

DENTAL CARIES

AETIOLOGY, EPIDEMIOLOGY, CLINICAL
PRESENTATION, MANAGEMENT AND
PREVENTION.

LECTURE TO MBChB

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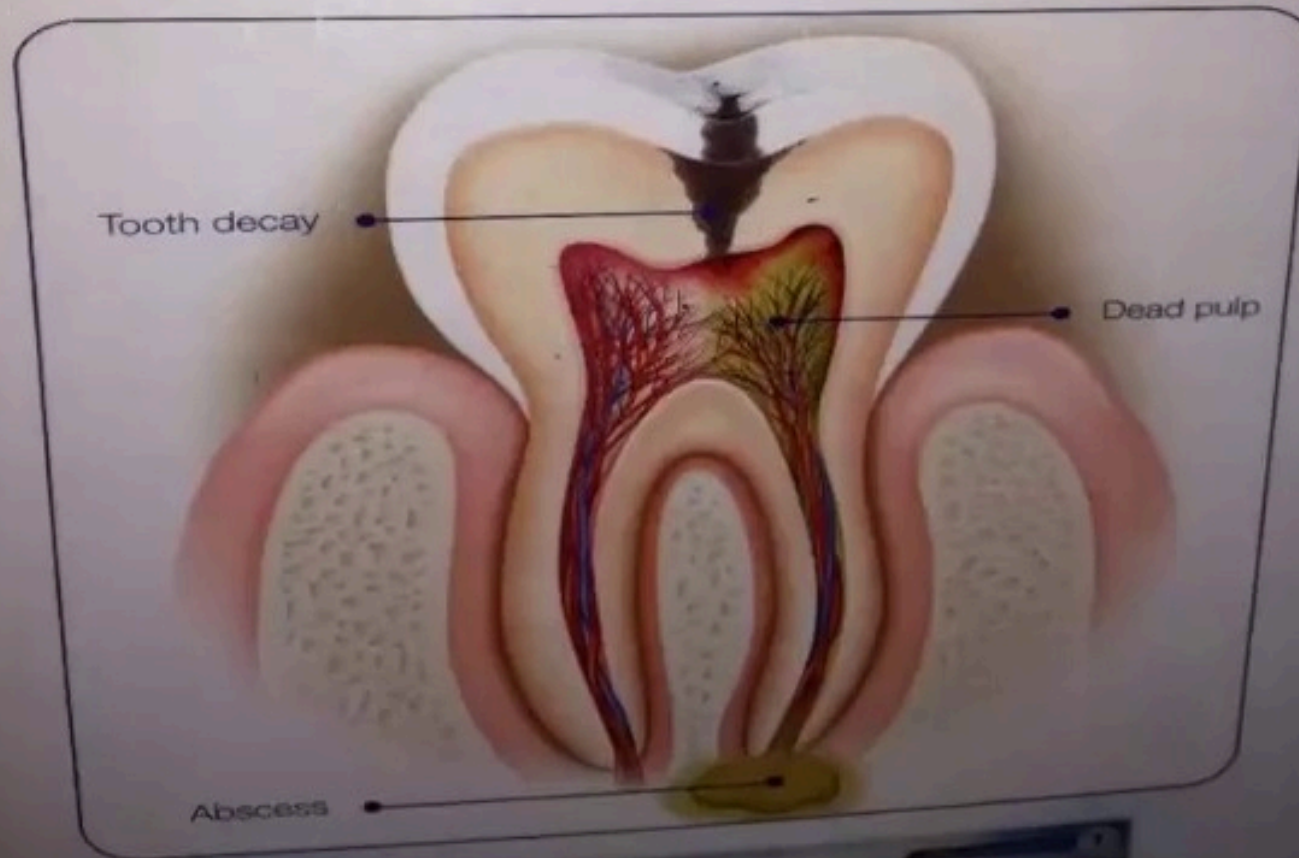
Healthy tooth

If your gums recede or your enamel wears down, dentine is exposed and your teeth can become sensitive to hot and cold and cause you pain.



