stadium 1, dengan menekankan keberhasilan terapi non-invasif serta pentingnya mengelola pemicu psikosomatis.

Seorang mahasiswi berusia 19 tahun datang dengan keluhan gusi bengkak, merah, nyeri hebat, dan mudah berdarah di seluruh rongga mulut, disertai demam tinggi dan malaise sejak satu minggu. Gejala tersebut mengganggu kemampuan menyikat gigi, makan, dan menelan. Riwayat sosial menunjukkan stres akademik berat dan kurang tidur akibat menghadapi ujian, yang memicu penurunan asupan nutrisi dan higiene mulut buruk.

Pemeriksaan intraoral menunjukkan gingivitis marginalis generalisata dengan karakteristik crater-like ulcer yang ditutupi pseudomembran keabu-abuan di sepanjang sisi palatal gigi 11 hingga 25. Pemeriksaan gula darah sewaktu menyingkirkan faktor diabetes melitus. Berdasarkan klasifikasi Horning dan Cohen, diagnosis akhir ditetapkan sebagai ANUG Stadium 1.

Intervensi awal diprioritaskan pada terapi non-invasif guna menstabilkan kondisi pasien tanpa trauma mekanis. Terapi meliputi amoxicillin sistemik (500 mg), paracetamol (500 mg), dan kortikosteroid jangka pendek dexamethasone (0,5 mg) selama 3 hari untuk meredakan inflamasi, dikombinasikan dengan kontrol plak kimiawi menggunakan obat kumur klorheksidin glukonat 0,12%. Tindakan scaling dan root planing (SRP) ditunda hingga fase akut terkontrol.

Pada evaluasi hari ke-7, pendekatan non-invasif berhasil mencapai penghilangan rasa nyeri secara total, epitelialisasi penuh pada lesi ulserasi, serta penurunan peradangan gusi yang esensial, sehingga pembersihan mekanis lanjutan dapat diselesaikan dengan aman.

ANUG Stadium 1 dapat dikelola secara efektif melalui protokol bertahap non-bedah. Kontrol plak kimiawi dini dan antimikroba terarget menghasilkan resolusi jaringan yang cepat. Mengatasi faktor pemicu stres psikologis dan menjaga kepatuhan kontrol jangka panjang merupakan poin manajemen kunci untuk mencegah kekambuhan.

Kata Kunci: acute necrotizing ulcerative gingivitis; klorheksidin glukonat; kurang tidur; laporan kasus; stres akademik

INTRODUCTION

Acute Necrotizing Ulcerative Gingivitis (ANUG) represents a distinct, rapidly destructive, and non-contagious inflammatory entity within the spectrum of periodontal diseases. Clinically characterized by painful papillary necrosis, spontaneous hemorrhage, and a distinct fetor oris, this condition primarily targets the marginal gingiva and interdental papillae. If left untreated, it leads to irreversible architectural destruction of the periodontium. Historically identified by various eponyms, including Vincent’s disease and "trench mouth" due to its prevalence among wartime military personnel, the contemporary epidemiological landscape of ANUG has shifted dramatically toward young adults, particularly college-aged individuals between 20 and 24 years old.

Globally, the prevalence of ANUG exhibits a stark epidemiological disparity. In developing regions, such as parts of Sub-Saharan Africa and Southern Asia, it remains a critical public health concern exhibiting a prevalence of up to 25% among high-risk, severely malnourished, or immunocompromised pediatric and young adult cohorts, where it frequently progresses into devastating cancrum oris (noma). Conversely, in developed and middle-income regions, the disease presents sporadically and is heavily correlated with modern lifestyle factors, substance abuse, smoking, and psychosocial triggers. Recent data highlights modern academic environments as potent incubators for these psychosomatic responses, where university students experience a profound convergence of chronic sleep deprivation, poor nutritional choices, and intense anxiety during examination periods.

In the local context of Indonesia, the disease presents a significant Diagnostic Challenge. The relevance of early ANUG detection is amplified by a demographic profile heavily weighted toward young adults. Despite access to modern dental care, individual cases among tertiary students are frequently underreported or misdiagnosed as simple aphthous stomatitis or conventional marginal gingivitis. This diagnostic ambiguity poses a severe clinical challenge; delaying the correct intervention accelerates rapid, irreversible loss of interdental papillae and attachment, permanently compromising long-term oral health. To directly enhance the diagnostic capacity within primary healthcare services (Puskesmas) and address this challenge, two strategic proposals are essential: first, the integration of standardized clinical training programs focused on the pathognomonic signs of acute necrotizing periodontal lesions into the continuing professional development of Puskesmas dental practitioners; and second, the establishment of formalized, digitalized tele-dentistry collaboration with comprehensive secondary or tertiary referral facilities to ensure timely, multidisciplinary specialist intervention for complex cases.

The pathogenesis of ANUG relies on an opportunistic shift within the oral microbiome, where indigenous spirochetes (such as Treponema denticola) and fusiform bacteria proliferate rapidly. However, this microbial shift requires a compromised host immune response to trigger tissue necrosis, with psychological stress acting as the primary systemic catalyst. Elevated stress levels activate the hypothalamic-pituitary-adrenal (HPA) axis, increasing salivary and systemic cortisol levels. This hypercortisolemia suppresses local mucosal immunity, alters microvascular perfusion in the gingiva, and reduces polymorphonuclear leukocyte (PMN) function, rendering the periodontal tissues highly susceptible to bacterial invasion.

Managing ANUG in a young student requires a strategic, phased approach that balances immediate symptom relief with long-term stability. The traditional immediate application of aggressive mechanical debridement such as scaling and root planing (SRP) during the acute phase can induce extreme mechanical trauma to devitalized, highly painful tissues, potentially risking bacteremia. Consequently, a non-invasive initial protocol utilizing targeted empirical antimicrobials, short-term anti-inflammatory agents, and chemical plaque control offers a superior clinical alternative to rapidly dampen the acute inflammatory cascade and control the microbial load before mechanical intervention.

This case report details the successful phased management of Stage 1 ANUG in a 19 year old female university student triggered by severe examination-related academic stress. By highlighting a non-invasive stabilization phase, this report aims to provide clinicians with a clear, practical protocol to achieve rapid tissue resolution, minimize patient discomfort, and emphasize the critical necessity of addressing psychosomatic triggers to prevent recurrence in both local and global academic populations.

CASE PRESENTATION

A 19-year-old female university student presented to the community health center (Puskesmas Gunung Padang Panjang) with a rapid onset of severe oral symptoms developing over the preceding week. The patient's past medical history revealed no chronic systemic diseases, such as diabetes mellitus, blood dyscrasias, or hypertension, and she was not under any long-term pharmacological therapy. However, a relevant familial history of type 2 diabetes mellitus was noted. Social and lifestyle history indicated that the patient was currently facing an intense academic workload with a highly demanding lecture schedule. She was undergoing extreme psychological stress and severe sleep deprivation due to preparation for her upcoming final university examinations, which directly contributed to an irregular lifestyle, characterized by inadequate rest and a heavily compromised, deficient nutritional intake

The chief complaint consisted of diffuse, painful, and severely swollen gums that bled easily, characterized by generalized marginal gingivitis. This condition was accompanied by a high fever with an objective tympanic temperature of 38.9°C, generalized malaise, and a severe sore throat. The patient described an agonizing, constant burning sensation throughout her mouth, reporting a pain intensity score of 8 out of 10 on the Visual

Analog Scale (VAS), which caused profound odynophagia and extreme difficulty during mastication and deglutition, leading to a significant decrease in overall oral intake. Due to the severe, exquisite tenderness elicited upon the slightest physical contact with her gingival tissues, she reported an inability to perform effective mechanical toothbrushing, which progressively worsened her oral hygiene and resulted in an intense, foul breath odor (halitosis).

