Periodontal Emergencies in Clinical Practice

**Paul Erhabor, **Adesuwa Ebomwonyi
*Department of Restorative Dentistry, Edo Specialist Hospital ** Department of Family Dentistry, Edo Specialist Hospital

Correspondence
Dr. Paul Erhabor
Department of Restorative Dentistry
Edo Specialist Hospital
Benin City, Edo State, Nigeria.
E-mail: onemandepot@gmail.com

Background: People place high importance on management of medical emergencies. They, however, pay less attention to management of periodontal emergencies as they do to other medical emergencies. Interestingly, prompt intervention in periodontal emergencies is integral to general health of the individual. Various factors have been identified for the underestimation of the importance of management of periodontal emergencies including access to oral health facilities, cultural practices, low socioeconomic status (educational and income level) and low level of oral health awareness. More enlightenment campaign by dental professionals and improve government policy and implementation in the area of oral health care is needed to raise the level of oral health awareness and need for prompt management of periodontal emergencies.
Objective: Periodontal emergencies commonly present to the dental clinic. This review article reported the clinical features and management of oral diseases that constitutes periodontal emergencies.
Methods: Periodontal emergencies were categorized into components of periodontal emergencies, prevalence of periodontal emergencies, clinical features and management of oral diseases that constitutes periodontal emergencies and level of awareness of oral health in general.
Conclusion: The importance of prompt management of oral diseases that constitutes periodontal emergency cannot be over emphasized as it has been shown to greatly affect the general well-being and health of the individual. Periodontal emergencies, when not timely managed, can lead to residual destruction of periodontal tissues and indeed the oral cavity which can affect the quality of life of the individual. A healthy mouth in a health body is one to be sought by all.
Keywords: periodontal emergency, management, oral diseases

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Paul Erhabor
https://orcid.org/0000-0003-3313-4454
Adesuwa Ebomwonyi
https://orcid.org/0000-0003-3752-8960

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CLINICAL IMPLICATIONS

Clearer understanding on periodontal emergencies will greatly enhance the provision of adequate oral health services geared towards improving the general well-being and health of the individual as well as the quality of life of citizens.

INTRODUCTION

A medical emergency is an acute injury or illness that poses an immediate risk to a person's life or long-term health. It requires prompt intervention from a qualified professional to prevent mortality and/or morbidity [1]. Medical emergency can occur in any field of medicine and dentistry, including periodontology. Periodontal emergency is a medical emergency for which immediate periodontal intervention is the only way to manage the condition successfully. Periodontal emergency requires active intervention for periodontal pathological conditions that are of sudden onset, limited duration and with well-defined clinical features [2].

Components of periodontal emergencies

Components of periodontal emergencies include traumatic lesions (Fig. 1), gingival abscess/periodontal abscess (Fig. 2), aphthous ulceration (Fig. 3 & 4), erythema multiforme, contact hypersensitivity (Fig. 5), acute herpetic gingivostomatitis (Fig. 6, 7), and bacterial infections such as acute necrotizing ulcerative gingivitis (Fig.8)

