Schematic outline of Periodontal diseases, which includes causes and treatment approaches

ABSTRACT

A severe inflammatory response that eventually destroys the underlying tissue characterizes periodontal diseases, which have a mixed microbiological origin.Several local or systemic risk factors can impact the appearance and progression of periodontal diseases.Although it can affect infants and teenagers as well, periodontal disease is more common in adults. The degree of tooth plaque development and gingival tissue degradation determines the prevalence of periodontal disorders. One of the most important characteristics of severe and persistent periodontitis is site specificity. The degree of attachment loss and tooth bone loss determined by the depth of the periodontal pocket determines the severity of this illness.This review article discusses the aetiology, diagnosis, medical management, and pathophysiology of periodontal disease.

Keywords:Periodontal disease, Histopathology, Epidemiology,Etiology, Pathophysiology, Medication.

INTRODUCTION

All supporting tissues of teeth, including gingiva, periodontal ligament, cementum, alveolar bone, and so on, are affected by periodontal diseases, which include a broad spectrum of inflammatory illnesses that degenerate the periodontium and ultimately result in tooth loss(1).An infection of the periodontium is called periodontitis. In contrast, the terms "dont" and "itis" denote teeth and inflammation, respectively, and "perio" refers to the gingiva and other tissues surrounding teeth. Thus, "periodontitis" refers to long-term dental cementum, alveolar bone, and gingiva inflammation. The World Health Organization (WHO) states that it is a chronic illness that spreads widely around the globe(2,3).There are two primary types of periodontal diseases, which are characterized as inflammatory conditions brought on by pathogenic microorganisms arranged in biofilms around the teeth. Periodontitis, on the other hand, corresponds to a deeper condition linked to the deterioration of tissues supporting teeth and can result in tooth loss. Gingivitis is a superficial and reversible infection of the gingiva without destruction of the alveolar bone. The aetiology of periodontal illnesses is closely associated with periodontal bacteria, including Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, and

Prevotella intermedia(4,5).It has been determined that systemic diseases, ranging from ageing, smoking, and psychological stress are significant risk factors for periodontitis(6,7).Severe periodontitis affects 5–20% of adult populations worldwide and can lead to tooth loss.Any of the numerous types of periodontitis, including aggressive, chronic, and those that occur as a symptom of systemic disorders, can affect children and adolescents.It is now widely accepted that nearly all types of periodontal disease are caused by mixed microbial infections, in which particular harmful bacterial populations coexist(8).As the most common type of periodontal disease, gingivitis affects up to 90% of people. This word refers to the inflammation of the gingiva brought on by the build-up of dental plaque, or germs and debris between the gum line and the tooth. Gingivitis is a reactive illness that can be treated with better dental hygiene. When gingivitis is no longer the only periodontal condition present and has developed into a damaging, irreversible, and chronic inflammatory disease state, it is called periodontitis. At that point, the bacteria have a greater opportunity to enter the surrounding periodontium and tissues. This sets off the host's defense mechanism against the invasive microorganisms. Nevertheless, the host's defenses against the germs also cause the periodontium to be destroyed in the process.Periodontitis causes the periodontium to lose its connection, which then causes alveolar bone loss and may lead to the loss of the afflicted tooth(9–11).A diagnosis is the acknowledgement that a condition exists. The identification of diverse indications and symptoms in the periodontal tissues that signal a decline in health is the first step towards a clinical diagnosis of periodontal disease. A thorough understanding of periodontal health is necessary for the diagnosis of periodontal disease. Only the gingival tissues of the healthy periodontium can be directly seen, and they are described as being stippled, pale pink or coral pink in Caucasians, and varying degrees of pigmented in other ethnicities. It is closely tailored to the underlying tissues, and when it meets the tooth, it has a knife-edge margin. When there is no pathology, the gingival edge is found at the cemento-enamel junction.It has a scalloped edge arrangement that is lowest buccally and lingually and highest interdentally, where it forms the interdental papilla. Where it borders the tooth, there is a 1-3 mm deep gingival fissure. When gently prodded, there is no blood from the crack. A tiny amount of gingival crevicular fluid, or interstitial fluid, will be visible in the health crevice.The free gingival margin is formed by the lateral wall of the crevice. The attached gingival, whose breadth ranges from 1 to 9 mm and has a stippled surface, extends from the most apical extent of the free gingival to the mucogingival junction. This mucoperiostium, which is a keratinized mucosa that is well-suited to withstand damage, is an immobile tissue that is firmly attached to the bone(9,12–14).Periodontitis, often known as periodontal disease, is the primary term for the ailment that causes a space known as a "periodontal pocket" between the gingiva and the tooth. The development of microbial plaque determines the disease's severity. Many techniques have been used for this disease's screening and assessment, leading to the determination of the periodontal ligament's degree of severity. There are numerous techniques available to diagnose periodontal disease, including tissue engineering, hematological screening, laser treatment, and radiography. Depending on the disease's chronology, a variety of therapeutic options-both surgical and non-surgical-are available to slow the disease's advancement. Maintaining proper dental hygiene and providing intensive care are the two main ways in which this disease is maintained(15–17).

