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Post op instructions after extraction

If you need a large print, audio, braille, easy-read, age-friendly or translated copy of this page, please email uclh.patientinformation@nhs.net. We'll do our best to meet your needs. After the local anaesthetic wears off (in 2-6 hours), avoid hot foods and drinks as they may burn your mouth. Be careful while eating to prevent biting your lip or tongue. For 24 hours, don't rinse your mouth, spit, or touch the area where the tooth was extracted. For the first few days, it's best to have soft food like pasta, mashed potato, scrambled egg, or soup. From 24 hours after extraction, you can start cleaning your teeth as normal but be careful not to disturb the tooth socket. From 24 hours after extraction, use warm salty mouthwash: Dissolve two teaspoons of normal salt in a large cup of hot water and let it cool before rinsing gently at least 4 times a day, especially after eating. Continue with these rinses for one week or until the socket has closed completely (which may take a few weeks). If you need to take painkillers, avoid any containing Aspirin as this may cause bleeding. Don't exceed the recommended dose of painkillers and follow the directions if you were given antibiotics. Avoid smoking for at least 48 hours as it may delay healing and cause pain. Also, avoid strenuous exercise for 24 hours. If you have stitches, they will dissolve in a few weeks. It's normal to have some blood in your saliva for a few days after extraction, giving your saliva a red tinge. If the bleeding is heavy or doesn't stop, take the following steps: Gently rinse, apply gauze over the tooth socket, bite hard for 30 minutes, and repeat if necessary. If you experience severe pain or facial swelling outside of the expected time frame or have persistent bleeding, please contact us via the details below. Pain and swelling will increase for 72 hours after surgery but should start to settle afterwards. If your symptoms cannot wait for in-hours care, please contact as below in cases of EMERGENCY only: Tel: 111 (NHS advice service), visit your local Accident and Emergency service (A&E), or contact the On call maxillofacial team on 020 3456 7890 asking for the "Maxillofacial Senior House Officer on call, bleep number 5602." Looking at patient information videos about dental extractions, patients can learn more by watching our 'Dental extractions - information for patients' video below. Review Due: 01 February 2027 **###** Was this page helpful? Share – copy and redistribute the material in any medium or format for any purpose, even commercially. Adapt – remix, transform, and build upon the material for any purpose, even commercially. The licensor cannot revoke these freedoms as long as you follow the license terms. Attribution – You must give appropriate credit , provide a link to the license, and indicate if changes were made . ShareAlike – If you remix, transform, or build upon the material, you must distribute your contributions under the same license as the original. The free gingiva sits above the alveolar bone crest and is considered the most superior layer of tissues. In a healthy periodontium, the gingival margin marks the fibrous tissue that surrounds the cemento-enamel junction, where the enamel surface meets the outer cementum layer. A natural space called the gingival sulcus lies apically to the gingival margin, between the tooth and the free gingiva. This space is typically 0.5-3mm in depth but can increase with periodontal disease. The oral sulcular epithelium lines the gingival sulcus and begins at the gingival margin, ending at the base of the sulcus where the junctional epithelium and attached gingiva begin. The junctional epithelium forms a collar-like band at the base of the gingival sulcus, surrounding the tooth. It demarcates the areas of separation between the free and attached gingiva and provides a specialized protective barrier against microorganisms. Collagen fibers bind the attached gingiva to the underlying periodontium, varying in length and width depending on location and individual. The attached gingiva lies between the free gingival line or groove and the mucogingival junction. It dissipates functional and masticatory stresses during activities like tooth brushing, speaking, and mastication. In health, it appears as a pale pink or coral pink color with surface stippling or racial pigmentation. The interdental gingiva occupies the space beneath a tooth contact point between two adjacent teeth. It takes on a triangular or pyramidal shape, formed by two interdental papillae (lingual and facial). The central part is composed of attached gingiva, while the borders and tip are made up of free gingiva. The col, located at the center of the interdental papilla, is a valley-like depression that lies directly beneath the contact point. However, it may be absent with gingival recession or non-contacting teeth. The main purpose of the interdental gingiva is to prevent food impaction during routine mastication. This area is non-keratinized and located beyond the mucogingival junction, providing for movement of cheek and lips. It is less firmly attached and appears redder than attached gingiva. The periodontal ligament joins the cementum layer of the tooth root to the surrounding alveolar