Decayed tooth

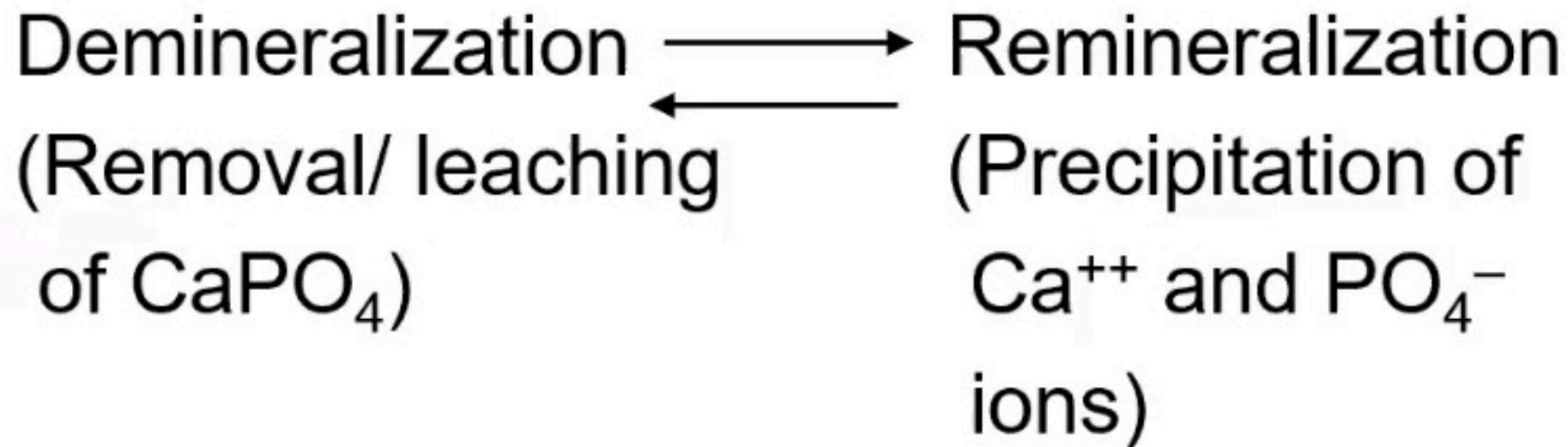
Tooth decay is caused by specific bacteria in the mouth, which converts sugar and carbohydrates in the diet into acid. This acid gradually dissolves the tooth resulting in tooth decay.



DENTAL CARIES

Definition: Disease of the hard tissues of the teeth characterized by the demineralization of the inorganic component of the tooth followed by the breakdown/dissolution of the organic component.

A Dynamic Process



Demineralization lead to softening and weakening of the affected tooth surface.

AETIOLOGY

Dental Caries (DC) is a Product of a complex interaction among 4 major factors:

- Bacterial agents concentrated in plaque
- Suitable substrate
 - Mainly sucrose
- Susceptible Host / tooth (enamel e.t.c.)
- Time factor

4 factors form an **Ecosystem**. All factors must be present for DC to occur.

DENTAL CARIES ECOSYSTEM

Plaque Bacteria

Susceptible Host
(Tooth enamel)

Time Factor

Substrate
(e.g. Sucrose)

DENTAL CARIES

PREVENTIVE MEASURES AIMED AT
UPSETTING THE ECOSYSTEM

Role of the Four Factors

1) **Plaque Bacteria.** The most important are:

- Acidogenic bacteria– acid producing
- Aciduric bacteria– ability to survive in low pH.

The most important specific bacterial organisms associated with DC:-

- i. Streptococci mutans
- ii. Lactobacilli Acidophilus
- iii. Actinomyces.

2) **Suitable Substrate**

Mainly refined/ Fermentable carbohydrates. Sucrose – most important Substrate for bacterial metabolism resulting in acid production e.g lactic and pyruvic acids.

Sucrose—The Arc Criminal

Dietary practices that increase risk of dental caries

- a) Diet rich in fermentable CHO.
- b) Frequency of consumption
- c) Timing of intake –between meals
:favour growth of Lactobacilli
- d) Bed-time snacks.

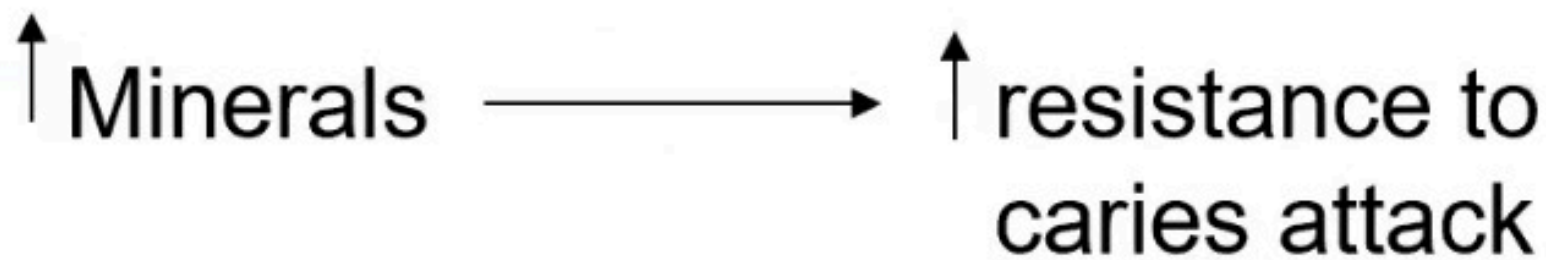
3) **Susceptible Host**

Tooth enamel that is susceptible to dissolution by acids.