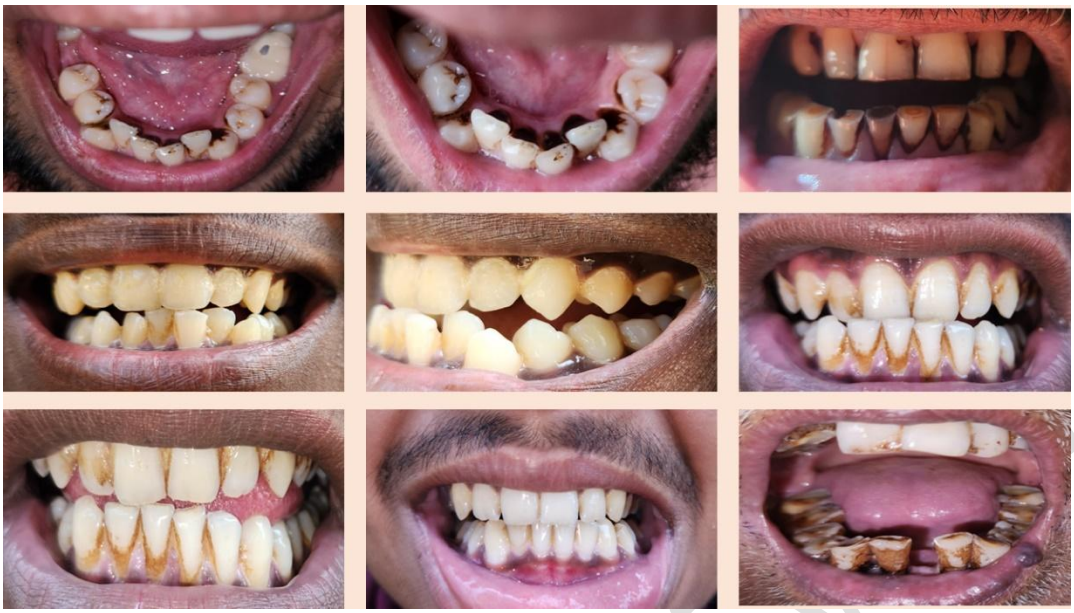

Fig.1:Periodontal diseases.

HISTOPATHOLOGY

The histology of periodontal disorders was initially characterized by Page and Shroeder. The progression of the disease occurs in four separate stages, each of which considers the tissue's histological and clinical appearances. The first lesion is identified by a plaque that causes vascular alterations and the production of intercellular gaps, which raises the quantity of gingival crevicular fluid (GCF). Polymorphonuclear neutrophils are drawn to the lesion site by adhesion molecules. T cells particularly change the local fibroblast population. Clinically, the lesion is benign at this point. It is referred to as the early lesion as redness appears there. PMNs (polymorphonuclear neutrophils) invade the region and eliminate the apoptotic fibroblasts.Collagen fiber disintegration brought on by the invasion also creates more room for infiltrates. The matrix of marginal connective tissue is degrading. Leukocyte aggregation and B cells, either plasma cells or lymphocytes, are the main factors influencing the formed lesion. They start the site's transformation by converting the junctional and sulcular epithelium into the pocket epithelium. The pocket epithelium is delicate and highly porous. Clinically, this shows up as bleeding when the gingival tissues are gently prodded. The last phase is a transition to periodontitis and is referred to as an advanced lesion. The migration of biofilm to the pocket produces the advanced lesion and provides a perfect environment for the growth of anaerobic bacteria.Histologically and clinically, there is an irreversible loss of connection and bone loss. This stage is characterized by the loss of alveolar bone and gingival fibers. The microbial components themselves have a significant influence on this lesion, which can result in various modifications based on the organism and host(18,19).