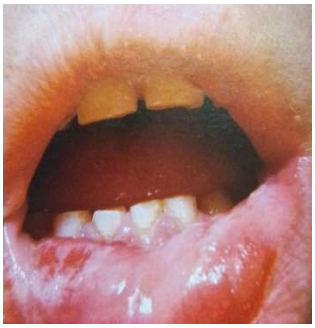

Figure 1: Self-inflicted trauma to the lower lip

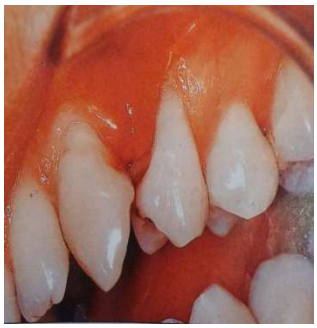

Figure 2: Gingival abscess

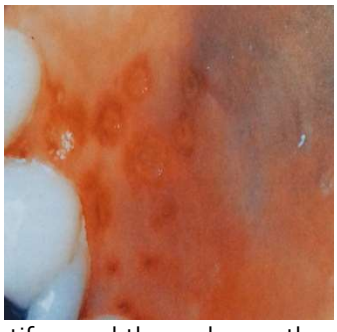

Figure 3: Herpetiform aphthous ulcer on the palatal mucosa

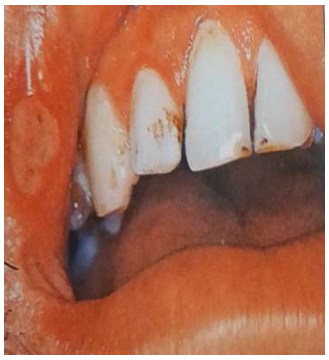

Figure 4: Major aphthous ulcer on the inner upper lip mucosa

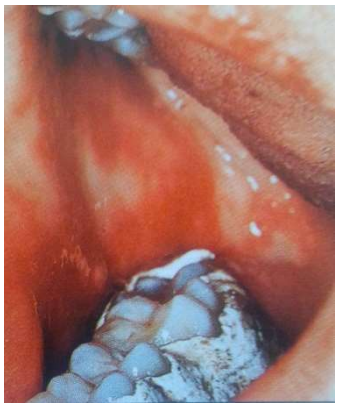

Figure 5: Contact hypersensitivity to a component of Coepack ®, on the buccal mucosa

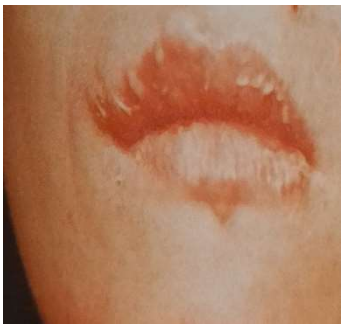

Figure 6: Secondary herpetic simplex "cold sore"

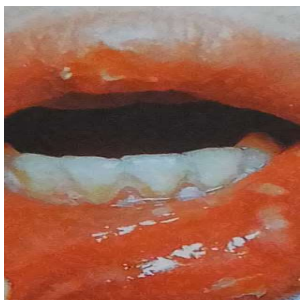

Figure 7: Primary acute herpetic gingivostomatitis infection

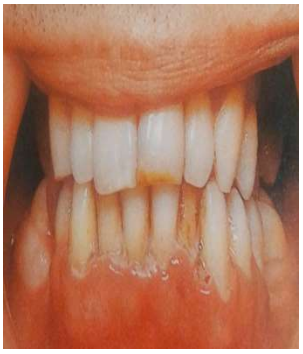

Figure 8: Localized ANUG affecting the lower anterior teeth
All pictures are adapted from Eley, Soory and Mason [3].

Prevalence of periodontal emergencies

The prevalence of oral diseases that constitute periodontal emergencies is variable, depending on the type of oral condition and the study group. All age groups and social strata are affected: children, teenagers, middle-aged group and the elderly [3].

Globally, the estimated point prevalence of oral ulcers is 4% [4]. Vieiera-Andrade *et al.* reported traumatic oral ulcer prevalence of 21.5% among lower classes of Brazilian population. Cury *et al.* reported a 7.5% prevalence of traumatic oral ulcer among men with crack and cocaine addiction. Silva *et al* reported a prevalence of 16.4% for traumatic oral ulcers in patients with diabetes mellitus in a health facility in Brazil [5, 6, 7].

Periodontal abscess is the third most common dental emergency and has a higher incidence among patients with preexisting periodontal pockets [8]. A study reported an incidence of more than 50% among patients with periodontal disease [9].

The prevalence of aphthous ulcer worldwide and the United States of America is about 25% and 40% respectively[4]. Arotiba *et al* (2005) reported a prevalence of 15% for aphthous ulcer in HIV-infected Nigerian patients [10].

