Republic of Iraq Ministry of Higher Education and Scientific Research University of Baghdad College of Dentistry



Clinical Significance of Dental Caries in Pulp Pathology

A Project Submitted to The College of Dentistry, University of Baghdad, Department of Restorative and Esthetic Dentistry In Partial Fulfillment for the Bachelor of Dental Surgery

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2023 A.D

بسم الله الرحمن الرحيم

وَنَوْكُلْ عَلَى ٱللهِ وَكَفَى بِاللهِ وَحِيلًا

صدق الله العظيم

Certification of the Supervisor

I certify that this project entitled " Clinical Significance of Dental Caries in Pulp Pathology " was prepared by the fifth-year student Mena Mohammed Kassim under my supervision at the College of Dentistry/University of Baghdad in partial fulfillment of the graduation requirements for the Bachelor Degree in Dentistry.

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Dedication

This project is dedicated to my parents who providing me unfailing support and continuous encouragement throughout my years, who have never failed to give me love and support, for teaching me that even the largest task can be accomplished. To my brothers, my best friend and to all of those who have always been there for me during this journey all these years and make it better.

Acknowledgment

First and lastly, all gratefulness, faithfulness and thankfulness to ALLAH for providing me with patience, perseverance and the ability to undertake and finally complete this study.

I would like to express my gratitude to Dr. Raghad Al-hashimi, Dean of College of Dentistry, University of Baghdad for his continuous care.

I would like to thank Prof. Anas Mahdee Chairman of Restorative and Esthetic Department for her continuous care.

I would like to express my deepest gratitude and thanks to my supervisor Lecturer Alaa Jawad Kadhim for her excellent scientific guidance and support.

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Introduction

Dental caries (caries-from the Latin, decay) simply means decay or rotting of the teeth (L. M. Silverstone N. W. Johnson et al., 1981). Dental caries is a major public health problem. U.S. Department of Health and Human Services. (Oral Health in America Report of the Surgeon General, 2016). It is one of primary causes of tooth loss and can lead to negative impacts on quality of life. (Ministério da Saúde 2004).

Interaction of lifestyle, behaviour or systemic diseases with dental caries and periodontal diseases. A tooth is made up of 3 layers. The outermost layer of the tooth crown is the enamel, which is the hardest or most mineralized part of the tooth—it is harder than bone **(Ross, Michael H.et al 2002)**.

The next layer is the dentin, which has a higher percentage of organic material (collagen) and water than the enamel has and is softer. Because it is softer than enamel, it decays more rapidly and is subject to severe cavities if not properly treated, but dentin still acts as a protective layer and supports the crown of the tooth (Cate, A. R. Ten 1998).

In the center of the tooth is the pulp (Cate, A. R. Ten 1998) consisting of nerves and blood vessels(Ross, Michael H.et al 2002), which keeps the tooth alive and provides sensation to the tooth. The outermost layer of the tooth root is cementum (Ross, Michael H.et al 2002) (instead of enamel), followed by dentin, which encases the pulp tissues contained within the root canals. So the caries is a form of progressive destruction of enamel, dentine and cementum initiated by microbial activity at the tooth surface (L. M. Silverstone N. W. Johnson et al. 1981), without treatment, the inflammation will get worse it will go through stages till it became inflammation of the pulp.

Aim of study

This aim of this study to know the cause of dental caries and their clinical significance and their prognosis and how it will effect on pulp of the tooth.

Chapter one: literature review

1.1 Etiology of dental caries

Dental caries is a term that refers to both the disease and the resulting lesion. The caries process occurs in the biofilm, which is permanently active with every pH fluctuation (Kidd EA. 2010), and the lesion manifests in the dental hard tissues.

Dental caries occurs when the biofilm microbiota that normally resides in the oral cavity in homeostasis change to an acidogenic, aciduric, and cariogenic population due to the frequent consumption of sugars. (Schwendicke F, Frencken JE, et l 2016) The result of this shift can be clinically invisible or lead to a net mineral loss within the tooth's hard structures, resulting in a visible carious lesion. (Kidd EA. 2010) - caries, the process, can exist without caries, the visible lesion.

Therefore, dental caries is considered a dietary-microbial disease (Pitts NB, Zero DT, et al 2017) that requires a cariogenic biofilm and regular exposure to fermentable carbohydrates (glucose, fructose, maltose, and sucrose) from the diet.