Extraoral examination revealed a notable, healing ulceration on the vermilion border of the lower lip, which had begun to dry out, alongside palpable and tender localized lymphadenopathy. A comprehensive periodontal charting was performed to establish baseline tissue destruction. The simplified Plaque Index (PI) and Calculus Index

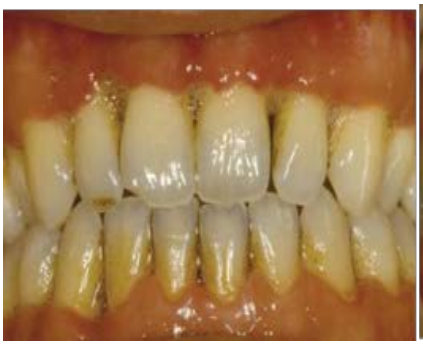
(CI) yielded scores of 2.6 and 2.4, respectively, confirming poor oral hygiene. The bleeding score via Bleeding on Probing (BOP) was exceptionally high at 88%. Pocket probing depths (PD) were strictly maintained within a shallow physiological range of 1 to 3 mm across all quadrants, and there was a complete absence of clinical attachment loss (CAL), confirming that the acute destructive process was strictly confined to the gingival structures without involving the deeper periodontal ligament or alveolar bone.

Intraoral examination demonstrated severe, generalized marginal gingivitis encompassing the maxillary and mandibular arches across the buccal, labial, palatal, and lingual surfaces. Deep, punched-out, crater-like ulcerations covered by a thick, unattached grayish-white pseudomembrane were prominently observed along the palatal aspect of the maxillary anterior to premolar region, extending specifically from teeth 11 to 25. Furthermore, significant accumulation of supragingival and subgingival dental calculus was noted on the labial and lingual surfaces of the mandibular anterior dentition.

Table 1. Summary of Clinical Presentation, Diagnostic Findings, and Phased Management Protocol

Clinical Parameter	**Patient Presentation & Key Findings** **Clinical Interpretation**
Chief Complaint	Severe, painful, and generalized bleeding gums accompanied by a high fever and malaise developing over the course of one week.
Functional Impairment	Severe inability to brush teeth, eat, or swallow due to intense oral pain.
Psychosocial & History Factors	• 19-year-old female university student. • Extreme academic stress and severe sleep deprivation due to upcoming examinations. • Resultant nutritional neglect and poor oral hygiene compliance.
Intraoral Examination	• Generalized marginal gingivitis. • Characteristic crater-like ulcerations covered by a thick, grayish pseudomembrane. • Lesions localized predominantly along the palatal aspect of teeth 11 to 25.
Supporting Examination	Random Blood Glucose (RBG) testing within normal physiological limits (rules out underlying Diabetes Mellitus as a systemic contributor).
Diagnostic Classification	Stage 1 Acute Necrotizing Ulcerative Gingivitis (ANUG) (Classified according to Horning and Cohen's diagnostic criteria)

Phase 1: Initial Non-Invasive Therapy (Aims: Controlled inflammation, infection eradication, and pain management without mechanical trauma)	• Systemic Antimicrobial: Amoxicillin 500 mg (3 times daily). • Systemic Anti-inflammatory: Dexamethasone 0.5 mg (short course, 3 days). • Analgesic: Paracetamol 500 mg (taken as needed for pain and fever relief). • Chemical Plaque Control: 0.12% Chlorhexidine Gluconate mouthwash.
Phase 2: Definitive Mechanical Therapy (Deferred until the resolution of acute symptoms)	Scaling and Root Planing (SRP) performed once the local tissue stabilization and pain relief were achieved.
Clinical Outcomes (7-Day Follow-Up)	• Complete resolution of severe oral pain. • Extensive epithelialization of the crater-like palatal ulcers. • Substantial reduction in generalized gingival inflammation, facilitating safe mechanical debridement.
Long-Term Maintenance Strategy	Addressing academic psychosocial stressors, lifestyle counseling (sleep and nutrition), and strict long-term periodontal monitoring to prevent disease recurrence.



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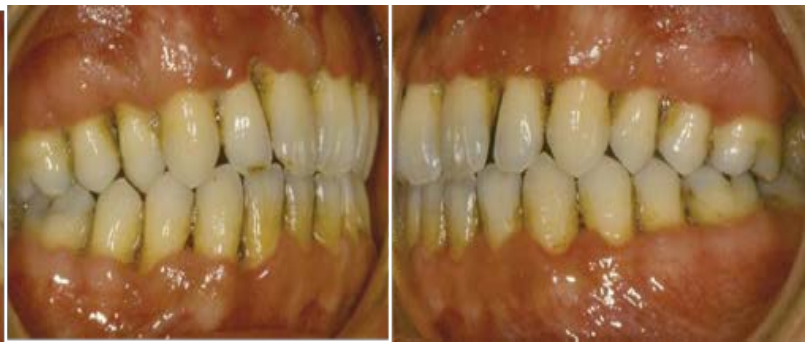

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Furthermore, significant accumulation of supragingival and subgingival dental calculus was noted on the labial and lingual surfaces of the mandibular anterior dentition

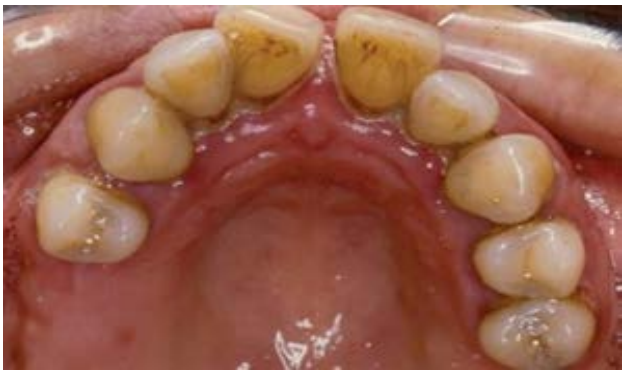


Figure : 3

Following the completion of the acute treatment phase (7 days post-initial intervention), a subsequent follow-up examination demonstrated complete resolution of the pathognomonic crater-like ulcers and significant regression of the generalized gingival inflammation. Quantitative clinical parameters showed a reduction in the Visual Analog Scale (VAS) pain score from 8 to 0, a tympanic temperature of 36.5°C, and a decrease in the Bleeding on Probing (BOP) score to 18%. The Plaque Index (PI) and Calculus Index (CI) improved to 0.8 and 0.5, respectively, following the initial debridement. Serial follow-up photographs (Figure 4 and Figure 5) document the chronological sequence of tissue reorganization and complete re-epithelialization of the previously necrotic interdental papillae