EPIDEMIOLOGY

Periodontal disease is the most prevalent dental disease worldwide, affecting up to 90% of the population. According to cross-sectional studies conducted in the United States alone, up to 80%

of adults have had periodontal disease at some point in their lives, and about 50% of persons presently have gingivitis. It has been demonstrated that some groups are more likely to develop periodontal diseases. These demographics include men, African Americans, and the elderly. A severe case of periodontitis was also linked to lower levels of education and income(1,20).Comprehensive nationwide data on the prevalence of periodontal disease are provided by the nationwide Oral Health Survey of India (2002). Nevertheless, there isn't a yearly or five-yearly oral health survey conducted in India to actively track the prevalence of disease. Furthermore, periodontal epidemiological approaches have not proved reliable, in contrast to tooth caries. Changes in measurement instruments, probing sites, the amount of oral cavity analyzed, and the kinds of probes employed by oral epidemiologists are the causes of this discrepancy. Additionally, because periodontal disease is a chronic condition and surveys are expensive, it is not possible to regularly monitor the condition. The combination of these variables is to blame for the dearth of sufficient information and, consequently, the low priority of oral health initiatives(21).

ETIOLOGY

A number of variables, including both patient-specific risk factors and poor oral hygiene, can lead to periodontal disorders. One can categorize the risk factors into two groups: those that can be changed, such as smoking, oral hygiene, diabetes mellitus, pregnancy, and age; these that cannot be changed, such as inheritance and genetic illnesses. The onset and progression of periodontal diseases are significantly influenced by poor oral hygiene practices. Bad dental hygiene habits can cause germs and plaque to accumulate on teeth, which can cause gingivitis and eventually lead to periodontitis. Studies have shown that there is a clear correlation between the severity and prevalence of periodontal illnesses and the amount of dental plaque that accumulates on teeth.Insufficient dental hygiene can allow the anaerobic microorganisms that cause periodontal illnesses to spread to deeper parts of the periodontium, where they can do their damaging work. Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Treponema denticola, and Tannerella forsythia are the principal bacteria implicated in periodontitis. These organisms cause inflammation when they are let to delve far into the periodontium because they cause the host to release inflammatory mediators and other defensive substances(22–25).Tobacco smoking is the most notable significant modifiable risk factor for periodontal disorders. The odds ratio between smoking and chronic periodontitis is 5.4, meaning that it can raise the risk of periodontal disorders by a factor of 5 to 20. Furthermore, compared to non-smokers, tobacco users had higher rates of bone loss, attachment loss, deep periodontal pockets linked to the illness, and tooth loss. Tobacco smoking is linked to a marked decline in treatment efficacy in addition to a heightened severity of periodontal disorders.Another significant risk factor for periodontal diseases is diabetes mellitus. This illness is linked to specific pathologic processes, like slowed wound healing, that promote periodontal degradation. The complications category contains more relationships between diabetes mellitus and periodontal disorders. Compared to individuals without or with mild periodontal disease, those with diabetes mellitus who have severe disease have a higher chance of dying(25–32).Hormone changes brought on by pregnancy have been demonstrated to incite an inflammatory response that is connected to periodontitis and gingivitis. Maternal hormones have been demonstrated to

positively link with Porphyromonas gingivalis levels, a crucial bacterium in the advancement of periodontal disease, for reasons that are still unclear. It has been demonstrated that hyper- and hypoestrogenism both contribute to gingivitis.Age is a risk factor for periodontal diseases that cannot be changed and has been extensively studied in the literature. It has been demonstrated that older people respond to plaque deposition with a more severe inflammatory response that includes more inflammatory cells. The deterioration of the periodontium is more likely to occur in elderly people due to this inflammatory cell accumulation. Furthermore, because aging is linked to a decline in dexterity, oral hygiene practices are typically less proficient in older adults. Higher levels of plaque are the consequence, and this is a known risk factor for the emergence of periodontal disorders. Moreover, studies have shown that those 60 to 90 years old have higher clinical attachment loss (CAL) than people under 5050 years old.Finally, it has been demonstrated that a number of genetically associated systemic disorders can present as periodontal diseases. The research has also shown the origin of the development of periodontal diseases within these systemic disorders. These conditions include Crohn's disease, Down syndrome, and Ehlers-Danlos syndrome (types IV and VIII)(33–42).