Labh and Ramani reported a male predilection for periodontal abscess with a prevalence of 68.2% compared to female (31.8%) among patients attending a private dental clinic in India [11]. Lewis *et al* reported a prevalence of 6 to 7% of periodontal abscess among patients in general dental practice in the United Kingdom whereas Umoh and Azodo reported a prevalence of periodontal abscess among patients attending a Nigerian specialist periodontology clinic of 0.43% [12, 13]. Socioeconomic status as well as geographic variation may account for the different prevalence rate seen in these studies.

In Europe, the prevalence of contact hypersensitivity to dental materials has been reported to be about 0.01% [14]. Oral lichenoid eruption, a group of diseases with a clinical appearance of a lacy pattern similar to the wickham striae found in cutaneous lichen planus and encompassing such diseases as oral lichen planus, oral contact lesions and oral delayed hypersensitivity reactions, has been estimated to have prevalence between 0.02 to 1.5% in India, 2.1-3.8% in South East Asia and 1.9% in Sweden [15].

Primary herpetic gingivostomatitis typically occurs in children younger than the age of 5 years but can also occur in older children or adults. Worldwide, the prevalence rate of primary herpetic gingivostomatitis for populations who are seropositive for Herpes Simplex Virus-1 by 35 years of age, has been estimated to be about 90%. Half of the individuals who have had a primary herpetic infection early in life develop recurrent infection in the form of herpes labialis years later [16, 17].

In western countries, Acute Necrotizing Gingivitis (ANUG) is usually seen in the 16-30years age group with many epidemiological studies from the 1950s reporting about 5% incidence of this condition, particularly among military personnel and college students [3]. More recently, however, with the HIV infection so widespread, ANUG has become widely recognized as a lesion which is strongly pathognomonic of the infection, especially when seen in healthy looking young adults. Reports of the prevalence of ANUG among HIV infected patients vary between 4.3% and 16.0% [18]. In developing countries, like those in Sub-Saharan Africa, ANUG is often seen in poor children with malnutrition and infectious diseases such as measles and herpes simplex infection [19]. In Nigeria, reports indicates that the incidence of ANUG is increasing among children

with a prevalence as high as 23% in children under 10 years of age [18].

Clinical features and management of oral diseases that constitutes periodontal emergencies

Traumatic lesions

Oral traumatic lesions are acute lesions which are of sudden onset and with well-defined clinical features. Oral traumatic lesions can impair patients' normal oral function and cause pain during mastication. Oral traumatic lesions are more common in men than women (2.7:1). They are seen on the mucobuccal folds and gingiva. They can also be seen on the lower lips as in the case of lip biting after inferior alveolar nerve block [3, 20].

Oral traumatic lesions include physical and chemical oral injuries. Physical injury can be mechanical or thermal. Mechanical injury can result from accidental tooth bite, hair grip, writing materials as in pencil or pens pushed into the mouth, carelessly wielded toothbrush or wood stick, sharp piece of food such as a fish-bone and dental treatment such as orthodontic treatment due to irritation of braces or appliance wires. Thermal injury results from hot food and drink, cigarette burn etc. Chemical injury includes aspirin placed against the gingiva to alleviate toothache, escharotics such as silver nitrate, even hydrogen peroxide and sodium hypochloride used too strong. Careless use of a caustic by the dentist such as phenol, trichloroacetic acid can also cause oral mucosa chemical burn. Oral traumatic lesions can be inadvertent (accidental or iatrogenic) or deliberate (self-inflicted) [3, 21].