CLINICAL REASONING

The clinical reasoning pathway for this patient necessitated a meticulous differential diagnosis to differentiate ANUG from other acute inflammatory and ulcerative disorders of the oral cavity. The primary differentials considered were Primary Herpetic Gingivostomatitis (PHG), Generalized Chronic Marginal Gingivitis, and Acute Necrotizing Ulcerative Gingivitis (ANUG). PHG, induced by the Herpes Simplex Virus-1 (HSV-1), commonly presents with high fever, malaise, diffuse gingival erythema, and severe pain. However, PHG is strongly characterized by the initial formation of clusters of small, spherical vesicles across any part of the oral mucosa including the attached gingiva, buccal mucosa, and tongue—which subsequently rupture to form shallow, circular fibrin-covered ulcers. In contrast, this patient presented with isolated, deep, crater-like ulcerations strictly confined to the interdental papillae and marginal gingiva without any history or clinical evidence of vesicular eruptions, thereby effectively ruling out a viral etiology. Generalized Chronic Marginal Gingivitis was also evaluated due to the extensive presence of dental plaque and calculus. While chronic gingivitis induces marginal redness, swelling, and bleeding upon probing, it is fundamentally a painless, slow-progressing condition that does not cause tissue necrosis, ulceration, systemic symptoms like fever, or severe halitosis. The rapid, acute onset of agonizing pain, spontaneous hemorrhage, systemic involvement, and localized tissue destruction observed in this patient clearly excluded simple plaque-induced chronic gingivitis. The definitive diagnosis of ANUG was established based on the pathognomonic triad: rapid onset of severe localized pain, necrosis of the interdental papillae manifesting as "punched-out" crater-like lesions, and spontaneous gingival hemorrhage. The patient's clinical presentation perfectly aligned with Stage 1 of Horning and Cohen's classification, where the necrotizing process is strictly localized to the tips of the interdental papillae. The clinical reasoning was further reinforced by the presence of multiple classic psychosomatic risk factors, including severe academic stress, exhaustion from sleep deprivation, and subsequent nutritional neglect, which are known to downregulate host polymorphonuclear leukocyte (PMN) function and alter gingival microcirculation, allowing opportunistic fusospirochetal overgrowth.

DIAGNOSTIC ASSESSMENT

The diagnosis of ANUG is primarily pathognomonic and definitively established through comprehensive clinical correlation of the objective signs and subjective symptoms. In accordance with the guidelines established by the American Dental Association, microbiological smear tests or tissue biopsies are not mandatory for a definitive diagnosis of ANUG, as the associated microflora comprises opportunistic organisms that are also isolated from individuals with simple periodontitis or even healthy oral cavities.

However, targeted diagnostic assessments were utilized in this case to screen for potential underlying systemic predispositions. Given the patient's family history of diabetes mellitus and the severe, generalized nature of the acute periodontal destruction, a random blood glucose (RBG) laboratory test was indicated during the follow-up period to evaluate for immediate metabolic anomalies. The laboratory findings revealed an RBG level of 96 mg/dL, indicating no current evidence of hyperglycemia during the presentation. While an RBG level within normal physiological limits cannot definitively rule out early-stage or controlled diabetes mellitus which would ideally require a fasting blood glucose or HbA1c threshold assessment the finding suggested that acute metabolic derangement was not actively driving the severe clinical presentation. Clinical presentation therefore remains the gold standard for diagnosing necrotizing periodontal diseases.

Table 2. Summary of relevant diagnostic findings

Examination	Result	Clinical Interpretation
Visual Intraoral Inspection	Crater-like ulcers with gray pseudomembrane on palatal 11-25; generalized marginal erythema.	Pathognomonic signs of Stage 1 Acute Necrotizing Ulcerative Gingivitis.
Subjective Pain & Symptom Assessment	Acute severe pain, oral burning sensation, odynophagia, fetor oris.	Reflects acute tissue necrosis, nerve ending exposure, and anaerobic bacterial activity.
Systemic & Physical Evaluation	History of fever (1 week duration), dried lower lip ulcer, tender local lymphadenopathy.	Indicates systemic inflammatory response to acute localized infection.
Random Blood Glucose (RBG) Test	96 mg/dL	Within normal limits; rules out diabetes mellitus as an immunocompromising factor.
Subgingival Microbiological Plak Analysis	Presence of Actinomyces naeslundii	Non-specific heterogeneous oral flora; confirms diagnosis relies on clinical features.

THERAPEUTIC INTERVENION

The therapeutic management of Acute Necrotizing Ulcerative Gingivitis (ANUG) must follow a structured, phased protocol designed to immediately control the acute localized infection, minimize tissue destruction, alleviate severe pain, and eliminate contributing predisposing risk factors. Due to the exquisite pain and active tissue necrosis presenting during the initial visit, aggressive mechanical debridement (such as extensive scaling and root planing) was contraindicated, as it could delay epithelial healing, cause severe bleeding, and induce intolerable pain. Therefore, the first phase focused entirely on empirical pharmacological therapy and chemical plaque control to stabilize the acute condition. The initial pharmacological regimen comprised a combination of systemic antimicrobial, analgesic, and anti-inflammatory agents. Systemic amoxicillin (500 mg administered orally every 8 hours for 7 days) was prescribed as the first-line empirical antibiotic. Amoxicillin provides a broad-spectrum bactericidal effect that is highly effective against the diverse microbial entities implicated in ANUG pathogenesis, successfully arresting the progression of the necrotizing process. To manage the agonizing pain and burning sensation that caused severe odynophagia, paracetamol (500 mg administered orally every 8 hours) was prescribed as an analgesic. Additionally, dexamethasone (0.5 mg administered orally every 8 hours) was incorporated as a short-term anti-inflammatory agent to rapidly reduce localized marginal gingival edema

and erythema. For chemical plaque control, the patient was prescribed a 0.12% chlorhexidine gluconate mouthwash, to be used twice daily (rinsing for 30 seconds). Chlorhexidine gluconate serves as the gold standard for chemical biofilm control in acute periodontal lesions, effectively reducing the oral anaerobic bacterial load when mechanical brushing is impaired by pain. Crucial Communication, Information, and Education (CIE) was provided to the patient. She was instructed to maintain oral hygiene using a very gentle brushing technique, to strictly comply with the prescribed rinsing protocol, and to optimize her host immune response by ensuring adequate rest, managing psychological stress, and consuming a soft, nutrient-rich diet with high fluid intake.

