Oral pathology

Lecture 3

Disorders of the dental pulp (pulp diseases, pulpitis)

The most significant diagnostic problem that the dentists may face in their practice is to determine the extent of the pulp disease that has taken place within a symptomatic (painful) tooth.

An evaluation of the damage to the pulpal tissue is essential, since the pulp can neither be seen nor touched, an indirect assessment is required.

Inflammation is the single most important disease process affecting the dental pulp and accounts for virtually all pulpal diseases of any clinical significance. The decision to be made by the dentist based on clinical assessment of the pattern of pulp inflammation (pulpitis) is one of three:-

1- To restore the defective tooth structure ((conservative)).
2- To remove the pulp tissue ((endodontic)).
3- To remove the entire tooth.

In making such a decision, the clinician should decide whether the pulp damage ((pulpitis)) is reversible or irreversible pulpitis.

Pulpitis

The dental pulp is a delicate connective tissue, containing tiny blood vessels, lymphatic, myelinated, unmyelinated nerves, and undifferentiated mesenchymal cells like other connective tissues throughout the body; it reacts to noxious stimuli by an inflammatory response. This response is not significantly different from that seen in other tissues,

the final result can be different because of certain peculiar (anatomical) features of the pulp which includes:-

1-the pulp is enclosed within the calcified walls of the dentin, which precludes the excessive swelling of the tissue that occurs in hyperemic and edematous phases of inflammation in other tissues.

2-the blood vessels supplying the pulp tissues must enter the tooth through a tiny apical foramen, this precludes the development of an extensive collateral blood supply to the inflamed part.

Causes of pulpitis:-

1- Bacterial-caries in crown, periodontal pockets.
2- Traumatic-crown fractures, root fractures, partial avulsion, bruxism, abrasion.
3- Iatrogenic -Chemical-Thermal.

Heat generation, depth of preparation, dehydration of tubules, pulp exposure, and volatile/toxic disinfectant filling materials.Of these causes, the bacterial effects are the most important.

Bacteria can damage the pulp through toxins or directly after extension from caries or transportation via the vasculature ((this is a debatable issue))

Barotrauma (aerodontalgia)

Dental pain has been described by air crew flying at high altitudes in unpressurized aircraft. And in divers subjected to too rapid decompression following deep sea diving. **This pain has been attributed to the formation of nitrogen bubbles in the pulp tissues or vessels**, similar to the decompression syndrome elsewhere in the body, however, gas bubbles are seldom found in decompressed organs and the possibility of fat emboli from altered lipoproteins and platelet thrombi around the fat is suggested by some investigators. Aerodontalgia is really a marker of inadequate pulp protection

from the atmosphere and this usually means caries. It is not a direct cause of pulpitis, rather an exacerbating factor

Pulpitis can be classified as

- Acute or chronic
- Subtotal or generalized
- Infected or sterile

The best classification system is one that guides the appropriate treatment ((1 of the 3 choices))

Reversible pulpitis denotes a level of pulpal inflammation in which the tissue is capable of returning to a normal state of health if the noxious stimuli are removed.

Irreversible pulpitis implies that a higher level of inflammation has developed in which the dental pulp has been damaged beyond the point of recovery

When external stimuli reach a noxious level, degranulation of mast cells, decreased nutrient flow and cellular damage occur. Numerous inflammatory mediators (histamine, bradykinin, and prostaglandins) are released. These mediators cause vasodilation, increased blood flow, and vascular leakage with edema. Normally this should promote healing through removal of inflammatory mediators. However, the dental pulp exists in a very confined area.

If the inflammatory process continued for an extended period of time can lead to increased pulp injury or even death of the pulp.

Previous studies suggested that the associated increased vascular pulpal pressures could compress venous return and lead to (self-strangulation) and pulp necrosis.

Recent studies recognize that the associated increased vascular pulpal pressures could compress venous return and lead to (self-strangulation) and pulp necrosis.

Recent studies recognize that the increased fluid pressure usually is localized to the area of inflamed pulp immediately adjacent to the affected dentin, increased interstitial pressure in area of inflammation leads to increased flow of fluid back into capillaries of adjacent uninflammed tissue and increased drainage. In this manner, the increased fluid pressure from inflammation is counteracted and typically does not lead to generalized increase in pulp fluid pressure, effectively preventing (self-strangulation).