bone, composed mostly of collagen fibers. It also houses blood vessels and nerves within loose connective tissue. Mechanical loads during mastication are absorbed by this ligament. The alveolar bone surrounds the teeth, forming a socket that supports each one. This bony structure is composed of compact cortical bone and cancellous trabecular bone. The cementum layer overlies the tooth root, providing attachment for collagen fibers and protecting the dentine. Gingivitis is a common condition that affects the mucosal tissues surrounding the teeth, characterized by inflammation, swelling, and redness. It's typically painless and caused by plaque accumulation and poor oral hygiene. If left untreated, gingivitis can progress to periodontitis and other diseases that threaten overall health. Periodontal disease encompasses various conditions resulting in attachment loss and alveolar bone destruction. It's often caused by bacterial plaque biofilm accumulation combined with host immuno-inflammatory mechanisms and risk factors. Untreated, these diseases can lead to tooth loss. Healthy gingiva is characterized by a stippled or pale color, shallow pockets between the tooth and gingival margin, and absence of bleeding on gentle probing. Diseases can be caused by various factors, but dental plaque is a significant contributor. When left untreated, it leads to an imbalance between the host and bacteria, potentially resulting in disease. Other local and systemic factors, such as age, socio-economic status, oral hygiene habits, and diet, also play a role in periodontal disease development. Systemic factors like uncontrolled diabetes or tobacco smoking can exacerbate the condition. Signs of periodontal disease include bleeding gums, gingival recession, bad breath, loose teeth, ill-fitting dentures, and plaque buildup. Individual risk factors are diverse, including gender, smoking, stress, and genetic predisposition. In 2017, the American Academy of Periodontology and European Federation of Periodontology collaborated on a revised classification system to provide personalized patient care. The 2018 disease classification for periodontal health and gingival diseases is as follows: * Healthy gingiva * Gingivitis (dental biofilm-induced) * Drug-influenced gingival enlargement * Gingival diseases (nondental biofilm-induced) + Genetic or developmental disorders + Specific infections + Inflammatory and immune conditions + Reactive processes + Neoplasms + Endocrine, nutritional, and metabolic diseases + Traumatic lesions + Gingival pigmentation * Periodontal recession * Bone loss in periapical radiographs The classification system categorizes periodontitis into three forms: necrotizing periodontal disease, necrotizing gingivitis, and necrotizing periodontitis. These conditions are further divided into subcategories based on severity and complexity of management. The classification is based on the primary systemic disease according to ICD codes. The 2018 Disease Classification of Periodontal Disease and Conditions categorizes various conditions that affect the periodontium, including systemic diseases, other periodontal conditions, and factors such as trauma, orthodontic forces, and dental prostheses. The classification system divides these conditions into four subcategories: peri-implant health, peri-implant mucositis, peri-implantitis, and peri-implant soft and hard tissue deficiencies. Prevention is key, with the most effective methods being those achievable at home, such as proper tooth brushing technique, interdental cleaning aids, and regular dental check-ups. Specialist periodontist treatment can also be used, including routine scale and cleans, plaque removal techniques, and a plaque index to identify areas of plaque. The primary cause of periodontal disease is the biofilm of dental bacteria, which adheres to non-shedding surfaces such as teeth, restorative materials, and dental appliances. The stages of biofilm formation include the selective absorption of salivary and gingival crevicular fluid molecules through electrostatic affinity with hydroxyapatite, bacterial transportation, reversible interaction, and irreversible interaction. Planktonic cells stick together due to co-adhesion, which involves single-culture cells adhering to already attached organisms on a surface. Early colonizers make initial contact with the surface, allowing later co-adhesion of bacteria and facilitating complex multispecies dental biofilms. The growth and maturation of existing plaque microorganisms and recruitment of new colonizers lead to multiplication. As the bacterial community stabilizes, it reaches a climax community where it has sufficient nutrients and protection to survive. This complex biofilm is often found in hard-to-clean areas and relies on dietary consumption for supra-gingival organisms and blood/CFF for sub-gingival ones. Detachment from one surface to another or within the biofilm allows colonization at remote sites, protected by a slimy extracellular matrix that shields bacteria from environmental factors and chemotherapeutic agents. Antibiotics are not typically used in treating periodontal disease due to this protective mechanism. Mechanical removal via toothbrushing, interdental cleaning, or periodontal debridement performed by a dental professional is the most effective way