THERAPEUTIC SEQUENCE

The chronological clinical management of this case was systematically executed across three sequential phases to ensure complete resolution and prevent recurrence:

- Phase I (Acute Therapeutic Phase): Conducted during the initial visit, this phase aimed to alleviate severe subjective symptoms and suppress active polymicrobial destruction through a combination of targeted local and systemic interventions.
  - Local Intervention & Debridement: Due to the extreme tissue tenderness (VAS 8) and high risk of bacteremia, conventional scaling was strictly avoided. Local therapy commenced with gentle irrigation using a warm 0.12% chlorhexidine gluconate solution to flush out food debris and loose necrotic architecture. This was followed by careful superficial debridement using sterile cotton pellets soaked in 3% hydrogen peroxide to gently wipe away the unattached, grayish pseudomembrane and superficial bioburden from the palatal lesions of teeth 11–25, effectively introducing oxygen to disrupt the strictly anaerobic microenvironment.
  - Specific Oral Hygiene Instruction (OHI): The patient was explicitly instructed to suspend all mechanical toothbrushing and interdental flossing for the first 24 to 48 hours in the affected maxillary anterior region to prevent further mechanical trauma to the denuded connective tissue. Mechanical plaque control was temporarily replaced by the chemical action of 0.12% chlorhexidine gluconate mouthwash, used to rinse gently twice daily (15 mL for 60 seconds) for a strict duration of 7 days. The patient was advised to switch to an ultra-soft bristled toothbrush using a gentle roll technique only after the resolution of acute pain.
  - Pharmacological Durations: Systemic empirical pharmacotherapy was precisely timed to manage both infection and collateral inflammation: systemic amoxicillin (500 mg every 8 hours) was maintained for a full 7-day course to ensure eradication of tissue-invasive bacteria; systemic dexamethasone (0.5 mg every 8 hours) was strictly limited to a brief 3-day course to bridge the window of severe inflammatory edema and odynophagia without causing systemic immunosuppression; and paracetamol (500 mg every 8 hours, as needed) was prescribed for analgesia during the first 3 to 5 days.
  - Criteria for Scaling and Root Planing (SRP): Complete mechanical instrumentation, including supragingival scaling and subgingival root planing (SRP) to eliminate the heavy calculus accumulation on the mandibular anterior teeth, was deferred until Day 3, once the initial 48-hour therapeutic window had passed. The criteria to initiate SRP required a complete absence of spontaneous gingival hemorrhage, a reduction in the pain threshold to a manageable level (VAS less than or equal to 3), and visible signs of initial epithelial consolidation over the papillae. Conducted during the first visit, aiming to alleviate severe subjective symptoms and suppress active polymicrobial destruction. This involved immediate empirical prescription of systemic amoxicillin, paracetamol, dexamethasone, and 0.12% chlorhexidine gluconate mouthwash, supplemented by comprehensive lifestyle and oral hygiene modifications.
- Phase II (Predisposing and Systemic Factor Evaluation & Mechanical Therapy Initiative)
- Conducted at the 7-day recall visit (second visit), this phase shifted focus toward systemic screening, local baseline stabilization, and the initiation of definitive mechanical therapy.
  - Local Intervention & Definitive Instrumentation: Clinical inspection revealed complete resolution of the necrotic pseudomembrane, closure of the crater-like ulcers, and a pain score of zero (VAS 0). Local therapy commenced with copious irrigation of the gingival sulci using a 0.12% chlorhexidine gluconate solution to mechanically flush out loose bacterial biofilm. Having met the strict clinical criteria to initiate subsurface instrumentation—specifically the complete absence of spontaneous hemorrhage, visible epithelial consolidation over the margins, and a manageable pain threshold—thorough supragingival

scaling and subgingival root planing (SRP) were performed using ultrasonic scalers and sharp Gracey curettes to completely eliminate the heavy chronic calculus accumulation, particularly on the mandibular anterior teeth.

- Specific Oral Hygiene Instruction (OHI): The patient was instructed to transition from purely chemical plaque control back to active mechanical hygiene. The OHI specified the use of an ultra-soft bristled toothbrush utilizing the modified Bass technique, directing the bristles at a 45-degree angle into the sulcus with gentle vibratory strokes to avoid traumatizing the newly epithelialized papillae. The patient was also introduced to gentle interdental flossing to manage the altered interdental architecture and prevent biofilm stagnation in the resolving papillae.
- Pharmacological Durations & Management: Having successfully completed the full 7-day empirical course, systemic amoxicillin was concluded. No further systemic analgesics or corticosteroids were prescribed. To support early tissue remodeling and prevent opportunistic recurrence while the patient mastered the new mechanical brushing technique, chemical plaque control via 0.12% chlorhexidine gluconate mouthwash was extended for a final 7 days (twice daily, 15 mL for 60 seconds), establishing a strict cumulative limit of 14 days of chlorhexidine therapy to prevent severe extrinsic tooth staining.
- Systemic Screening: The patient's random blood glucose (RBG) was evaluated via a finger-prick capillary test to screen for immediate metabolic anomalies, yielding a normal level of 96 mg/dL, indicating no current evidence of active hyperglycemia.

- Phase III (Chronic Condition Correction & Long-Term Maintenance Phase)
- Conducted at the 14-day recall visit (third visit) and structured for long-term monitoring, this phase aimed to correct residual chronic local etiologies, reinforce tissue healing, and manage long-term systemic risks.
  - Local Intervention & Re-evaluation: Local intervention at this stage involved selective coronal polishing using a low-abrasion paste to remove minor extrinsic chlorhexidine stains and residual soft debris. A comprehensive periodontal re-evaluation was conducted: periodontal charting confirmed pocket depths remained stable within the physiological range of 1–3 mm, the Bleeding on Probing (BOP) score decreased to 18%, and the Plaque Index (PI) fell to 0.8, demonstrating excellent tissue stabilization and the complete absence of secondary clinical attachment loss.
  - Specific Oral Hygiene Instruction (OHI): Chemical plaque control (chlorhexidine mouthwash) was completely discontinued to allow the re-establishment of normal oral microflora. The OHI was upgraded to long-term mechanical maintenance, reinforcing the daily use of a medium-soft toothbrush, standard fluoride toothpaste, and daily interdental flossing. The patient was educated on the permanent anatomical changes—specifically the slight blunting of the interdental papillae between teeth 11 and 25—and the critical importance of meticulous interdental cleaning to prevent future plaque entrapment.
  - Stress & Lifestyle Management (CIE): A vital component of this phase was comprehensive Communication, Information, and Education (CIE) targeting the patient's primary psychosomatic triggers. The patient was counseled on stress-coping mechanisms, the physiological necessity of maintaining a minimum of 6–8 hours of sleep during academic examination periods, and the implementation of a balanced diet rich in micronutrients (Vitamins C and B complexes) to maintain optimal host polymorphonuclear leukocyte (PMN) function and gingival microcirculation.
  - Maintenance Protocol: To address pre-existing chronic local etiologies and control psychosomatic stress factors permanently, the patient was placed on a strict 3-month supportive periodontal therapy (SPT) recall schedule for the first year to monitor for early signs of recurrence and ensure sustained compliance with oral hygiene practices.