Usually there is little doubt about the diagnosis as the patient is aware of the accident. Traumatic injury to the oral mucosa causes pain and discomfort to patients and may be followed by several days of soreness and sensitivity to further irritation. A localized area of inflammation and ulceration may form. In the case of oral chemical burn, there may be vesicle formation followed by ulceration. The ulceration is seen as a bright red area denuded of epithelium and with rough edges which can be felt by the tongue. There is peripheral keratosis of mild to severe degree associated with the ulcer. The bottom of the ulcerative lesions is covered by whitish or yellowish pseudomembrane. Healing is rapid, within 1-2 weeks, unless there is secondary infection, especially in debilitated patient. With secondary infection, the wound may suppurate and may be accompanied by lymph gland enlargement and malaise. Abscess formation may follow damage by a piece of wood stick or bone if the foreign object is not removed.

Frequently, the wound heals without active intervention. Patients should avoid irritant foods and drinks. Cold water or warm saline mouth rinse help in relieving pain. Bonjela gel or topical corticosteroid preparation, such as 0.1% triamcinolone may be helpful in cases that are painful and persistent. Analgesics such as paracetamol can be prescribed. If there is secondary infection, an antibiotic may need to be prescribed.it can be helpful to protect the wound with a bland dressing such as carboxymethylcellulose gelatin paste (orabase), which is gently spread over the wound several times a day [3, 22, 23, 24].

Gingival abscess/Periodontal abscess

Gingival abscess is an abscess confined entirely to the gingivae. It is often associated with physical damage to the gingival margin by a wood stick; fish bone etc. with subsequent infection of the wound but it can also arise within the wall of the gingival pocket where drainage has been impeded [3]. The abscess appears as a localized, shiny red swelling which is painful; associated teeth are sensitive to percussion. The abscess may discharge spontaneously or spread into the underlying tissue to form a periodontal abscess. Periodontal abscess is described as a localized accumulation of pus within the gingival wall of a periodontal pocket. Periodontal abscess can develop de novo in patients with active periodontal disease i.e directly related to periodontal disease (periodontitis related) [8]. Blockage or obstruction of periodontal pocket by calculus accumulation, dislodged calculus during debridement or foreign body impaction has been reported to be the etiologic factor for periodontal abscess [8]. The obstruction impedes the clearance of gingival crevicular fluid thereby causing an accumulation of bacteria. Local factors that increase the risk of a periodontal abscess include invaginated tooth, root grooves cracked tooth, external root resorption and use of antimicrobials without mechanical debridement in patients with periodontal disease has also been linked to periodontal abscess [25]. Use of antimicrobials without mechanical debridement in patients with periodontal disease is believed to cause a change in the subgingival bacteria, resulting in a super infection [26]. Other factors that predispose to periodontal abscess include endodontic-periodontal lesions and altered

host response as in diabetes mellitus [3]. Gram negative anaerobic bacteria, commonly *Porphyromonas gingivalis* is the predominant microorganism in periodontal abscess [25].

The onset of symptoms in periodontal abscess can be sudden with pain on biting and a deep throbbing pain. The involved tooth may feel high and mobile. The overlying gingiva becomes red, swollen and tender. Pus becomes present and may discharge into the periodontal pocket.

If the cause of gingival abscess is still present, it should be removed carefully. The abscess can be incised, if it is pointing under local anesthesia and warm saline mouth bath every 3 hours and systemic antibiotic prescribed. Any residual pocketing can be removed by thorough subgingingival curettage or localized gingivectomy. For periodontal abscess, in addition to the aforementioned treatment modalities, the periodontal condition of the whole mouth must be treated by thorough subgingival scaling and root debridement and appropriate periodontal surgery where necessary. Endodontic-periodontal lesions would require root canal treatment prior to periodontal surgery being carried out [3].