According to the above mentioned explanation of the pulp response to injury, it seems that the pulp defense mechanisms may work well with many mild-moderate injuries and rarely result in widespread necrosis. Localized pulp abscesses may heal after eliminating the injury and formation of reparative dentin, however sever localized pulpal damage can overwhelm the system, leading to pulp necrosis.

1-Reversible pulpitis (focal reversible pulpitis)

This denotes that the pulp is capable of full recovery if the irritating factors subside or removed.

The symptoms reflect an **irritated pulp** tissue that reacts with the **mildest** and **earliest forms** of the inflammatory response, consisting of **vasodilation, transudation,** a slight infiltrate of **acute inflammatory** cells underlying the area of affected dentinal tubules. Tertiary dentin may be noted in the adjacent wall.

On clinical examination the pain is mild-moderate in intensity and responds to sudden change in temperature. The **pain generally remains for 5-10 minutes** and seldom lasts longer than 20 minutes. The tooth remains symptomless until it is stimulated again. Changing body positions do not affect the pattern of pain, or duration of pain. The pain is mostly provoked by cold, although hot, sweet, or sour food may also cause pain.

The tooth responds to electric pulp testing **at lower levels** of current than normal tooth. Percussion and mobility tests are negative. If the tooth is treated, the condition is reversible and the pulp will heal, if pulpitis is allowed to progress, then irreversible pulp damage will occur.

2-Irreversible pulpitis

The patient with early irreversible pulpitis presented with sharp, sever pain on thermal stimulation, and the pain continues after removal of the stimulus. Cold is the most uncomfortable, although heat or sweet and acidic food can cause pain. The pain may be spontaneous or continuous and may be exacerbated when the patient lies down. The tooth responds to **electric pulp testing at lower levels** of current. At this stage (early), the pain often can be **localized easily** to the individual affected tooth. **With time the patient discomfort is increasing and can no more be able to identify the offending tooth**

In the later stages of irreversible pulpitis, the **Pain increases** in intensity and experienced **as throbbing**, which keeps the patient awake at night. At this point **heat increases the pain**, while cold may produce relief. The tooth responds to electric **pulp testing at higher levels** of current or demonstrates no response. Mobility and sensitivity to percussion are negative.

Histopathological features of irreversible pulpitis

Irreversible pulpitis often demonstrates congestion of the venules that results in focal necrosis. This necrotic zone contains polymorphonuclear leukocytes and histiocytes. The surrounding pulp exhibits fibrosis and a mixture of plasma cells, lymphocytes and histeocytes.

3-Chronic hyperplastic pulpitis

This is a unique pattern of pulpal inflammation, it occurs in **children and young** adults who have large exposures of the pulp in which the **entiredentinal roof often** is missing. The most frequently involved teeth are the **deciduous or permanent** molars, which have large pulp chambers in these age groups. Mechanical irritation and bacterial result in a level of chronic inflammation that produces **hyperplastic granulation** tissue that extrudes from pulp chamber and often fills the associated dentinal defect. The **apex may be open and reduces the chance of pulp necrosis** secondary to venous compression. The tooth is asymptomatic except for a feeling of pressure on mastication.

Histopathological features of chronic hyperplastic pulpitis

This demonstrates a cap of **subacutely inflamed granulation** tissue that fills the entire space of the original pulp chamber. The surface of the polyp may or may not be covered with **stratified squamous epithelium**, which migrates from the adjacent gingiva or arise from sloughed epithelium within the oral fluids. The deeper pulp tissue within the canals typically demonstrates **fibrosis and chronic** inflammation.

The process of irreversible pulpitis may be acute or chronic pulpitis.

Acute pulpitis-this may be **a progression of focal reversible** pulpitis or may present as an acute exacerbation of an already **established chronic pulpitis**. Pulpal damage may range in severity from simple acute inflammation marked by vessels dilatation, exudation and neutrophil chemotaxsis to focal liquefaction necrosis (pulp abscess) to total pulp suppurative necrosis.

Chronic pulpitis-this is an inflammatory reaction that results from **long term low grade injury** or occasionally from quiescence of an **acute process symptoms**,

characteristically mild and often intermittent, appear over an extended period. A **dull pain** may be the presenting complaint, or the patient may have no symptoms. As the pulp becomes necrotic, responses to thermal and electric stimuli are reduced.

Histopathological features

Lymphocytes, plasma cells and fibrosis appear in the chronically inflamed pulp. If there is an acute exacerbation of chronic process, neutrophils will be seen.