CASE TIMELINE

The chronological sequence of clinical events, diagnostic interventions, therapeutic adjustments, and clinical outcomes is summarized in the table below:

Table 2. Clinical case timeline

Time	Clinical Event	Investigation	Treatment	Outcome
Day 0 (Initial Visit)	Patient presented with severe generalized gum swelling, pain, spontaneous bleeding, fever, halitosis, and odynophagia.	Comprehensive anamnesis, extraoral and intraoral visual examination.	Prescription of Amoxicillin 500mg, Paracetamol 500mg, Dexamethasone 0.5mg, and 0.12% Chlorhexidine mouthwash; extensive CIE provided.	Immediate initiation of acute pharmacological therapy; mechanical debridement postponed due to severe pain.
Day 7 (First Follow-up)	Patient reported complete elimination of pain and oral burning sensation; marked improvement in mastication.	Intraoral clinical inspection; Laboratory Random Blood Glucose (RBG) test.	Discontinuation of systemic antibiotics and anti-inflammatories; continuation of 0.12% Chlorhexidine mouthwash.	Substantial clinical healing of crater-like ulcers; gingival inflammation significantly regressed; RBG within normal limits (96 mg/dL).
Day 14 (Second Follow-up)	Patient was completely asymptomatic; gingival tissues showed stabilized tissue resolution.	Routine intraoral clinical evaluation and plaque index assessment.	Professional supragingival and subgingival scaling and root planing (SRP); intensive oral hygiene instruction (OHI).	Complete resolution of marginal gingivitis; successful transition to supportive periodontal therapy and stress reduction protocol.

OUTCOME AND FOLLOW-UP

The clinical outcome of the stepped therapeutic protocol demonstrated excellent, rapid healing of the necrotic periodontal tissues. At the 7-day follow-up recall (Day 7), the patient reported complete resolution of the agonizing localized pain, fever, and oral burning sensations. She was able to masticate and swallow comfortably without any discomfort, which successfully restored her nutritional intake. Objective intraoral examination revealed that the pathognomonic, crater-like ulcers along the palatal aspect of teeth 11 to 25 had undergone significant epithelialization and healing. Marginal erythema and edematous swelling across all quadrants showed a remarkable reduction compared to the initial presentation.

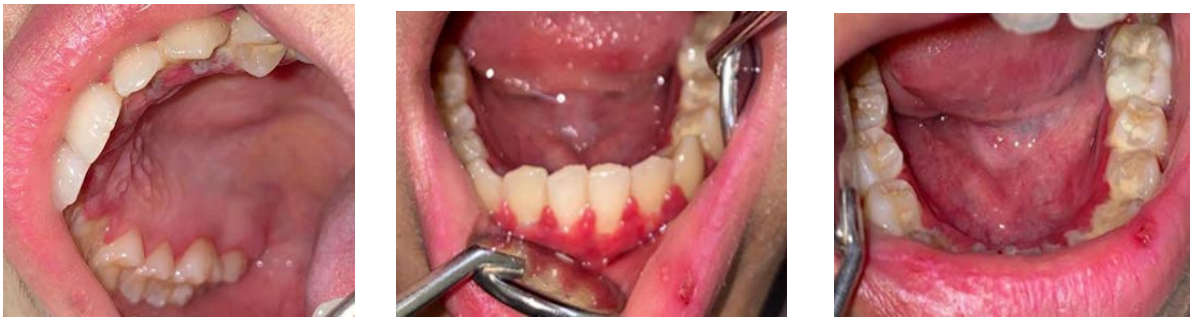
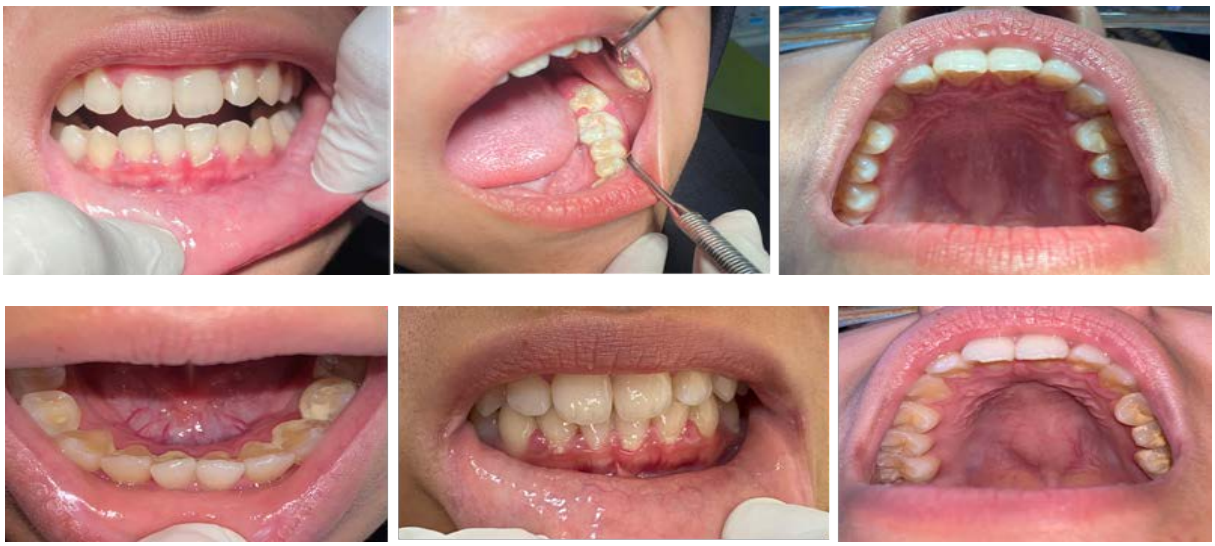


Figure : 4

The laboratory assessment conducted during this visit confirmed a normal random blood glucose level of 96 mg/dL, ruling out systemic diabetic complications

By the third clinical visit (Day 14), the patient remained entirely asymptomatic, and her gingival health had improved significantly. With the acute necrotizing infection fully controlled and the tissue no longer tender to physical contact, the patient successfully underwent thorough mechanical scaling and root planing to eliminate the pre-existing dental calculus and bacterial plaque matrix on her mandibular lingual surfaces. The patient demonstrated excellent compliance with the oral hygiene instructions and lifestyle modifications. She was advised to maintain a strict 3-month periodic recall schedule for long-term supportive periodontal therapy, to ensure adequate daily sleep, and to implement effective stress-coping mechanisms during future academic examination periods to prevent any potential disease recurrence



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DISCUSSION

Acute Necrotizing Ulcerative Gingivitis (ANUG) is an acute, destructive periodontal infection that demands immediate diagnosis and prompt therapeutic intervention. Misdiagnosis or delayed treatment can exacerbate the condition, leading to extensive attachment loss or progression into more severe necrotizing diseases, such as necrotizing ulcerative periodontitis (NUP) or necrotizing stomatitis. In this case, the patient presented with pathognomonic clinical features, including deep crater-like ulcers along the palatal surfaces, spontaneous hemorrhage, severe pain, an oral burning sensation, fever, and fetor oris. These findings strongly support the diagnosis of ANUG, which, based on Horning and Cohen's classification, was categorized as Stage 1 due to the necrosis being isolated to the tips of the interdental papillae.