Aphthous ulcer

Aphthous ulcer, also known as canker sore, is of unknown etiology that usually affects the keratinized or non-keratinized oral mucosa. It is one of the most common clinically obvious oral ulcerations and is among the oral mucosal conditions that dentists and physicians see most commonly in their clinic [27]. Studies have reported a predilection for females with onset of aphthous ulcer between the 2nd and 3rd decade of life [28]. There are three types of aphthous ulcer: minor aphthous ulcers, major aphthous ulcers and herpetiform ulcers. Minor aphthous ulcer has been reported to be the more common type of aphthous ulcer [3]. Minor aphthous ulcer occurs on non-keratinized oral mucosa, especially the lips, cheeks, vestibule and margins of the tongue. They are shallow ulcers less than 10mm in length with a surrounding zone of inflammation and slight swelling. Major aphthous ulcer are much less common than the minor variety and are larger (up to 30mm) in size. They can be found anywhere on the oral mucosa: keratinized or non-keratinized and tend to heal with a scar. Herpetiform ulcers occur as a group of pin-head ulcers which may coalesce to form larger painful ulcers, they can occur on any part of the oral mucosa, including the tongue, palate and oropharynx in which case they cause dysphagia (discomfort or pain on swallowing). Aphthous ulcer in all its forms presents as painful lesions which appear without reason, last for several days or weeks, heal and then after a variable interval recur. The ulceration is seen as a well circumscribed area denuded of epithelium with a whitish or yellowish floor. The margin of the ulcer is reddish or erythemathous [3, 29].

The cause of aphthous ulcer is unknown but it is thought to be due to manifestation of autoimmunity to a component of the oral mucosa, local trauma, hormonal changes, stress, iron deficiency anemia or nutritional deficiency such as folic acid and Vitamin B12 [3, 29]. Aphthous ulcer, like all other acute lesions, impairs patients' normal oral function and disturbs patients' daily activities [3, 29, 30]. Aphthous ulcer could occur in apparently healthy individuals, or it could be present in individuals with an underlying recognizable clinical condition as in Behcet's disease and celiac disease [31].

The clinical diagnosis of aphthous ulcer is based on characteristic clinical appearance [29]. laboratory investigation to rule out predisposing factors such as iron deficiency anemia, deficiencies in folic acid and vitamin B12, hormonal changes as well as genetic analysis may be necessary to confirm the diagnosis [31, 32].

Treatment for all aphthous ulcers is symptomatic and usually depends on the ulcer frequency ans severity [3]. In some cases, treatment may be unnecessary but where painful topical anaesthetics such as Bonjela, antimicrobial or corticosteroids preparation such as 0.1%triamcinolone may be beneficial [3, 33]. Systemic corticosteroids may be necessary in severe cases, especially in cases where there are no underlying nutritional or endocrine disorders. 0.2% chlorhexidine mouthwash may be useful in patients who may have difficulty in keeping the mouth clean [3, 34].

Erythema multiforme

Erythema multiforme (EM) is an acute, self-limited inflammatory mucocutaneous disorder characterized by distinctive target lesions. It's an immune mediated condition [35]. In about one-third of cases, the condition is recurrent [36]. It is a syndrome of multiple etiologies, with a wide spectrum of clinical features. Such etiologies include allergy to drugs like sulphonamides, penicillins and barbiturate and *Mycoplasma pneumonia*. In many cases, the etiology

is idiopathic. It usually affects young adults between 20-40 years of age. Males are more usually affected than female [37].

EM is conveniently separated into EM minor and EM major. The major form of the disease produces systemic involvement while the minor form produces local manifestation only. In the major form, there is a skin eruption with conjunctivitis along with lesions of the mouth and upper respiratory tract. The patient becomes progressively ill over the next 1-2 weeks with fever and malaise. In the mouth, there is diffuse inflammation of the oral mucosa and gingivae, there are widespread erosions on the mucosa and these have a red velvety base and bleeds freely. Some vesicles may form. The lips are severely involved with extensive crusting. Eating, talking and oral examination are painful. On the skin, there is extensive erythmathous and macular rash. Iris target lesions with a central bulla which breaks down to crust may be seen. The hands, feet and flexural surfaces are most involved. There is diffuse conjunctivitis which can become secondarily infected to produce corneal ulceration. The upper respiratory tract is often involved with epistaxis, dysphagia and tracheitis. In the minor form, there are only local manifestations in the mouth or the skin or both and no fever, weakness or exhaustion [3, 37, 38].