Behavioral, psychological, and social factors also play a significant role in the disease process.(Reisine S, Litt M. Social 1993 Jun).

Fluoride's capacity to prevent caries is a well-known fact, and insufficient fluoride exposure should also be considered contributing factor in the disease process.

(Zero DT. 2004).

1.2 Classification of dental caries (Vimal K Siri 2017)

A) Based on Location

1-Pits and fissure caries (Figure 1).



Figure .1 Pits and fissure caries

2-Smooth surface caries (Figure 2).



Figure .2 Smooth surface caries

3-Root surface caries (senile caries) (Figure 3).



Figure .3 Root surface caries

B) Based on Rapidity of Caries

1-Acute dental caries (rampant caries)(Figure 4).



Figure.4 rampant caries

2-Chronic dental caries (Figure 5).



Figure .5 Chronic dental caries

3-Arrested dental caries (Figure 6).



Figure .6 Arrested dental caries

Based on the Number of Surfaces Involved

1-Simple caries: Caries involving only one surface of the tooth. (Fig 7)



Figure . 7 Simple caries

2-Compound caries: Caries involving two surfaces of the tooth. (Fig 8)



Figure . 8 Compound caries

3-Complex caries: Caries involving three or more surfaces of the tooth.(Fig 9)



Figure. 9 Complex caries

C) Based on Extent of Caries

1-Incipient caries (reversible) (Figure 10)



Figure .10 Incipient caries

2-Cavitated caries (non-reversible) (Fig 11).



Figure .11 Cavitated caries

D) Based on the Age of the Patient

1-Nursing bottle caries: During early infancy, bottle-fed babies develop rapidly spreading caries usually on maxillary incisors (Figure 12).



Figure. 12 Nursing bottle caries

2-Adolescent caries: Caries seen in the teenage population due to dietary habits.3-Root caries: Caries of cementum; seen in older age patients (Figure 13)



Figure. 13 Root caries

- E) Based on whether it is a New or Recurrent Carious Lesion
- 1-Primary caries (Figure 14)



Figure. 14 Primary caries

2-Secondary caries/recurrent caries (Figure 15)



Figure. 15 Recurrent caries

F) Based on the Treatment and Restorative Design (GV Black)

1-Class I: Caries in structural defects of teeth like pits and fissures and some defective grooves and occlusal surface of molars and premolars, occlusal 2/3rd of buccal and lingual surfaces of molars and lingual surface of anterior teeth. (Figure16)



Figure. 16 class I Caries

2-Class II: Caries on the proximal surfaces of molars and premolars (Figure 17)



Figure. 17 class II Caries

3-Class III: Caries on the proximal surface of anterior teeth without involving incisal edge (Figure 18).



Figure. 18 Class III Caries

4-Class IV: Caries on the proximal surface of anterior teeth with involvement of incisal edge (Figure 19).



Figure. 19 Class IV Caries

4-Class V: Caries seen at the gingival third of facial and lingual surfaces of anterior and posterior teeth (Figure 20).



Figure. 20 Class V Caries

1.3 Signs and symptoms

The signs and symptoms of cavities vary, depending on their extent and location. When a cavity is just beginning, you may not have any symptoms at all. And you may not be aware of the disease (health promotion board dental caries) as the decay gets larger, it may cause signs and symptoms such as(Mayo Clinic 2017):

- Toothache, spontaneous pain or pain that occurs without any apparent cause
- Tooth sensitivity
- Mild to sharp pain when eating or drinking something sweet, hot or cold
- Visible holes or pits in your teeth
- Brown, black or white staining on any surface of a tooth
- Pain when you bite down

1.4 Treatment (Mayo Clinic.2017)

Regular checkups can identify cavities and other dental conditions before they cause troubling symptoms and lead to more-serious problems. The sooner you seek care, the better your chances of reversing the earliest stages of tooth decay and preventing its progression. If a cavity is treated before it starts causing pain, you probably won't need extensive treatment. Treatment of cavities depends on how severe they are and your particular situation. Treatment options include:

• Fluoride treatments. If your cavity just started, a fluoride treatment may help restore your tooth's enamel and can sometimes reverse a cavity in the very early stages. Professional fluoride treatments contain more fluoride than the amount found in tap water, toothpaste and mouth rinses. Fluoride treatments may be liquid, gel, foam or varnish that's brushed onto your teeth or placed in a small tray that fits over your teeth.