PATHOPHYSIOLOGY

The disease is initiated and spread by commensal oral bacteria through a process known as dysbiosis, or microbial imbalance. Up until a treatment measure is implemented or the disease process destroys the tooth and associated structures, perhaps leading to the loss of the tooth, the disease progresses cyclically with periods of activity and quiescence.An increasing number of anaerobic bacteria, including Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans, occupy deeper periodontal pockets as periodontal disease advances from gingivitis to periodontitis, inciting an inflammatory response in the host. Tumor necrosis factor-alpha (TNF-a), matrix metalloproteinases (MMPs), interleukins (IL-1 and IL-8), and other neutrophil and macrophage chemicals are produced and distributed during this reaction. C-reactive protein (CRP) is a biomarker of inflammation. An increased level of CRP in the serum indicates a possible correlation between cardiovascular pathology and the inflammation caused by periodontitis. Furthermore, smoking accelerates the progression of the illness by creating an environment that is more conducive to the proliferation of periodontal bacteria(10,23,38,43,44).

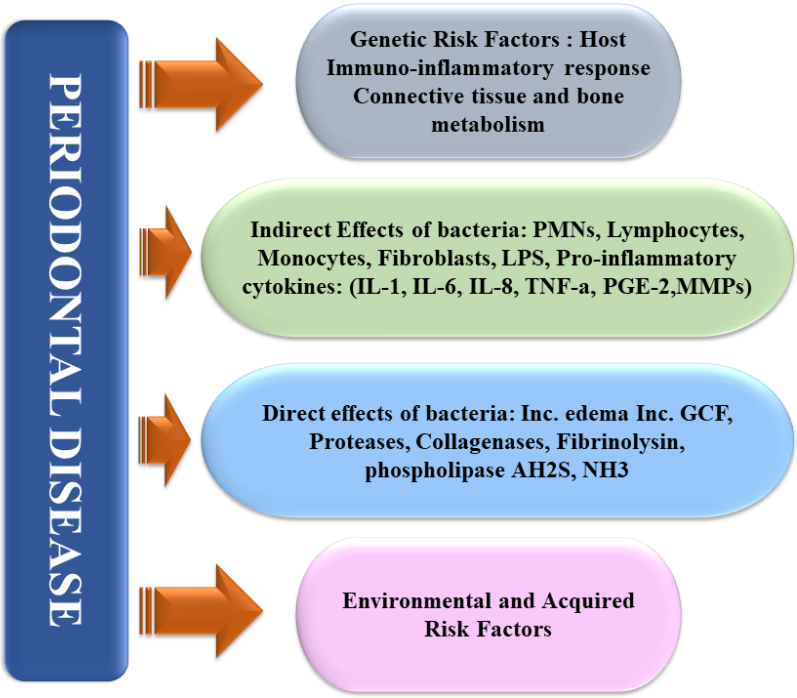

Fig.2:Pathophysiology of thePeriodontal Disease.

CHALLENGES ASSOCIATED WITH PERIODONTAL DISEASE

Tooth loss is one of the most well-known and immediate effects of periodontal disease. As the disease progresses, the periodontium-which includes the alveolar bone and periodontal ligament that typically support the teeth-becomes more and more destroyed. Nevertheless, studies have demonstrated a connection between periodontal disease and a number of systemic illnesses, including diabetes mellitus, cardiovascular disease, and pregnancy difficulties such premature low birth weight babies.It has been demonstrated that those with diabetes mellitus have a higher chance of developing periodontal disease. Patients with severe periodontal disease and type 2 diabetes mellitus are projected to be 3.2 times more likely to die than those with diabetes without periodontal disease. Enhanced collagenolytic activity leading to an accelerated periodontal disintegration and compromised wound healing and host responses are some of the hallmark pathological mechanisms of this relationship. The increased severity of these symptoms is also linked to poorly controlled diabetes. Given that periodontal disease has been linked to hyperglycemia, poor glycemic control, and decreased glucose tolerance, it is possible that periodontal disease could be the initial cause of insulin resistance.Moreover, the hyperglycemia that this group experiences may facilitate the growth of the bacteria that cause periodontal disease, making it more severe in diabetics(22,38,45–48).Cardiovascular illness is linked to periodontal disease. As was previously established, people with periodontal disease have been shown to have higher levels of C-reactive protein (CRP), a critical indicator of inflammation. Additionally, there is a correlation between CRP and cardiovascular events and disease. Atherosclerosis and the bacterial level present in periodontal disease are directly related. But so far, no causal connection has been proven.Preterm low birth weight babies are another serious consequence linked to periodontal disease. The presence of periodontal disease in mothers and

the birth weight of their children are significantly correlated. Previous studies have demonstrated a tendency for newborn birth weight to decline when periodontal disease progresses from none to severe(38,49–57).