Characteristics of teeth that increase susceptibility to dental caries:-

- i. Tooth morphology – pits, fissures, grooves – not cleansable.

- ii. Tooth composition – Quantity of minerals/ inorganic matter in relation to organic matter.



4. Time Factor

There must be sufficient time and repeated attack from bacterial acids with insufficient time for recovery.

Demineralization with no sufficient time for Reminerization

INTERACTION AND CONSEQUENCES

Bacteria on
Tooth surface

Sucrose

Fermentation

Acids (Pyruvic, Lactic)

Ca^{++} , PO_4^{--}
 OH^- , F^-

Demineralization



Remineralization

Time factor

Progressive Destruction
of Tooth Substance

Time

TOOTH MORTALITY

EPIDEMIOLOGY

Indices of measuring Dental Caries

1. **DMF(T) Index**. Measures caries experience in permanent teeth i.e the number of teeth decayed (D), missing due to caries (M) and filled due to caries (F)

(D=3; M=1; F=0 : DMF(T) Score = 4)

2. **dmf(t)** index measures caries experience in deciduous teeth

Dental Caries Prevalence and Experience in Kenya

- The Kenya National Oral Health Report of 2015 showed the following:

Children

Age group (yrs)	Prevalence (%)	Caries Experience (DMF(T)/ dmf(t))
Total for 5, 12, 15yrs	23.9	0.8
5 years only	46.3	1.87

Adults

Age group (yrs)	Prevalence (%)	Caries Experience (DMF(T))
34 – 44 (adults)	-	0.56
60 + years (elderly)	-	0.9
Overall	34.3	0.72

- By international standards, Dental caries experience in Kenya is lower than that of many Developed countries.
- However, evidence from various studies show caries experience in Kenya is increasing

Sugar consumption and dental caries in Kenya

Macigo F.G, James R.M, Ogunbodede E
and Gathece L.W

International Dental Journal 2016

Findings showed Dental Caries in Kenya is increasing due to increasing sugar consumption

- i. Per capita sugar consumption increased from 35.5 g/day in 1969 to 60.8g/day in 2009

Critical sugar consumption 50 g/day

ii. Dental caries experience in deciduous teeth in children 3 – 5 Yrs increased from dmft/ index of 1.5 in 1980s to 2.95 in the early 2000s

Caries prevalence increased from 43.2% to 59.5%

iii. Dental Caries experience for permanent teeth at 12 yrs of age increased from DMFT of 0.2 to DMFT of 0.92 over same period

Caries prevalence increased from 11.7% to 44.5% over same period

OTHER RISK FACTORS PREDISPOSING TO DC

1. **Age:** DC may occur at any age post eruption of Deciduous and Permanent teeth.

Prevalence increases with age due to the cumulative effect of the disease

2. **Sex:** Many studies have demonstrated that females have a higher dental caries prevalence than males.

Also demonstrated by the 2015 Kenya National Oral Health Survey

Why? (Early eruption of teeth in girls, dietary habits)

3. OTHER FACTORS INCLUDE:

- ❖ Familial/ Hereditary Factors—role of genetic factors.
- ❖ Emotional disturbances- affecting mental health – high caries experience.
- ❖ Ethnicity and Race.
- ❖ Culture and religion
- ❖ Socio-Economic factors
- ❖ Low level of parental education especially the mother

- ❖ Unemployment status of parents.
- ❖ Low family income.
- ❖ Single parent hood.
- ❖ Geographic Factors.
- ❖ Malnutrition.
- ❖ Deficient quantities of dietary micro elements e.g fluorides, calcium
- ❖ Systemic illnesses
- ❖ Immunodefficiencies
- ❖ Diseases, Drugs with manifestation of Xerostomia

CLINICAL PRESENTATION

- i) First clinical sign of the process of dental caries is white spot/ white opaque area of the tooth surface (incipient carious lesion)

- ii) With time, the lesion may become brown, grey or dark in colour

iii) If the lesion progresses, there is a breakdown of the tooth surface [organic component] forming a physical defect leading to formation of a cavity

Symptoms

- Mild to severe sensitivity due to thermal, chemical changes or tactile touch
- Mild to severe pain especially at night
- Discolouration on the tooth surfaces,
- Cavities on tooth surfaces
- Food sticking between teeth-proximal cavities.
- Resulting complications such as bleeding or swelling of gums, Mandible, Maxilla

Symp...