Diagnosis of erythema multiforme is clinical and requires careful differential diagnosis to rule out such diseases as lupus erythamatosus, Steven-Johnson syndrome, toxic epidermal necrolysis, vasculitis etc[37].

In the minor form, topical corticosteroids may be used in the mouth. In the major form, systemic steroids and supportive treatment are necessary. The patient should be referred to a physician. If Mycoplasma pneumonia infection is present, a course of tetracycline is given [3, 38].

Contact hypersensitivity

Contact hypersensitivity have been reported a variety of substances including chewing gum, mouthwashes, toothpaste, sweets, cosmetics, topical antibiotics, dental materials e.g gold, acrylic, periodontal dressing, orthodontic wire and amalgam; flavoring agents e.g peppermint, menthol, cinnamon, eugenol etc [3].

Contact hypersensitivity occurs when substances of small molecular weight (haptens) penetrate the skin or mucosa (Fig. 9). In a sensitized subject, a hapten combines with mucosal protein to form an antigen. These small molecules become bound to the surface of Langerhans' cells in the epithelium which present the potential allergen to T lymphocytes in regional lymph nodes. The antigen presenting cells then release interleukin-1 by specific delayed hypersensitivity T-cells, thus inducing further cytokine and interleukine-2 release from lymphocytes. This promotes clonal expansion of T cells and their migration to the mucosa via the efferent lymphatic system and mucosal capillaries. In 1-2 weeks a sensitized person can respond to re-exposure to the antigen, causing cytokine release and thee recruitment of inflammatory cells that initiate a local delayed- type hypersensitivity reaction at the site of contact. Histologically, these reactions manifest as inflammatory changes including intercellular edema and vesiculation of the epithelium. Chronic inflammatory response consisting of primarily lymphocytes, mast cells, basophils, neutrophils and eosinophils in the connective tissue may also be evident [39, 40].

PROPOSED MECHANISM FOR CONTACT HYPERSENSIVITY

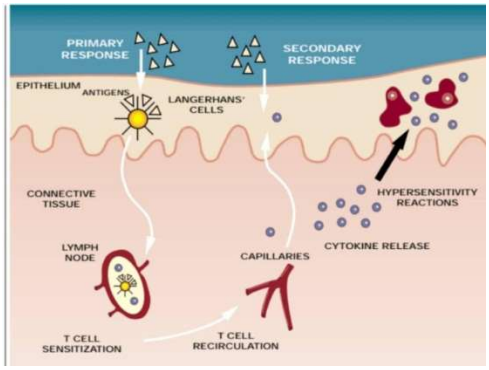

Figure 9

Picture is adapted from De Rossi and Greenberg [39].

Clinically, symptoms start with a burning sensation of the oral mucosa and swelling and redness of the tongue, lips and gingivae. The epithelium may peel off to leave very sore ulcerated areas. The gingivae are characteristically bright red and sensitive and because the patient cannot clean the mouth it can become very dirty [3].

Signs and symptoms of contact allergic reactions affecting the oral mucosa may mimic other common oral disorders like oral traumatic lesions, making diagnosis difficult. Epicutaneous patch testing, together with the clinical manifestations; constitute the most widely used diagnostic approach in such situations [41].

In contact hypersensitivity, the drug or chemical suspected must be immediately withdrawn. Antihistamines, such as Piriton are useful where symptoms are mild but more severe reactions e.g angioneurotic edema, may require injection of hydrocortisone hemisuccinate.

In anaphylactic shock, intramuscular injection of 0.5ml of 1:1000 adrenaline (epinephrine) is necessary. The mouth can be kept clean by frequent warm water or weak saline mouth rinses [3, 42].