Pulp necrosis

Pulp necrosis may follow either pulpitis or a traumatic injury to the apical blood vessels cutting off the blood supply to the pulp. A **coagulative** type of necrosis is seen after ischemia; trauma and the patient usually have no symptoms. If the necrosis follows pulpitis then breakdown of the inflammatory cells may lead to **liquefactive** type of necrosis which may become infected by bacteria from caries, this type is usually associated with foul odour when opened with endodontic treatment.

Diagnosis of pulp pain

The diagnostic procedures that are commonly used to assess the status of a symptomatic tooth and pulp are as follows.

1-history and nature of pain.

2-visual clinical examination.

3-reaction to thermal changes.

4-reaction to electric stimulation.

5-reaction to tooth percussion.

6-radiographic examination.

7-palpation of the surrounding area.

The diagnosis of pulp pain (pulpalgia) is made from a combination of all the above mentioned points. The value of these tests is sometimes less than optimal for e.g. when the procedures demonstrate that the pulp is disease free, the results are highly reliable. However, when a pulp appears to test positive for irreversible pulpitis, the histopathological examination may demonstrate no obvious evidence of pulp disease.

For this reason the entire test available should be used to reach a diagnosis aided by the personal judgment and experience of the dentist.

Future improvements in diagnostic methods such as laser Doppler flowmetry and pulse eximetry devices may help to increase accuracy.

Laser Doppler flowmetry

Laser Doppler Flowmetry is an inexpensive, noninvasive method of measuring the continuous circulation of blood flow on a microscopic level. This method of measuring the flux of red blood cells is growing in practical application.

Pulse Oximetry Technology:

The principle of pulse oximetry is based on the red and infrared light absorption characteristics of oxygenated and deoxygenated hemoglobin. Oxygenated hemoglobin absorbs more **infrared light** and allows more red lights to pass through. Deoxygenated (or reduced) hemoglobin absorbs more **red light** and allows more infrared light to pass through. Red light is in the 600-750 nm wavelength light band. Infrared light is in the 850-1000 nm wavelength light band.

Numerous disorders have been reported to mimic pulpalgia, e.g. migraine, headache, myofacial pain and angina pectoris. If these conditions are not considered then the results would be sequential extractions or endodontic treatment which is all not needed and inappropriate.

Treatment and prognosis:-

Reversible pulpitis--- removal and elimination of the cause, on occasion analgesics are required. Prognosis is good if action taken early. Pulp testing is essential periodically to ensure that irreversible damage has not occurred.

Irreversible pulpitis---both acute, chronic, chronic hyperplastic are treated by endodontic treatment or extraction

PULPAL CALCIFICATIONS

Calcifications within the dental pulp are not rare, but the frequency is difficult to determine. There are strong association between **long-standing chronic pulpitis** and the presence of pulpal calcification; in addition, the prevalence of pulpal calcifications increases **with age**. However, some examples appear to be developmental with a **familial tendency**.

There are three types of pulpal calcifications. All pulpal calcifications start out as **free bodies within the pulp** tissue, but many may become **attached** or **embedded in the dentinal walls of the pulp**.

1.**Denticles** Denticles consist of tubular dentin surrounding a central nest of epithelium..

2.**Pulp stones** develop around a central nidus of **pulp tissue** (e.g., collagen fibril, ground substance, necrotic cell remnants). Pulp stones are formed within the coronal portions of the pulp and may arise as a part of age related or local pathologic changes. Most pulp stones develop after tooth eruption

3.**Diffuse linear calcifications** exhibit areas of fine, fibrillar, **irregular calcification** that often parallel the vasculature. These calcifications may be present in the pulp chamber or canals, and the frequency increases with age.

CLINICAL AND RADIOGRAPHIC FEATURES

Denticles and pulp stones can reach sufficient size to be detected on intraoral radiographs as **radiopaque** enlargements within the pulp chamber or canal. Diffuse calcifications are not detectable radiographically. Other than rare difficulties during endodontic procedures**, pulpal calcifications are typically of little clinical significance.**

TREATMENT AND PROGNOSIS

No treatment is required.

Pulp obliteration is also seen in dentinogenesis imperfect and dentinal dysplasia.

Age changes in the pulp

The volume of the **pulp gradually decreases** with the age due to the continued **production of secondary dentine**, **decreased vascularity**, **reduction in cellularity** and **increase in collagen fiber** content have been reported, and these changes may impair the response of the tissue to injury and its healing potential.

It is generally accepted that the prevalence of the **pulp stones and diffuse calcification increase with age** but the evidence for this is inconclusive.