• Fillings. Fillings, also called restorations, are the main treatment option when decay has progressed beyond the earliest stage. Fillings are made of various materials, such as tooth-colored composite resins, porcelain or dental amalgam that is a combination of several materials.

• Crowns. For extensive decay or weakened teeth, you may need a crown a customfitted covering that replaces your tooth's entire natural crown. Your dentist drills away all the decayed area and enough of the rest of your tooth to ensure a good fit. Crowns may be made of gold, high strength porcelain, resin, porcelain fused to metal or other materials.

• Root canals. When decay reaches the inner material of your tooth (pulp), you may need a root canal. This is a treatment to repair and save a badly damaged or infected tooth instead of removing it. The diseased tooth pulp is removed. Medication is sometimes put into the root canal to clear any infection. Then the pulp is replaced with a filling.

• Tooth extractions. Some teeth become so severely decayed that they can't be restored and must be removed. Having a tooth pulled can leave a gap that allows your other teeth to shift. If possible, consider getting a bridge or a dental implant to replace the missing tooth.

1.5 Stages of dental caries (Tru Blu Dentistry 2018)

Five stages of Dental caries are (figure 21):

- 1st Stage: Chalky White Spots
- 2nd Stage: Decay of the Dental Enamel
- 3rd Stage: Decay of the dentin
- 4th Stage: Decay reaches the pulp
- 5th Stage: Abscess

STAGES OF TOOTH CARIES



Tooth stain

Enamel caries

Pulpitis

Periodontitis

Figure 21 Stages of dental caries

2. Pulpitis

Pulpitis is inflammation of dental pulp tissue. The pulp contains the blood vessels, the nerves, and connective tissue inside a tooth and provides the tooth's blood and nutrients. Dental pulp is sometimes called the root of the tooth. Much like the roots of a tree, it is the pathway for nutrients to go up into the tooth (figure 22). The pulp surrounded by a protective layer called the dentin. Pulpitis may be caused by bacteria from dental caries that penetrate through the enamel and dentin to reach the pulp, or it may be mechanical, a result of trauma, such as physical damage to the tooth (V. Gopikrisha 2021).



Figure 22: Pulpitis stages

Inflammation is commonly associated with a bacterial infection but can also be due to other insults such as repetitive trauma or in rare cases periodontitis The inflammation of dental pulp is mainly caused by an opportunistic infection of the pulp by a commensal oral microorganism. To reach the pulp, the most common route of the microorganism is through dental caries as well as from trauma, dentinal cracks and exposed dentin. Exposed dentin gives the microorganisms access to the pulp of the tooth through the dentinal tubules. (**Rechenberg, Dan-Krister et al 2016**).

In the case of penetrating decay, the pulp chamber is no longer sealed off from the environment of the oral cavity. (Kakehashi S, Stanley HR et al 1960).

When the pulp becomes inflamed, pressure begins to build up in the pulp cavity, exerting pressure on the nerve of the tooth and the surrounding tissues. Pressure from inflammation can cause mild to extreme pain, depending upon the severity of the inflammation and the body's response. Unlike other parts of the body where pressure

can dissipate through the surrounding soft tissues, the pulp cavity is very different. It is surrounded by dentin, a hard tissue that does not allow for pressure dissipation, so increased blood flow, a hallmark of inflammation, will cause pain (**Hargreaves**, **KM. Goodis, et al 2002**).

The infection can also come from the apical foramen of the root. (Anirudha Agnihotry, Wendy Thompson, et al 2019).

Cells in the dental pulp trigger an immune response from the invasion of foreign microorganisms. The inflammation of the pulp is a side effect of the immune response and causes pain. (Zanini, Marjorie et al 2017).

Pulpitis can often create so much pressure on the tooth nerve that the individual will have trouble locating the source of the pain, confusing it with neighboring teeth, called referred pain. The pulp cavity inherently provides the body with an immune system response challenge, which makes it very difficult for a bacterial infection to be eliminated (**Torabinejad, M. Walton et al 2008**).