to control plaque biofilm. The host's inflammatory response plays a crucial role in periodontal disease pathogenesis. Even with healthy-appearing gingiva, there's a constant low-level inflammatory response facilitated by the host to manage bacterial loads. Leukocytes and neutrophils phagocytose bacteria found in the gingival crevice or pocket, while damage to epithelial cells releases cytokines attracting leukocytes to assist with inflammation. When plaque bacteria exceed neutrophil capabilities, they degranulate, releasing toxic enzymes that cause tissue damage, leading to red, swollen, and inflamed gingiva. This can bleed when probed clinically or during tooth brushing due to increased capillary permeability and inflammatory cell influx. Established disease leads to further recruitment of macrophages and lymphocytes, initiating an immune response and producing pro-inflammatory cytokines that escalate inflammation. The progression of chronic systemic inflammation and disease results in collagen breakdown, infiltrate accumulation, and alveolar bone resorption. This stage involves the destruction of periodontal ligament and alveolar bone, leading to tooth loss if left untreated. Periodontal diseases have evolved from gingivitis to periodontitis, with irreparable loss of supporting structures. A risk factor is a characteristic linked to an increased rate of subsequent disease occurrence. Modifiable and non-modifiable factors contribute to disease development. Research studies are essential for determining risk factors, which often coexist with other variables. Risk factors can be genetic, environmental, behavioural, psychological, or demographic in nature. The primary aetiological factor for periodontal disease is bacterial plaque or biofilm. Understanding individual risk factors is crucial for diagnosis, treatment, and management of the diseases. Each person has a unique array of risk factors contributing to susceptibility and severity. Modifiable risk factors include tobacco smoking, which strongly correlates with periodontal tissue destruction. Smoking decreases oral tissue healing abilities by destroying blood vessels and preventing essential immune-defence organisms from penetrating tissues. This allows pathogenic bacteria to rapidly destroy periodontal tissues. Smokers have larger areas of deep pocketing and loss of clinical attachment compared to nonsmokers. Smoking cessation has been proven to prevent disease progression and return the oral microflora to a less pathogenic state. Moderate alcohol consumption increases risk for periodontal disease progression, while diabetes is a modifiable risk factor that can be controlled to aid periodontal disease control. A two-way relationship exists between blood glucose control and periodontal disease control. Periodontal disease is influenced by various factors, including disease severity and progression, diabetes, obesity, vitamin D deficiency, poor oral hygiene, stress, pregnancy, genetics, immune response, and osteoporosis. Individuals with diabetes mellitus tend to have poorer healing abilities, making them more susceptible to severe diseases if blood glucose control is inadequate. Obesity and vitamin D deficiency are both risk factors for periodontal disease. A diet high in fat, salt, and sugar contributes to obesity and leads to a lack of essential nutrients, including vitamins C and D. Poor oral hygiene is the most significant risk factor in initiating and progressing periodontal disease. Brushing and interdental cleaning can effectively remove dental plaque biofilm and prevent the disease. Cardiovascular disease has been linked to poor oral hygiene, as high cholesterol levels and oral bacteria contribute to atherosclerosis. Chronic stress increases the production of immune cells and interleukins, weakening the body's defense against pathogenic bacteria. During pregnancy, the oral tissues are affected due to decreased immune response and increased vascular blood supply. While pregnancy does not cause gingival or periodontal diseases, it may exacerbate existing inflammatory responses. Good oral hygiene through toothbrushing and interdental cleaning can prevent these effects. Non-modifiable risk factors include genetics and the host response. Periodontal disease development is also influenced by abnormal or decreased immune response rather than aggressive bacterial pathogens. Osteoporosis has been linked to less dense alveolar bone, but further research is needed to determine if it's a true risk factor for periodontal disease. Drug-induced disorders have also been identified as potential risk factors for periodontal disease. Poly-pharmacy patients require regular medical history reviews to assess their risk of periodontal disease, which can be exacerbated by conditions such as dry mouth and gingival enlargement. Haematological disorders can have a significant impact on the health of periodontal tissues, making it crucial for dental professionals to consider these factors when evaluating patient risk. Age, gender, socioeconomic status, education, and genetics also play a role in influencing periodontal disease. To effectively manage this multifactorial condition, dental professionals must understand the various risk factors and their mechanisms. In order to identify periodontitis and associated conditions, classification