The initiation of ANUG in this patient highlights the critical role of predisposing factors in altering host immune susceptibility. The patient was a 19-year-old college student experiencing extreme psychological stress and severe sleep deprivation due to an upcoming final university examination schedule. Psychosomatic stress acts as a major catalyst by activating the hypothalamic-pituitary-adrenal (HPA) axis, resulting in an elevated level of systemic corticosteroids. This hypercortisolemia directly suppresses the chemotactic and phagocytic functions of lymphocytes and polymorphonuclear leukocytes (PMNs), debilitating the host's primary defense against periodontal pathogens. Furthermore, increased sympathetic nervous system activity during emotional stress triggers the secretion of systemic adrenaline and peripheral noradrenaline, inducing intense vasoconstriction within the gingival microcirculation. The resulting localized ischemia, combined with endotoxins from opportunistic anaerobic microflora, leads to the characteristic aseptic tissue necrosis. This physiological crisis also negatively altered the patient's behavior, resulting in poor mechanical plaque control and nutritional neglect, which further accelerated the disease process.

The microbiological analysis in this case identified the presence of Actinomyces naeslundii. However, in alignment with the American Dental Association guidelines, microbiological testing holds limited definitive diagnostic value due to the high heterogeneity of the oral biofilm. Classic periodontopathic pathogens, such as Fusobacterium nucleatum, Prevotella intermedia, and Treponema species, are normal inhabitants of the oral cavity that proliferate opportunistically only when the host immune response is compromised. Hence, a definitive diagnosis of ANUG remains strictly grounded in pathognomonic clinical signs. Additionally, because severe generalized periodontal destruction can be a manifestation of underlying systemic conditions like diabetes mellitus or HIV, metabolic screening was essential. The patient's random blood glucose level of 96 mg/dL effectively ruled out undiagnosed diabetes mellitus, establishing psychological exhaustion as the primary driver.

The successful management of Stage 1 Acute Necrotizing Ulcerative Gingivitis (ANUG) in this patient highlights a critical paradigm shift aligned with the clinical guidelines of the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP): prioritizing initial non-invasive biochemical stabilization over immediate mechanical intervention, particularly when the disease is strongly driven by psychosomatic factors. While traditional classic guidelines often advocate for immediate mechanical debridement during the initial visit, this case demonstrates that a phased approach yields rapid tissue resolution while significantly minimizing patient morbidity.

The decision to defer immediate scaling and root planing (SRP) during the acute phase was strategic. Introducing mechanical instruments to devitalized, ulcerated interdental papillae coated with a friable pseudomembrane inflicts severe mechanical trauma, exacerbates intense localized pain, and risks inducing systemic bacteremia through exposed capillary beds. This approach aligns with the AAP/EFP consensus and recent comparative studies emphasizing that in the presence of acute systemic symptoms like high fever and malaise, chemical plaque control combined with targeted systemic therapy should always precede instrumentation. The initial protocol utilized 0.12% chlorhexidine gluconate mouthwash as a first-line chemical anti-plaque agent to disrupt the superficial bacterial biotype without mechanical friction. To target the deep

tissue-invading opportunistic pathogens—specifically the fusospirochetal complex containing Treponema denticola and Fusobacterium nucleatum—systemic amoxicillin (500 mg) was administered. This empirical antimicrobial regimen successfully halted the necrotizing cascade within 48 hours, an outcome comparable to recent clinical trials where systemic antibiotics achieved rapid microbial load reduction before mechanical debridement was initiated.

Furthermore, the short-term incorporation of systemic dexamethasone (0.5 mg) for 3 days served as a potent adjunct to modulate the host's hyper-inflammatory response. By rapidly dampening the acute inflammatory cascade, the systemic corticosteroid relieved the severe local edema and microvascular congestion in the marginal gingiva. This accelerated the alleviation of the patient's constant oral pain and resolved her profound odynophagia, allowing her to resume adequate nutritional intake and effective self-performed oral hygiene by day 3. At the 7-day follow-up, this non-surgical stabilization phase had transformed the highly fragile, bleeding gingival architecture into a stable, epithelialized, and non-tender tissue, allowing definitive SRP to be completed comfortably and efficiently without causing further attachment loss.

A pivotal aspect of this case management, which directly influenced the prevention of recurrence, was the identification and targeted management of the patient's psychological stressors. Modern periodontal literature formalized by the AAP/EFP increasingly recognizes ANUG not merely as an isolated infectious disease, but as a psychosomatic manifestation of host immune impairment under the classification of Necrotizing Periodontal Diseases (NPDs). The patient's status as a 19-year-old university student undergoing intense examination anxiety represents a classic modern demographic archetype for this condition. The biological mechanism linking academic stress to periodontal necrosis is mediated via the activation of the hypothalamic-pituitary-adrenal (HPA) axis.

Chronic anxiety and sleep deprivation trigger a sustained hyperactivation of the HPA axis, resulting in a marked elevation of systemic and salivary cortisol levels. Clinically, elevated salivary cortisol serves as a reliable objective biomolecular marker for acute academic stress and correlates directly with the severity of periodontal inflammation. This prolonged hypercortisolemia exerts a profound immunosuppressive effect on the local periodontium. It downregulates the expression of critical pro-inflammatory cytokines and alters cellular immunity by suppressing the chemotaxis, migration, and phagocytic function of polymorphonuclear leukocytes (PMNs). Furthermore, high cortisol levels impair local secretory IgA (sIgA) production in saliva—which serves as the first line of mucosal defense—and induce localized microvascular vasoconstriction within the delicate interdental capillary loops via alpha-adrenergic stimulation. Consequently, the gingival tissue loses its primary immunologic and vascular defense mechanisms, creating an ideal microenvironment for indigenous oral spirochetes to transition from commensal organisms into aggressive, tissue-invasive pathogens.

Compounding this physiological vulnerability, high psychosocial stress inherently degrades health-promoting behaviors, leading to a severe decline in oral hygiene compliance and nutritional neglect. In this case, addressing the microbiological component alone through antibiotics and chlorhexidine would only yield transient success if the systemic catalyst remained unaddressed. Therefore, incorporating psychological interventions—specifically stress-management counseling, sleep hygiene education, and dietary modification—was integrated as a core component of the definitive treatment phase. Research evaluating long-term outcomes in young adult ANUG patients indicates that individuals who receive comprehensive lifestyle and stress counseling exhibit significantly lower recurrence rates compared to those treated solely with conventional periodontal debridement. By equipping the patient with coping mechanisms to manage academic anxiety and emphasizing the physiological link between systemic stress and oral immunity, the clinical team successfully addressed the root etiology of the opportunistic infection.

In conclusion, this case underscores that Stage 1 ANUG in contemporary young adult populations demands a holistic, phased treatment protocol. Initial non-invasive pharmacological and chemical stabilization effectively prepares the oral environment for safe, trauma-free mechanical therapy. Concurrently, the integration of

psychological and lifestyle interventions stands as an indispensable requirement in modern case management, ensuring the long-term integrity of the periodontium and preventing the cyclical recurrence of this highly destructive disease.