If the teeth are denervated, this can lead to irreversible pulpitis, depending on the area, rate of infection, and length of injury. This is why people who have lost their dental innervation have a reduced healing ability and increased rate of tooth injury. Thus, as people age, their gradual loss of innervation leads to pulpitis. (Byers, M. R., Suzuki, et al (2003)



Figure 23 healthy tooth comparted to infected tooth

2.1 Diagnosed (Cleveland Clinic medical 2022)

One of the main ways to diagnose pulpitis is by assessing the sensitivity of the tooth. Types of sensitivity tests include:

- Electric pulp testing: This test determines if your pulp is still alive or if the infection has progressed to pulp necrosis. Your dentist uses an instrument to deliver a small electrical pulse to the tooth. If your pulp is alive, you'll feel the stimulation. If the pulp tissue is dead, you won't feel the electrical pulse.
- Heat or cold test: Your dentist will touch your teeth with a hot or cold substance. In irreversible pulpitis, sensitivity lasts for longer than a few seconds. If the pulp tissue is dead, you won't feel any sensitivity.
- **Tooth tapping:** This involves a gentle tapping on your tooth. Pain with tapping is a sign of irreversible pulpitis.

And also take dental X-rays to look for defects in the tooth and signs of infection.

2.2 Inflammatory diseases of the dental pulp (V. Gopikrisha 2021)

Inflammatory diseases of the dental pulp are:

(a) Reversible pulpitis

- Acute reversible pulpitis

-Chronic reversible pulpitis

(b) Irreversible pulpitis

-Symptomatic irreversible pulpitis (previously known as acute irreversible pulpitis)

-Asymptomatic irreversible pulpitis (previously known as chronic irreversible pulpitis)

-Chronic hyperplastic pulpitis (also known as pulpal hyperplasia)

-Internal resorption

2.3 Reversible Pulpitis (V. Gopikrisha 2021)

Reversible pulpitis is a mild-to-moderate inflammatory condition of the pulp caused by noxious stimuli in which the pulp is capable of returning to the uninflamed state following removal of the stimuli.

2.3.1 Types

• Acute reversible pulpitis: The pain has been present for a short time (e.g., a few hours or days); however, it immediately ceases on removal of the aggravating stimuli.

• Chronic reversible pulpitis: The pain has been present for a long time (e.g., months)

2.3.2 Cause

Reversible pulpitis may be caused by any agent that is capable of injuring the pulp. Specifically, the cause may be any of the following:

• Trauma, as from a blow or from a disturbed occlusal relationship

• Thermal shock, as from preparing a cavity with a dull bur or keeping the bur in contact with the tooth for too long, or as from overheating during polishing a fillingwever, it immediately ceases on removal of the aggravating stimuli.

• Excessive dehydration of a cavity or irritation of exposed dentin at the neck of a tooth

• Placement of a fresh amalgam filling in contact with, or occluding, a cast restoration

• Chemical stimulus, as from sweet or sour foodstuffs or from irritation of a filling; or bacteria, as from caries

2.3.3 Symptoms

• Symptomatic reversible pulpitis is characterized by short, sharp pain lasting for a moment.

• This pain is always specific to a stimulus.

• The pain is instantly relieved on removal of the stimulus.

• It is more often brought on by cold than by hot food or beverages and by cold air. It does not occur spontaneously and does not continue when the cause has been removed. The clinical difference between reversible and irreversible pulpitis is quantitative; the pain of irreversible pulpitis is more severe and lasts longer. In reversible pulpitis, the cause of the pain is generally traceable to a stimulus, such as cold water or a draft of air, whereas in irreversible pulpitis, the pain may come without any apparent stimulus

2.3.4 Diagnosis

Diagnosis is by a study of the patient's symptoms and by clinical tests. The pain is sharp, lasts but a few seconds, and generally disappears when the stimulus is removed. Cold, sweet, or sour usually causes it. Pain may become chronic. Although each paroxysm may be of short duration, the paroxysms may continue for weeks or even months. The pulp may recover completely, or the pain may last longer each time, and intervals of relief may become shorter, until the pulp finally succumbs. Because the pulp is sensitive to temperature changes, particularly cold, application of cold is an excellent method of locating and diagnosing the involved tooth. A tooth with reversible pulpitis reacts normally to percussion, palpation, and mobility, and the periapical tissue is normal on radiographic examination.