systems have been developed to categorize diseases based on severity, aetiology, and treatment. A thorough examination of patient medical, dental, and social histories is necessary to reach a diagnosis, which involves combining findings from intra and extra oral examinations with indices such as the PSR and CPITN. Once disease has been identified, a full periodontal analysis can be performed to assess the level of disease, including measurements of pocket depths, clinical attachment loss, and recession. This information is used to determine the best course of treatment, which may involve radiographs to assess alveolar bone levels and levels of destruction. Contemporary periodontal treatment follows a trimeric model, with four phases designed to ensure effective therapy and improve patient prognosis. The non-surgical phase is typically the first step in this process. Maintaining Oral Health Through Periodontal Treatment Periodontal therapy encompasses a series of treatments aimed at reducing and eliminating gingival inflammation, commonly known as gingivitis. The goal is to remove factors contributing to this condition, such as dental plaque and calculus, tooth decay, and defective restorations. Phase 1 focuses on treating emergencies, administering antimicrobial therapy, controlling diet, educating patients, addressing iatrogenic factors, and performing procedures like deep caries treatment, hopeless teeth extraction, and preliminary scaling. Patients are typically re-evaluated 3-6 weeks after the initial phase to assess the effectiveness of the treatment plan. This re-evaluation is crucial in severe cases of periodontal disease, where it's essential to monitor oral hygiene, bleeding, plaque scores, and diagnosis and prognosis. If necessary, patients may require surgical intervention, which involves managing periodontal pockets, irregular bony contours, or deep craters. The goal is to restore any defects with removable or fixed dental prosthesis or other restoration processes. The maintenance phase prioritizes preserving periodontal health through regular scheduled visits for maintenance care. This long-term approach contributes to the success of periodontal treatment and fosters a strong relationship between the oral health therapist, dentist, or periodontist and the patient. Establishing firm gingival margins prior to restorative treatment is crucial, as well as ensuring the absence of bleeding tissue during manipulation. Periodontal therapy aims to increase sufficient tooth length for retention and prevent complications like impression making and restoration failure. Non-surgical therapy serves as the golden standard, focusing on debridement, oral hygiene instructions, and patient motivation to eliminate and reduce putative pathogens, stabilizing periodontal disease and shifting microbial flora to a favorable environment. Debridement is the mainstay of therapy for inflammatory periodontal diseases and remains the gold standard for both surgical and non-surgical treatment during the initial stages. It is performed using hand instruments such as curettes or scalers, and ultrasonic instrumentation.[45] The procedure typically requires multiple appointments to effectively remove supragingival and subgingival calculus, especially when periodontal pockets are present. Debridement helps in periodontal healing and reduces periodontal pocketing by modifying the subgingival ecological environment.[45] Preventing periodontal disease and maintaining healthy periodontal tissues post-initial treatment relies heavily on the patient's ability to effectively remove dental plaque. This requires the patient to be motivated to improve their oral hygiene and undergo behaviour change in areas such as tooth brushing, interdental cleaning, and other oral care techniques.[46] Personal oral hygiene is considered crucial for controlling chronic periodontitis. Research highlights the importance of appreciating patient motivation behind behaviour changes stemming from improved oral hygiene practices.[46] Patients must be willing to enhance their oral hygiene and have confidence in their ability to do so. Clinicians play a pivotal role in encouraging such changes and educating patients accordingly. Motivational interviewing is an effective technique for asking open-ended questions and expressing empathy towards the patient. Oral health therapists, who are dual-qualified dental hygienists and dental therapists, collaborate with dentists and periodontists to treat gingival and periodontal diseases.[47] They conduct oral health assessments, diagnoses, treatments, and maintenance, as well as referrals when necessary. These professionals also provide oral health education and promotion to support patients in maintaining their at-home oral care.[47] Oral health therapists share the responsibilities of care with the dental team, providing essential services that enable dentists to focus on more complex cases or medically compromised patients.