The management of ANUG in this case followed a structured, phased protocol that strictly aligns with current periodontal therapeutic guidelines. Because active tissue necrosis, exposure of underlying nerve endings, and severe pain (VAS 8) precluded immediate mechanical scaling during the first visit, chemical biofilm disruption was prioritized as the initial line of defense. The use of a 0.12% chlorhexidine gluconate mouthwash served as a highly effective non-mechanical antimicrobial agent, targeted at downregulating the anaerobic load without inflicting further trauma to the friable connective tissue. This approach is strongly supported by the American Academy of Periodontology (AAP) guidelines, which advocate for chemical plaque control and superficial debridement during the acute phase of necrotizing periodontal diseases, deferring definitive subgingival instrumentation until the acute symptoms subside.

Systemic antimicrobial therapy was indicated in this patient due to clear evidence of systemic involvement, manifested by a high fever (38.9°C) and tender lymphadenopathy. Systemic amoxicillin (500 mg every 8 hours for 7 days) was selected as the empirical first-line bactericidal agent. While several international literatures, including the European Federation of Periodontology (EFP) protocols, frequently recommend metronidazole as the gold standard for necrotizing diseases due to its strict anaerobic spectrum, amoxicillin remains an equally effective alternative in clinical settings where metronidazole may be poorly tolerated or when a broader bactericidal action against a mixed polymicrobial flora is required, provided the patient has no penicillin allergies.

To manage the severe localized inflammation and associated odynophagia, paracetamol (500 mg) was combined with a short-term regimen of systemic dexamethasone (0.5 mg every 8 hours for 3 days). The incorporation of systemic corticosteroids in the acute phase of ANUG remains a subject of careful clinical evaluation in periodontal literature. While standard guidelines primarily emphasize non-steroidal anti-inflammatory drugs (NSAIDs) or simple analgesics to manage pain, short-term corticosteroid therapy can be clinically justified in cases presenting with debilitating edema and severe throat involvement that compromises nutritional intake, provided absolute contraindications such as active viral infections like Primary Herpetic Gingivostomatitis (PHG) are strictly ruled out. In this case, the rapid down-regulation of the inflammatory cascade facilitated a prompt return to proper nutrition and mechanical hygiene, leading to complete ulcer resolution and a reduction of the BOP score to 18% at the 7-day recall. This clinical outcome demonstrates that a meticulously timed combination of local chemical debridement, targeted empirical antimicrobials, and controlled anti-inflammatory therapy provides a safe and predictable resolution of acute necrotizing periodontal lesions

The successful management of Stage 1 Acute Necrotizing Ulcerative Gingivitis (ANUG) in this patient highlights a critical paradigm shift in contemporary periodontology: prioritizing initial non-invasive biochemical stabilization over immediate mechanical intervention, particularly when the disease is strongly driven by psychosomatic factors. While traditional classic guidelines often advocate for immediate, cautious mechanical debridement during the initial visit, this case demonstrates that a phased approach yields rapid tissue resolution while significantly minimizing patient morbidity.

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disrupts the superficial bacterial biotype without mechanical friction. To target the deep tissue-invading opportunistic pathogens—specifically the fusospirochetal complex containing Treponema denticola and Fusobacterium nucleatum—systemic amoxicillin (500 mg) was administered. This empirical antimicrobial regimen successfully halted the necrotizing cascade within 48 hours, an outcome comparable to recent clinical trials where systemic antibiotics achieved rapid microbial load reduction in immunocompromised or highly stressed patients before any mechanical debridement was initiated.

Furthermore, the short-term incorporation of systemic dexamethasone (0.5 mg) for 3 days served as a potent adjunct to modulate the host's hyper-inflammatory response. By rapidly dampening the acute inflammatory cascade, the systemic corticosteroid relieved the severe local edema and microvascular congestion in the marginal gingiva. This accelerated the alleviation of the patient's constant oral pain and resolved her profound odynophagia, allowing her to resume adequate nutritional intake and effective self-performed oral hygiene by day 3. At the 7-day follow-up, this non-surgical stabilization phase had transformed the highly fragile, bleeding gingival architecture into a stable, epithelialized, and non-tender tissue, allowing definitive SRP to be completed comfortably and efficiently without causing further attachment loss.

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Once the acute infection was completely resolved and tissue tenderness subsided, the patient successfully transitioned to Phase II therapy involving professional scaling and root planing (SRP) to eliminate the underlying chronic irritants (dental calculus). This stepped protocol underscores that early empirical management combined with long-term supportive periodontal therapy and stress reduction is key to preventing disease recurrence and maintaining periodontal integrity.

DIAGNOSTIC CHALLENGE

The diagnostic process in this case presented distinct clinical challenges, primarily arising from the limitations inherent to a primary healthcare setting (Puskesmas). In such facilities, advanced diagnostic modalities such as polymerase chain reaction (PCR) for specific anaerobic periopathogens, immunologic assays, or histopathological tissue biopsies are strictly unavailable. This logistical constraint required the clinical team to rely entirely on meticulous clinical correlation and physical examination to establish a definitive diagnosis. Because ANUG is relatively rare in immunocompetent individuals without severe underlying systemic diseases, relying solely on clinical presentation demanded a high level of diagnostic precision to prevent misdiagnosis.

A significant challenge was differentiating the lesion from viral conditions, specifically Primary Herpetic Gingivostomatitis (PHG), which shares overlapping systemic symptoms like high fever, malaise, and localized oral pain. The presence of a drying ulcer on the patient's lower lip further complicated the initial visual assessment, raising suspicion of a widespread herpetic infection. However, the diagnostic breakthrough was achieved by identifying the exact anatomic distribution of the tissue destruction; the necrosis was strictly localized as "punched-out" craters at the tips of the interdental papillae and marginal gingiva. The absence of diffuse spherical vesicular clusters on the attached gingiva or buccal mucosa effectively excluded a viral etiology.

Additionally, the acute and exquisite pain experienced by the patient created a physical barrier during the initial intraoral examination. The extreme tenderness made a thorough mechanical evaluation of the subgingival structures impossible during the first visit. Managing the patient's severe anxiety, addressing her inability to tolerate direct tissue manipulation, and obtaining an accurate clinical history despite her severe odynophagia represented a major clinical hurdle. Ultimately, this challenge underscored the necessity of a cautious, non-invasive initial inspection and a strong reliance on pathognomonic clinical markers to confidently initiate empirical therapy.

LIMITATION

In conclusion, this case report highlights that a structured, non-surgical phased protocol combining initial chemical debridement using 0.12% chlorhexidine gluconate and targeted systemic pharmacotherapy achieves predictable short-term clinical resolution of Stage 1 Acute Necrotizing Ulcerative Gingivitis (ANUG) within a 7- to 14-day window. The combination of amoxicillin and short-term dexamethasone effectively arrested active polymicrobial tissue destruction and suppressed debilitating local inflammation, facilitating a rapid restoration of oral function and mechanical hygiene capability. However, due to the lack of long-term follow-up beyond 14 days, no definitive conclusions can be drawn regarding permanent periodontal architectural remodeling or the efficacy of this protocol in preventing disease recurrence during subsequent periods of academic or psychological stress. Meticulous clinical screening to differentiate ANUG from viral lesions remains paramount in primary healthcare settings where advanced diagnostic modalities are unavailable, ensuring safe and highly effective immediate case management.