2.3.5 Differential Diagnosis (V. Gopikrisha 2021)

In reversible pulpitis, the pain is generally transitory, lasting a matter of seconds, whereas in irreversible pulpitis, the pain may last several minutes or longer. The patient's description of the pain, particularly regarding its onset, character, and duration, is often of inestimable help in arriving at a correct differential diagnosis. Thermal tests are useful in locating the affected tooth if unknown. The electric pulp test, using less current than on a control tooth, is an excellent corroborating test.

2.3.6 Treatment (V. Gopikrisha 2021)

The best treatment for reversible pulpitis is prevention. Periodic care to prevent the development of caries, early insertion of a filling if a cavity has developed, desensitization of the necks of teeth where gingival recession is marked, use of a cavity varnish or cement base before insertion of a filling, and care in tooth preparation and polishing are recommended to prevent pulpitis. When reversible pulpitis is present, removal of the noxious stimuli will usually bring the pulp back to a healthy state. Once the symptoms have subsided, the tooth should be tested for vitality to make sure that pulpal necrosis has not occurred. When pain persists despite proper treatment, the pulpal inflammation should be regarded as irreversible, the treatment for which is pulp extirpation.

2.3.7 Prognosis (V. Gopikrisha 2021)

The prognosis for the pulp is favorable if the irritant is removed early enough; otherwise, the condition may develop into irreversible pulpitis.

2.4 Irreversible pulpitis

2.4.1 Cause

The most common cause of irreversible pulpitis is bacterial involvement of the pulp through caries, although any clinical factor, chemical, thermal, or mechanical, already mentioned as a cause of pulp disease, may also cause pulpitis. As previously stated, reversible pulpitis may deteriorate into irreversible

2.4.2 Symptoms

• In the early stages of irreversible pulpitis, a paroxysm of pain may be caused by the following: sudden temperature changes, particularly cold, sweet or acid foodstuffs, and pressure from packing food into a cavity or suction exerted by the tongue or cheek.

• Symptomatic irreversible pulpitis exhibits pain usually caused by a hot or cold stimulus, or pain that occurs spontaneously. The pain persists for several minutes to hours, lingering after removal of the thermal stimulus.

• The pain often continues when the cause has been removed, and it may come and go spontaneously, without an apparent cause.

• The patient may describe the pain as sharp, piercing, or shooting, and it is generally severe.

• It may be intermittent or continuous, depending on the degree of pulpal involvement and on whether it is related to an external stimulus.

• The patient may also complain of postural pain, i.e., change of position (bending over or lying down) exacerbates the pain. This is due to the increase in intrapulpal pressure when the patient changes position from a standing posture to a supine (lying down) posture.

• The patient may also have pain referred to adjacent teeth, to the temple or sinuses when an upper posterior tooth is involved, or to the ear when a lower posterior tooth is affected.

• In later stages, the pain is more severe and is generally described as boring, gnawing, or throbbing, or as if the tooth was under constant pressure. The pulp need not be macroscopically exposed, but a slight exposure is generally present, or else

the pulp is covered with a layer of soft, leathery decay. When no outlet is present, whether because of a covering of decay or a filling or because of food packed into a small exposure in the dentin, pain can be most intense.

• Patients are often kept awake at night by the pain (nocturnal pain), which continues to be intolerable despite all their efforts at analgesia. Pain is increased by heat and is sometimes relieved by cold, although continued cold may intensify the pain. After exposure and drainage of the pulp, pain may be slight, manifesting itself as a dull consciousness, or may be entirely absent. Pain can return if food packs into the cavity or underneath a leaky filling, it may not be as intense because of degeneration of the superficial nerve fibers.

• Apical periodontitis is absent, except in the later stages, when inflammation or infection extends to the periodontal ligament.

2.4.3 Diagnosis (V. Gopikrisha 2021)

Inspection generally discloses a deep cavity extending to the pulp or decay under a filling. The pulp may already be exposed. On gaining access to the exposure, one may see a grayish, scum-like layer over the exposed pulp and the surrounding dentin. This layer is composed of food debris, degenerated polymorphonuclear leukocytes, microorganisms, and blood cells. The surface of the pulp is eroded. An odor of decomposition is frequently present in this area. Probing into the area is not painful to the patient until the deeper areas of the pulp are reached. At this level, both pain and hemorrhage may occur. If the pulp is not exposed by the carious process, a drop of pus may be expressed when one gains access to the pulp chamber. Radiographic examination may not show anything of significance that is not already known clinically. It may disclose an interproximal cavity not seen visually or may suggest involvement of a pulp horn. A radiograph may also show exposure of the pulp, caries

under a filling, or a deep cavity or filling threatening the integrity of the pulp. In the early stages of irreversible pulpitis, the thermal test may elicit pain that persists after removal of the thermal stimulus. In the late stages, when the pulp is exposed, it may respond normally to a thermal stimulus, but generally it reacts feebly to heat and cold. The electric pulp test induces a response with a marked variation in current from the normal. Results of examination for mobility and percussion and palpation tests are negative.