[48] A dental degree in Canada typically requires a minimum of three years and leads to an MSc or MDent degree, making graduates eligible for fellowship exams with the Royal College of Dentists of Canada. To become licensed, one must have a BDS, DDS, or DMD degree and be certified by the National Dental Examining Board of Canada. In the US, American Dental Association-accredited programs also follow this structure. The American Academy of Periodontology states that US-trained periodontists specialize in prevention, diagnosis, and treatment of periodontal diseases and oral inflammation, as well as dental implant placement and maintenance. Many periodontists also diagnose and treat oral pathology. The completion of post-graduate training makes a periodontist board-eligible for the American Board of Periodontology examination. Regular maintenance periodontal therapy is crucial after treatment to achieve long-term results and stabilize periodontal disease. Different types of periodontal diseases require varying levels of patient compliance, with gingivitis being easily preventable by thorough cleaning and plaque removal. Acute necrotizing ulcerative gingivitis and necrotizing ulcerative periodontitis are distinct conditions characterized by gum necrosis, pain, bleeding, halitosis, and discoloration. Treatment involves debridement, often under local anesthesia, followed by chlorhexidine mouthwash use, oral health instruction, interdental cleaning aids, nutritional guidance, and smoking cessation. Pain management options include ibuprofen or paracetamol/acetaminophen, while antibiotics may be prescribed for immunocompromised patients. Regular assessment of treatment is necessary to ensure resolution of symptoms and restoration of gingival health. Periodontitis is an ongoing infection of the gums that causes irreversible damage to the surrounding bone and teeth structure. This condition progresses slowly but can have sudden bursts, often caused by local factors such as poor oral hygiene, diet, plaque accumulation, smoking, or other lifestyle choices. Characterized by pocket formation and gum recession, periodontitis requires immediate treatment and maintenance to prevent further progression and alleviate inflammation. Treatment typically involves scaling and root planning, surgical therapy, and regenerative surgical therapy. After treatment, regular check-ups with a dentist are crucial to monitor the disease's recurrence and ensure complete eradication. To control plaque and maintain oral health, patients should practice good hygiene habits such as brushing their teeth twice daily and performing interdental cleaning once daily. Additionally, chlorhexidine mouthwash can be an effective tool in preventing disease recurrence. Regular dental check-ups every three months are essential for monitoring progress and adjusting treatment plans as needed.**The Effects of Various Factors on Periodontal Disease** Research has shown that smoking and smoking cessation can both impact periodontal healing after mechanical therapy (Harrap, 1997). Additionally, studies have found links between alcohol consumption and periodontal disease progression (Tezal et al., 2001). Nutritional factors also play a role in the development and treatment of periodontal disease. A study found that specific nutrients can influence the onset, progression, and treatment of the condition (Neiva et al., 2003). Other research has explored the relationship between serum lipids and periodontitis in elderly non-smokers (Izumi et al., 2009). Stress, anxiety, and other psychological factors have also been linked to an increased risk of periodontal disease (Hugoson et al., 2002). Furthermore, pregnancy has been shown to affect both periodontal and dental health (Laine, 2009). Genetic factors can also contribute to the development of adult periodontitis (Michalowicz et al., 2000). Moreover, research has highlighted the importance of resolving inflammation in the pathogenesis of periodontal diseases (Van Dyke & Serhan, 2016). Other studies have investigated the management of drug-induced gingival enlargement and the periodontal manifestations of systemic disease (Taylor, 2003; Kinane & Marshall, 2001). Finally, research has emphasized the importance of proper diagnosis and classification of periodontal diseases (Armitage, 2004) and the detection and prevention of periodontal conditions (Preshaw, 2015). A pilot study examined serum levels of cytokines in subjects with generalized chronic and aggressive periodontitis before and after non-surgical periodontal therapy (Duarte et al., 2010). Given article text here 2019年5月8日，美国牙科学会（American Academy of Periodontology）发布了关于口腔疾病诊断和治疗的指南。 Dental care for Periodontal Disease requires professional attention from a dentist. Your dentist will assess the affected tooth and surrounding gums before taking dental X-rays to evaluate bone levels and damage extent. Inform your dentist about medications, vitamins or supplements you are taking. Sedation options in dentistry are available for those experiencing anxiety or discomfort during procedures. Options include - Nitrous oxide (laughing gas) for light sedation - Oral conscious sedation with diazepam, midazolam, triazolam and lorazepam - Intravenous sedation for severe cases using midazolam and meperidine Apply gentle, consistent pressure using a firm touch. This will help decrease blood flow and allow for clot formation, which is a natural part of the recovery process - It accelerates healing and minimizes the likelihood of dry sockets. Remove the gauze once the bleeding has diminished sufficiently. It's normal to experience some light bleeding during the initial 24 hours.