Acute Herpetic Gingivostomatitis (AHG)

Primary infection by herpes simplex virus (HSV) type 1 usually occurs in children (1-10) but may affect older children or adults. The virus is transmitted by infected saliva or skin lesion contact. Infection in neonates can produce encephalitis or meningitis but in children or adults it produces either a febrile illness or subclinical infection. The incubation period is about 5 days. Symptoms appear abruptly with mild to severe fever. Temperatures may be raised as high as 39.4°C. There is lymph gland enlargement and malaise and the mouth and throat may be very painful. In young children there is irritability, profuse salivation and refusal to eat, even before the oral lesions become apparent. Small vesicles form on the gingivae, the tongue, buccal mucosa and lips, in fact anywhere in the mouth. Usually, the vesicles burst before they are seen and the resultant round or irregular ulcers form a grey membrane surrounded by bright red mucosa. There is acute gingivitis with redness, swelling and bleeding. This tends to be more severe in older patients with this condition. Symptoms subside in 10-21 days as the titre of protective antibodies rises [3]. Large proportions (30%) of patients who have had a primary herpetic infection early in life develop recurrent infection years later. The commonest recurrent lesion is on the lip or the adjacent skin up to the nostril (herpes labialis, fever blisters or cold sores) [3]. Herpes labialis is a bothersome, large, painful and disfiguring lesion which is preceded by an itching and burning sensation. From initial manifestation to complete healing between 7-10 days, occasionally 14 days, it has 5 clinical stages: Prodromal, blister, weeping, scabbing and healing [43]. The blisters occur as a result of reactivation of latent virus in the trigeminal ganglion. This can occur as a result of any infection which lowers the resistance or dries the skin or as a result of excessive exposure to sunlight.

Herpes labialis is contagious for the previously uninfected individuals and those with compromised immune systems such as HIV-infected individuals and those undergoing chemotherapy [44]. The clinical diagnosis of herpetic infection is based on case-specific history taking, characteristic clinical appearance and the location of the lesions [45]. However, confirmatory laboratory diagnosis is necessary in form of viral culture, polymerase chain reaction, serology, direct florescent antibody testing or Tzanck test.

The treatment of the oral infection is largely symptomatic and supportive i.e bed rest, cool, soft food and plenty of fluid. In infants, milk of magnesia or 55% Dequadin paint may be gently applied on cotton wool to the lesions. Benzocaine lozenges are useful in the older child or adult. Ibuprofen or paracetamol will help to reduce pain and temperature. Phernegan is a useful sedative in the child. In severe cases, acyclovir tablets (200mg 5times daily for 5 days or suspension (5ml, 5 times daily for 5 days) may be prescribed [3]. Acyclovir has been reported to be effective in decreasing the severity and duration of herpetic episodes and contributes to the prevention of recurrence of herpes labialis [46]. Other antiviral drugs e.g Valacyclovir, famciclovir and penciclovir may also be prescribed. Topical acyclovir (apply 5 times daily for 5 days) may also be used as a preventive measure for herpes labialis [3]. However, the value of antiviral therapy in the management of herpes labialis in immunocompetent patients remains a contentious issue [46].

Acute Necrotizing Ulcerative Gingivitis (ANUG)

ANUG, also known as Vincent's disease, is an acute necrotizing inflammatory disease produced by

endogenous infection, in particular spirochaetes and fusiform bacteria [3].

In developing countries as in Africa, environmental factors are entirely responsible for this situation because it often occurs in poor malnourished children as well as in children with infectious diseases such as measles and herpes simplex infections [19]. In developed countries, the condition is a disease of young adults and occurs equally in both sexes. It appears to be seasonal, occurring most frequently in autumn and winter months. The condition rarely occurs in a clean mouth and then only if there is a major predisposing factor [3].

Predisposing factors for ANUG include poor oral hygiene, smoking, emotional stress, blood dyscrasias such as acute leukaemia, infections such as AIDS, malignant neoplasms, chemotherapy and indeed any condition in which the immune system is compromised [3, 18, 19].