2.4.4 Differential diagnosis (V. Gopikrisha 2021)

In the asymptomatic stage of irreversible pulpitis, the exposed pulp exhibits little or no pain, except when food is packed into the cavity. More current is required to elicit a response to the electric pulp test than in a control tooth. In the early symptomatic stage, less current than what is normal is needed to elicit a response to the electric pulp tester, and the pulp is often abnormally responsive to a cold stimulus. The induced or spontaneous pain that occurs is sharp, piercing, and readily identified with a specific tooth. Other symptoms may develop, such as diffuse, dull, constant pain, characterized by throbbing and gnawing, and the tooth may respond abnormally and severely to heat. This response is generally indicative of a later stage of irreversible pulpitis. In this stage of irreversible pulpitis, the symptoms may simulate those of an acute alveolar abscess. Such an abscess, however, causes at least some of the following symptoms, which help to differentiate it from irreversible pulpitis: swelling, tenderness on palpation, tenderness on percussion, mobility of the tooth, and lack of response to pulp-vitality tests. In addition, the patient may have symptoms of systemic toxicity such as fever and nausea. The pain of pulpitis is easy to localize by the patient at the onset. Once discomfort increases, the patient loses the ability to identify a particular tooth in the quadrant. A previous history of pain may help one to localize the origin of the pulpalgia. When pulpal pain is difficult to

localize, the application of heat with a consequent abnormal response is indicative of irreversible pulpitis in that tooth

2.4.5 Treatment (V. Gopikrisha 2021)

Treatment consists of complete removal of the pulp or pulpectomy. In posterior teeth, in which time is a factor, the removal of the coronal pulp or pulpotomy should be performed as an emergency procedure. Surgical removal should be considered if the tooth is not restorable.

2.4.6 Prognosis (V. Gopikrisha 2021)

The prognosis of the tooth is favorable if the pulp is removed and if the tooth undergoes proper endodontic therapy and an appropriate postendodontic restoration.

2.5 Chronic Hyperplastic Pulpitis

Chronic hyperplastic pulpitis, also known as pulpal hyperplasia or pulp polyp, is a productive pulpal inflammation due to an extensive carious exposure of a young pulp. This disorder is characterized by the development of granulation tissue, covered at times with epithelium and resulting from long-standing, low-grade irritation.

2.5.1 Cause

Slow, progressive carious exposure of the pulp is the cause. For the development of hyperplastic pulpitis, a large, open cavity; a young, resistant pulp; and a chronic,

low-grade stimulus are necessary. Mechanical irritation from chewing and bacterial infection often provide the stimulus.

2.5.2 Symptoms

Chronic hyperplastic pulpitis is symptomless, except during mastication, when pressure of the food bolus may cause discomfort.

2.5.3 Diagnosis

This disorder is generally seen only in the teeth of children and young adults. The appearance of the polypoid tissue is clinically characteristic; a fleshy, reddish pulpal mass fills most of the pulp chamber or cavity or even extends beyond the confines of the tooth

At times, the mass is large enough to interfere with comfortable closure of the teeth, although in the early stages of development, it may be the size of a pin. Polypoid tissue is less sensitive than normal pulp tissue and more sensitive than gingival tissue. Cutting of this tissue produces no pain, but pressure thereby transmitted to the apical end of the pulp does cause pain. This tissue bleeds easily because of a rich network of blood vessels. If the hyperplastic pulp tissue extends beyond the cavity of a tooth, it may appear as if the gum tissue is growing into the cavity. To differentiate a pulp polyp from proliferating gingival tissue, one should raise and trace the stalk of the tissue back to its origin, the pulp chamber. It should not be difficult to diagnose chronic hyperplastic pulpitis by clinical examination alone. The hyperplastic pulp tissue in the pulp chamber or cavity of a tooth is characteristic in appearance. Radiographs generally show a large, open cavity with direct access to the pulp chamber The tooth may respond feebly or not at all to the thermal test, unless one uses extreme cold, as from an ethyl chloride spray. More current than what is normal may be required to elicit a response by means of the electric pulp tester.