DIAGNOSIS

Comparing results to a normal periodontium is necessary for the identification of periodontal disorders. This comparison makes use of radiographic bone levels assessment, periodontal probing, and ocular inspection. Stippled, pale pink gingiva that is perfectly matched to the underlying bone makes up the normal periodontium. There is a 1 to 3 mm physiological sulcus between the gingiva and the tooth, which is generally not bleeding. In contrast, symptoms of periodontal disease include pain, foul taste or odor, radiographic bone loss, clinical attachment loss, periodontal pocketing, and active bleeding in response to minimal or no tissue stimulation.A patient with periodontitis will have alveolar bone loss near a deep periodontal probing depth on their radiographs. These results imply that the periodontal bacteria that are actively inducing the host response are located in a deep periodontal pocket connected to the tooth. The related tooth will become mobile and finally fall out if the bone loss is not corrected, leading to insufficient tooth support(9,10).

- **Vitality test**

To determine a tooth's pulp vitality, an electric pulp tester or thermal stimuli are utilized(58).

- **Radiograph**

The prognosis of patients is determined by periapical radiography, bitewing radiography, panoramic X-ray, or by a combination of all of these. A radiograph gives specific details on the patient's dental health. A radiograph can be used to determine the pattern and quantity of bone loss as well as the degree of bone loss and depth of the periodontal pocket(59).

- **Additional tests**

Comprehensive hematological evaluation. a test for blood glucose. Monetary plaque sampling, or INR(60,61).

PERIODICTIONAL DISEASES: MEDICATION-BASED MANAGEMENT

A step-by-step method is used to treat periodontal disease, starting with more conservative measures. A professional dental cleaning, which involves scaling the teeth and root planning to remove dental calculus and plaque from both above and below the gum line, is the first step in treating any kind of periodontitis. The patient's at-home oral hygiene regimen is enhanced by the dental professional's oral hygiene instructions, which constitute a significant component of this dental cleaning. After the cleaning appointment, the patient needs to go back to the dentist for a reevaluation of their periodontal status. This entails an examination to evaluate how the periodontium is doing and to assess the depth of the probing to see if the disease process has been stopped.Periodontitis is a chronic ailment that might reactivate in the right circumstances, therefore even if the condition is verified resolved, the patient should still visit the dentist for routine cleanings. Treating risk factors is the primary step in managing periodontal disease.One

of the main causes of periodontal disease is poor dental hygiene. Promoting appropriate self-performed oral hygiene as well as periodic professional maintenance, contingent on each patient's risk, are key components in the prevention of bad oral hygiene practices. Brushing, flossing, and rinsing are the three daily steps in the self-care regimen that is advised. It's also advised to arrange follow-up appointments to track the disease's progression and to have your teeth professionally cleaned by a dentist(25,26).Tobacco smoking is a significant modifiable risk factor that also has to be addressed. In addition to being linked to a much higher chance of acquiring periodontal disease, tobacco use has also been demonstrated to have a more severe course of the disease and a notably reduced response to periodontal therapy. When smoking is stopped, the correlation between smoking and periodontal disease diminishes.There is ample evidence linking diabetes mellitus to periodontal disease, and it may exacerbate the damage caused by periodontitis. Furthermore, there is a connection between poor glycemic control and faster disease progression. Patients with severe periodontal disease are more likely to die from uncontrolled glucose levels. Consequently, in order to enhance the results of periodontal therapy, diabetes mellitus and prediabetes mellitus care may be necessary(26,32,62–64).Depending on the severity of the condition, antibiotics can be used both locally and systemically in cases of persistent periodontal disease that is resistant to non-pharmacologic therapy.A typical antibacterial substance used as an adjuvant to mechanical periodontal therapy is chlorhexidine gluconate. Although it is typically used as a mouthwash, it can also be applied as a gel, varnish, or subgingival chip. When combined with consistent brushing, chlorhexidine helps reduce dental plaque accumulation, which makes it an excellent treatment for chronic periodontitis. For the treatment of periodontal disease, medication has made some recent advances. After cleaning is finished, a chlorhexidine gluconate chip is put into the periodontal pocket to offer long-term, sustained-release of the compound into the afflicted area.Another choice is to utilize an adjuvant antibacterial agent after mechanical periodontal therapy. It is made up of microspheres containing minocycline hydrochloride that are inserted into the surrounding pockets after mechanical debridement. It works similarly to the chlorhexidine chip in that it efficiently lessens the accumulation of tooth plaque(65–68).Systemic antibiotics are rarely necessary, however they can be when deep periodontal pockets are persistent. The following are the most often prescribed antimicrobial agents: Nitroimidazole compounds, Quinolones, Macrolides, Tetracyclines, Penicillins, and Cephalosporins. These pharmacological drugs can be provided to patients with a variety of susceptible microorganisms, including those that are resistant to antibiotics, and have varying mechanisms of action. To increase their use even further, these medications might be prescribed separately or in combination(69–71).