The condition is very painful and plaque accumulates around the affected areas. Patients complain of gingival soreness which is sometimes severe and eating becomes difficult. There may be spontaneous gingival bleeding, an objectionable taste and a powerful halitosis. The disease is characterized by necrotic ulceration of the affected gingival margins. In the early stages of the disease the gingival papillae becomes red and swollen and the tips of the papillae becomes ulcerated. Necrotic ulceration of the papillae increases and the ulcer may spread laterally along the gingival margins. The ulcers are painful to touch and are covered by a yellowish-grey slough. They have a characteristic 'punched out' appearance and if the 'false membrane 'of sloughing tissue is removed a raw and bleeding surface is exposed. The ulceration may be localized to one area or involved the whole mouth. Localized infections are most often seen around the lower anterior teeth. They may also be related to sites of bacteria stagnation such as partially erupted lower third molar. There are frequently no systemic symptoms, although cervical or submandibular lymphadenopathy is commonly present. In some severe cases, there may be mild to moderate fever and malaise and more marked lymphadenopathy. When ANUG occurs in association with HIV infection the lesion may spread more deeply and lead to exposure and infection of the underlying bone [3, 47, 48].

The diagnosis of ANUG is easily made on clinical grounds without the need to take a bacterial smear to show the fusospirochaetal flora. Nevertheless, it is important to take a very careful history to determine the underlying predisposing factor in each individual case [3, 48].

Without treatment, the acute symptoms may subside and heal within 10-14 days with a residual saucer-shaped deformity of the gingiva. Once an episode of ANUG has occurred, there is a tendency for recurrence and in a susceptible individual this can occur more than once a year with progressive destruction of the periodontal tissues.

The acute phase of ANUG is managed by cleaning the wound with warm water or 5-volhydrogen peroxide solution three times daily and scaling of the teeth carried out over the next few days. The patient is prescribed oral Metronidazole 200-400mg 8hourly for 5 days, Amoxicillin 250-500mg 8hourly for 5 days. Erythromycin and clindamycin may be used if both Metronidazole and Amoxicillin are contra-indicated. A 2%chlorhexidine mouthwash used for a short period if tooth brushing is compromised, may be useful. Meticulous and deep scaling as well as maintenance of a high standard of oral hygiene need to be carried out after control of acute phase to prevent recurrence. Any residual gingival deformity needs to be corrected by periodontal surgery.

Patients who suffer from unexplained recurrence should undergo medical examination and blood screening for major predisposing factors [3, 48, 49, 50].

Level of awareness of oral health in general

Oral health is integral to general health of an individual [51]. People however, are unaware of its importance. Erhabor and Ikpefan reported on a number of reasons for the low level of dental awareness including cultural practices, low socioeconomic status (educational and income level), lack of enlightenment campaigns in both print and electronic media as well as few available oral health facilities, both public and private dental clinics [52].

Despite the fact that dental care is presently a component of primary health care, the first level of contact in the national health systems in Nigeria is usually the secondary health care centers. These secondary health care centers are mostly sited in state capitals and some cities in the country.

It has also been reported that adequate knowledge by health workers at all levels of health care will enable them to properly educate patients on good dental care and make appropriate referrals to the dentists [53].

The proposed solution to low oral health awareness include: Increase enlightenment campaign and improve government policy and implementation in the area of oral health care. Good interpersonal and work relationship between the dental care practitioner and other medical colleagues on the one hand and patients on the other hand is advised to improve oral health awareness by individuals, people and community at large.

CONCLUSION

The importance of prompt management of oral diseases that constitutes periodontal emergency cannot be over emphasized as it has been shown to greatly affect the general well-being and health of the individual. Periodontal emergencies, when not timely managed, can lead to residual destruction of periodontal tissues and indeed the oral cavity which can affect the quality of life of the individual. A healthy mouth in a health body is one to be sought by all.

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