2.5.4 Differential diagnosis

Diagnosis The appearance of hyperplastic pulpitis is characteristic and should be easily recognized. The disorder must be distinguished from proliferating gingival tissue

The tissue in the pulp chamber is often transformed into granulation tissue, which projects from the pulp into the carious lesion. The granulation tissue is a young, vascular connective tissue containing polymorphonuclear neutrophils, lymphocytes, and plasma cells. The pulp tissue is chronically inflamed. Nerve fibers may be found in the epithelial layer.

2.5.5 Treatment

Treatment should be directed toward elimination of the polypoid tissue followed by extirpation of the pulp, provided the tooth can be restored. When the hyperplastic pulpal mass has been removed with a periodontal curette or spoon excavator, the bleeding can be controlled with pressure. The pulp tissue of the chamber is then completely removed, and a temporary dressing is sealed in contact with the radicular pulp tissue. The radicular pulp is extirpated at a later visit. If time permits, the entire procedure, pulpectomy, can be completed in a single visit.

2.5.6 Prognosis

The prognosis for the pulp is unfavorable. The prognosis for the tooth is favorable after endodontic treatment and adequate restoration



Figure .24

2.6 Internal Resorption Definition(V. Gopikrisha 2021)

Resorption is defined as a condition associated with either a physiologic or a pathologic process resulting in loss of dentin, cementum, or bone. Internal resorption is an idiopathic slow or fast progressive resorptive process occurring in the dentin of the pulp chamber or in the root canals of the teeth.

2.6.1 Cause

The cause of internal resorption is not known, but such patients often have a history of trauma.

2.6.2 Symptoms

Internal resorption in the root of a tooth is asymptomatic. In the crown of the tooth, internal resorption may be manifested as a reddish area called pink spot. This reddish area represents the granulation tissue showing through the resorbed area of the crown.

2.6.3 Diagnosis

s Internal resorption may affect either the crown or the root of the tooth, or it may be extensive enough to involve both. It may be a slow, progressive, intermittent process extending over 1 or 2 years; it may develop rapidly and may perforate the tooth within a matter of months. Although any tooth in the mouth can be involved, those most readily recognized are the maxillary anterior teeth. Usually, internal resorption is diagnosed during routine radiographic examination. The appearance of the "pink spot" occurs late in the resorptive process, when the integrity of the crown has been compromised. The radiograph usually shows a change in the appearance of the wall in the root canal or pulp chamber, with a round or ovoid radiolucent area.

2.6.4 Differential Diagnosis

When internal resorption progresses into the periodontal space, a perforation of the root occurs; it is difficult to differentiate from external resorption; in internal resorption, the resorptive defect is more extensive in the pulpal wall than on the root surface. This defect usually is recognized by means of a radiograph.

the pulp is removed. Multinucleated giant cells or dentinoclasts are present. The pulp usually is chronically inflamed. Metaplasia of the pulp, i.e., transformation to another type of tissue such as bone or cementum, sometimes occurs.

2.6.5 Treatment

Extirpation of the pulp stops the internal resorptive process. Routine endodontic treatment is indicated, but obturation of the defect requires a special effort, preferably with a plasticized gutta-percha method. In many patients, however, the condition progresses unobserved because it is painless, until the root is perforated. In such a case, mineral trioxide aggregate (MTA) is recommended to repair the

defect. When repair has been completed, the canal with its defect is obturated with plasticized gutta-percha

2.6.6 Prognosis

The prognosis is best before perforation of the root or crown occurs. In the event of a root–crown perforation, the degeneration does not usually cause definite clinical symptoms. The tooth is not discolored, and the pulp may react normally to electric and thermal tests. As degeneration of the pulp progresses, the tooth may become discolored, and the pulp will not respond to stimulation.



Figure 25 . "Pink spot" indicative of an internal resorptive defect seen in the palatal aspect of the crown of the maxillary central incisor



Figure 26 Radiographic image of the same tooth showing an internal resorptive defect with intact crown margins.