- **Tooth brushing**

There are toothbrushes with manual and electrical functions for removing dental plaque. A study conducted in 2005 by Robinson et al. found that powered toothbrushes that rotate and oscillate are more effective in eliminating dental plaque(72).

- **Adjunctive pharmacological agent**

To boost the effectiveness of mouthwashes and toothpaste, numerous pharmaceutical ingredients have been introduced. Chlorhexidine gluconate is a commonly used medication that is regarded

as the gold standard for treating gingivitis and plaque. It is mostly added to toothpaste, gel, and mouthwash(73).

- **Interdental cleaning**

Only 65% of the tooth surface can be cleaned by an efficient toothbrush; therefore, interdental cleaning-which includes using dental floss, tape, and powered flossing devices-is also required to eliminate dental plaque overall. Dental floss and tape are recommended to patients when the interdental papillae are fully emaciated, since this improves the clinical results of periodontal therapy(74).

- **Antibiotics**

Antibiotics used topically or orally are used to prevent the growth of microbial biofilms. Topical antibiotics are injected into periodontal pockets or the gingival sulcus by implants, gels, etc. On the other hand, oral antibiotics eradicate infections brought on by germs on the surfaces of teeth and gums(75,76).

- **Scaling**

Scaling aids in the removal of microbial biofilms and calculus from the gums. It can be controlled by an ultrasonic device or by hand devices(77).

- **Root planning**

Root planning prevents more tartar accumulation while also smoothing the root surface. Additionally, it eliminates harmful byproducts to lessen inflammation and promote healing of the gums' adhesion to the tooth surface(78).

- **Supportive therapy**

This treatment is advised to maintain periodontal health and prevent disease recurrence. As part of this therapy, the patient will have routine check-ups to assess their periodontal condition and to retrain them in plaque control techniques and oral hygiene maintenance. Handling: Periodontal disease has the power to stop the growth of bacteria and regulate how the disease progresses. However, appropriate management along with the right treatments is necessary for periodontal disease therapy to be successful(79,80).Removal of supragingival and subgingival dental plaque is the first step in treating periodontal disease, which is followed by the healing of tooth loss.Typically, three months pass between treatments in order to manage periodontal disease's chronicity. The length of the maintenance phase has been adjusted based on the disease's severity. Since the goal of supportive therapy is to maintain the condition over the long term, appropriate steps are taken to increase patient compliance and slow the course of the sickness(81,82).

CONCLUSION AND FUTURE DIRECTION

Our review articles contain succinct information about the pathogenesis, diagnosis, and treatment options of periodontal diseases. While there are advantages to pharmaceutical drugs, they can also have negative effects such as heart failure and kidney damage. To learn more about the best course of action for treating periodontal disorders, more randomized controlled research are required. We plan to conduct a more thorough investigation of periodontal disorders. To assess patients' mental and physical health and provide a more comprehensive understanding of periodontal diseases and efficacious therapy, a second counseling study will be carried out in our country or state with the assistance of our colleagues.

ETHICAL STATEMENT

The goal of a discrete, thoughtful, and compassionate pharmacist is to enhance each patient's quality of life.

INFORMED CONSENT

Using websites, review articles, and other sources to produce research content.

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