LEARNING POINTS

1. **Pathognomonic Clinical Diagnosis: Diagnosis** of early-stage ANUG relies definitively on identifying the pathognomonic clinical triad severe localized pain, "punched-out" interdental papillary necrosis, and spontaneous bleeding rendering complex microbiological tests unnecessary for initial intervention.
2. **Stepped Therapeutic Protocol:** Acute necrotizing periodontal lesions must be managed using a phased approach; initial mechanical debridement should be postponed during the highly painful necrotic phase in favor of chemical plaque control (0.12% chlorhexidine) and systemic antimicrobials to ensure rapid, trauma-free tissue resolution.
3. **Psychosomatic Risk Factor Elimination:** Addressing underlying lifestyle and psychosomatic predisposing factors, such as academic stress and sleep deprivation, is just as crucial as local antimicrobial therapy in restoring host immune function and preventing disease recurrence.
4. **Systemic Screening:** Screening for systemic immunocompromising conditions (e.g., blood glucose testing for diabetes) is highly recommended in cases of sudden, generalized acute periodontal destruction to rule out complex systemic involvements.

CONCLUSION

Stage 1 Acute Necrotizing Ulcerative Gingivitis (ANUG) can be successfully managed in a primary healthcare setting through a prompt, structured, and stepped therapeutic protocol. The combination of systemic amoxicillin, short-term anti-inflammatory therapy, and 0.12% chlorhexidine gluconate mouthwash provides immediate symptom relief and rapid epithelial healing without the need for aggressive initial mechanical instrumentation. This case demonstrates that severe academic stress and sleep deprivation can serve as potent systemic triggers for opportunistic periodontal infections in young adults. Ultimate clinical success and the prevention of recurrence depend on transitioning the patient to supportive periodontal therapy including scaling and root planing once the acute phase is controlled, alongside active stress management and lifestyle modifications.

ETHICAL CLEARANCE

Institutional ethical approval was not required for the publication of this clinical case report based on the institutional guidelines of Puskesmas Gunung and the editorial policies regarding retrospective single-patient analyses.

This exemption is justified based on the following criteria:

Ethical Approval and Consent to Participate: As this manuscript describes standard, evidence-based, and non-invasive clinical interventions (pharmacological therapy and non-surgical periodontal debridement) conducted entirely within routine primary oral healthcare delivery, formal institutional review board (IRB) review was exempt. No experimental procedures, novel drugs, or deviations from established clinical protocols were introduced.

Patient Anonymity and Confidentiality: The case report strictly adheres to the ethical principles outlined in the Declaration of Helsinki. To guarantee complete patient anonymity and protect personal privacy in accordance with local healthcare confidentiality standards, all personally identifiable metrics—including the patient's name, medical record numbers, exact residential address, and distinctive facial features—have been completely omitted or masked to ensure the individual's identity cannot be reverse-engineered.

Informed Consent: Explicit written and verbal informed consent was voluntarily obtained from the patient prior to the documentation. This consent granted full permission for the use of clinical case data, diagnostic laboratory values, and serialized intraoral photographs for the sole purpose of scientific documentation and academic publication. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

PATIENT CONSENT

Written informed consent was voluntarily obtained from the patient for the publication of this clinical case report, along with all accompanying intraoral photographs, diagnostic laboratory values, and related clinical data. To rigorously protect patient confidentiality, all personal identifiable information including the patient's name, initials, student registration details, and specific demographic metrics has been completely omitted and anonymized. All clinical images have been strictly cropped to focus solely on the intraoral lesions, ensuring that no identifying extraoral facial features are disclosed. A copy of the signed written consent form is available for review by the Editorial Board of this journal upon request.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest related to the writing and publication of this article. If a conflict exists, it must be clearly disclosed.

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AUTHORS' CONTRIBUTION

- **Arnetty**: Conceptualization of the case report, primary clinical data collection during the patient's initial presentation, administration of the acute pharmacological therapy, and drafting of the initial manuscript.
- **Bambang Ari Putranto**: Direct participation in the follow-up clinical data collection, monitoring of the 7-day therapeutic outcome, laboratory assessment interpretation, and compilation of the case timeline matrix.
- **Hafifah Hanum**: Execution of the Phase III non-surgical periodontal therapy (scaling and root planing), oral hygiene reinforcement, structural design of the clinical reasoning phase, and literature review acquisition.
- **Mega Apriliani**: Clinical analysis validation, diagnostic assessment review, critical manuscript revision for intellectual content, and oversight of the therapeutic sequence alignment.
- **Zahara Hijriani**: Technical drafting supervision, final academic proofreading, alignment with primary healthcare institutional guidelines, and final approval of the version to be published.

DATA AVAILABILITY

The primary clinical data, diagnostic laboratory metrics, and case parameters supporting the findings and conclusions of this case report are fully integrated within the text and tables of the published manuscript. To maintain absolute patient confidentiality and adhere to institutional ethical restrictions, the raw medical charts, signed consent forms, and uncropped extraoral clinical photographs cannot be deposited in a public repository. However, the fully anonymized intraoral clinical images and relevant diagnostic documentation are available from the corresponding author upon reasonable request, subject to approval and compliance with patient privacy regulations.

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Lastly, the authors sincerely thank the patient for her exceptional compliance, trust, and willingness to contribute her clinical data to the advancement of periodontal education and scientific documentation.

LIST OF ABBREVIATIONS

- **ADA** = American Dental Association
- **ANUG** = Acute Necrotizing Ulcerative Gingivitis
- **CIE** = Communication, Information, and Education
- **HPA** = hypothalamic-pituitary-adrenalin
- **HSV-1**= Herpes Simplex Virus-1
- **OHI** = Oral Hygiene Instruction
- **PHG** = Primary Herpetic Gingivostomatitis
- **PMN** = Polymorphonuclear Leukocyte
- **RBG** = Random Blood Glucose
- **SRP** = Scaling and Root Planing

FIGURE AND TABLE LEGENDS

Table 1. Summary of relevant diagnostic findings.

Table 2. Clinical case timeline.

Figure 1. Maxillary intraoral palatal view at baseline presentation. Prominent presentation of classic "punched-out," crater-like necrotizing ulcers covered by a distinctive grayish-white pseudomembrane along the palatal aspect of the anterior teeth (regio 11 to 25).

Figure 2. Mandibular labial view demonstrating severe acute marginal gingivitis. Marked, generalized erythematous margins and significant edematous swelling of the free and attached gingival tissues prior to treatment.

Figure 3. Heavy supragingival dental calculus accumulation on the mandibular lingual surfaces. Pre-treatment visual documentation of the primary local predisposing factor (bacterial plaque matrix and calculus) contributing to severe tissue irritation.

Figure 4. Intraoral clinical follow-up 7 days post-treatment (Second Visit). Significant regression of marginal gingival inflammation, complete resolution of the crater-like ulcers, and advanced epithelial healing following the completion of the empirical pharmacological phase.

Figure 5. Post-scaling intraoral view during the third clinical visit. Comprehensive resolution of the periodontal infection showing healthy gingival architecture, firm tissue consistency, and complete elimination of local calculus burdens following non-surgical periodontal therapy (SRP).

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