Chapter Two: Conclusion

Conclusion:

The following points are concluded:

- Without treatment the inflammation caused by dental caries will get worse
- The decay will go through stages till it become inflammation of the pulp

References:

1-Anirudha Agnihotry, Wendy Thompson, Zbys Fedorowicz, Esther J van Zuuren, Julie Sprakel (30 May 2019). <u>"Antibiotic use for irreversible pulpitis, a systematic review"</u>

2-Byers, M. R., Suzuki, H. and Maeda, T. (2003), Dental neuroplasticity, neuropulpal interactions, and nerve regeneration. Microsc. Res. Tech

3-Cate, A. R. Ten (1998). Oral Histology: development, structure, and function (5th ed.). Mosby.

4-Cleveland Clinic Medical 2022)

https://my.clevelandclinic.org/health/diseases/23536-pulpitis

5-Hargreaves, KM. Goodis, HE. Seltzer and Bender's Dental Pulp. Quintessence, 2002.

6-Kakehashi S, Stanley HR, Fitzgerald RJ. The effects of surgical exposures of dental pulps ingerm-free and conventional laboratory rats. Oral Surg Oral Med Oral Pathol 1965

7-Kidd EA. Clinical threshold for carious tissue removal. Dent Clin North Am. 2010 Jul

8- L. M. SILVERSTONE N. W. JOHNSON J. M. HARDIE R Dental Caries Aetiology, Pathology and Prevention

9-MS (Ministério da Saúde). Projeto SB Brasil 2003 – Condições de saúde bucal da população brasileira 2002–2003 – Resultados principais. Brasília: Coordenação Nacional de Saúde Bucal, 2004

10- Mayo Clinic. "Cavities/Tooth Decay - Diagnosis and Treatment - Mayo Clinic." *Mayoclinic.org*, 2017, <u>www.mayoclinic.org/diseases-</u> conditions/cavities/diagnosis-treatment/drc-

 Mayo Clinic. "Cavities/Tooth Decay - Symptoms and Causes." *Mayo Clinic*, 2017, www.mayoclinic.org/diseases-conditions/cavities/symptomscauses/syc-20352892.

12-Oral health in America Report of the SUREGEON GENERAL (2016) https://www.nidcr.nih.gov/research/data-statistics/surgeon-general 13-(Okutan Dental 2022)

https://www.okutandental.com/5-stages-of-tooth-decay-and-treatment-methods 14-Pitts NB, Zero DT, Marsh PD, Ekstrand K, Weintraub JA, Ramos-Gomez F, Tagami J, Twetman S, Tsakos G, Ismail A. **Dental caries**. Nat Rev Dis Primers. 2017 May

15-Rechenberg, Dan-Krister; Galicia, Johnah C.; Peters, Ove A. (2016-11-29). <u>"Biological Markers for Pulpal Inflammation: A Systematic</u>

16-Reisine S, Litt M. Social and psychological theories and their use for **dental** practice. Int Dent J. 1993 Jun

17-Ross, Michael H.; Kaye, Gordon I.; Pawlina, Wojciech (2002). Histology: a Text and Atlas (4th ed.). Baltimore: Lippincott Williams & Wilkins.

18-Schwendicke F, Frencken JE, Bjørndal L, Maltz M, Manton DJ, Ricketts D, Van Landuyt K, Banerjee A, Campus G, Doméjean S, Fontana M, Leal S, Lo E, Machiulskiene V, Schulte A, Splieth C, Zandona AF, Innes NP. Managing Carious Lesions: Consensus Recommendations on Carious Tissue Removal. Adv Dent Res. 2016 May;

19-Torabinejad, M. Walton, RE. Endodontics: Principles and Practice. 4th Edition. Elsevier Health Sciences, March 2008.

20-(Tru Blu Dentistry 2018)

https://www.trubludentistry.com/blog/five-stages-of-dental-decay/20352898.

21-V. Gopikrisha 2021 (Grossman endodontic practice)

22-Vimal K Siri 2017 Dental Caries

23-Zanini, Marjorie; Meyer, Elisabeth; Simon, Stéphane (2017-07-01). "Pulp Inflammation Diagnosis from Clinical to Inflammatory Mediators: A Systematic Review". Journal of Endodontics.

24- Zero DT. Sugars - the arch criminal? Caries Res. 2004 May-Jun