In children:

- anxiety, fear
- refuse to feed
- loss of sleep
- uncooperative behaviour
- loss of attention

Diagnosis

1) **Visual inspection** (tooth must be clean & dry). Look for:

- white opaque spots, brown, grey discoloured areas
- Physical defects
- Discontinuity of tooth surface
- Frank cavities with or without discolouration
- Food impaction between teeth

carious lesion on the mesial aspect of a premolar
on the buccal surface of the tooth. Instead, dental
has been partly worn away. The lesion has forma
tion next to a removable partial denture. 19





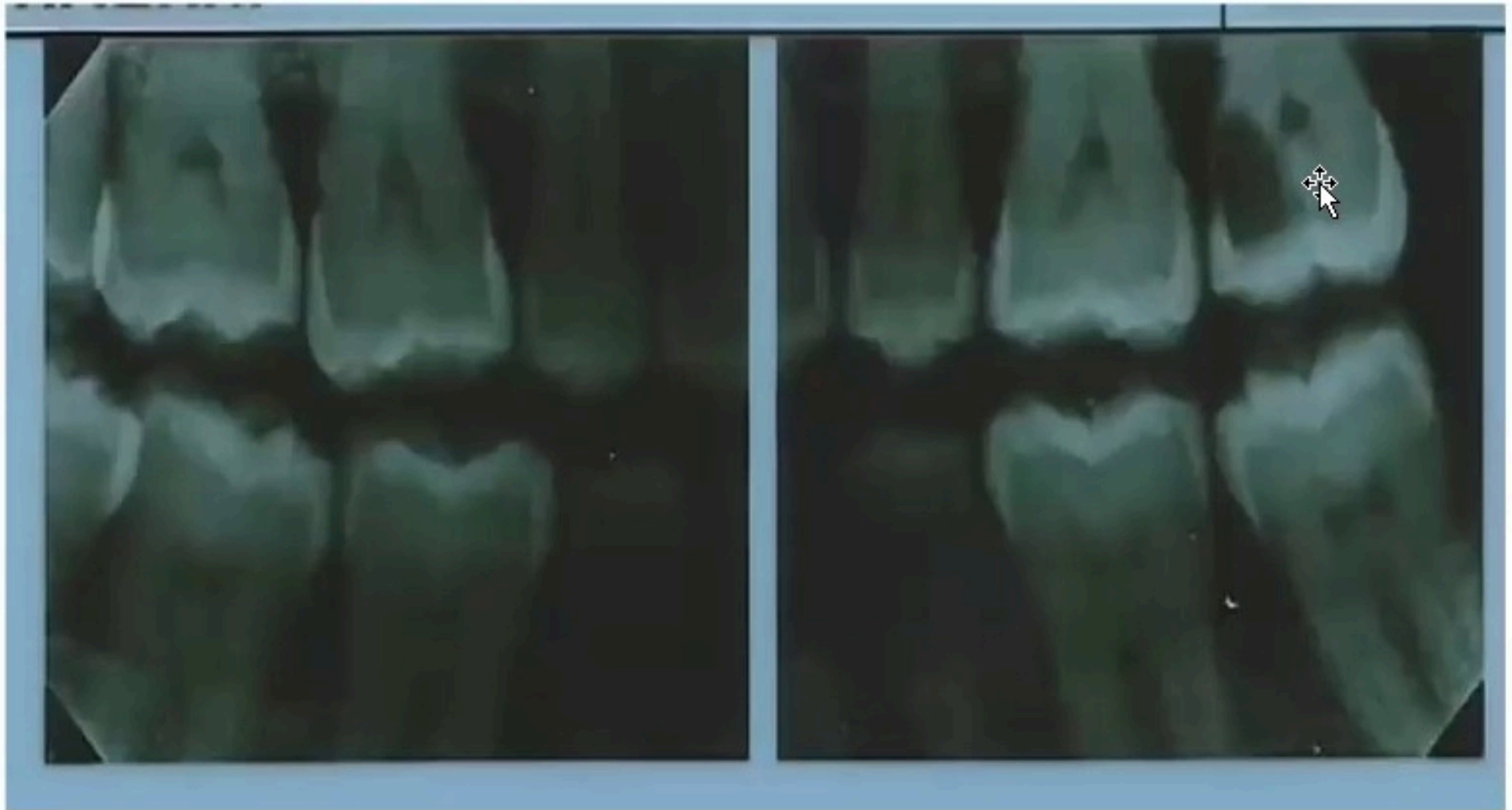




2. **Use of diagnostic tools** – mirror and probe. Sickle shaped probe
- the probe usually catches on a softened floor of a cavity (resists withdrawal)



3. **Bitewing radiographs.** DC Appears as radioluscent lesions on one or several surfaces of a tooth



4. Exploration/Trial cavity

Where clinical and Radiographic examination fail to detect a cavity but symptoms persist.

MANAGEMENT

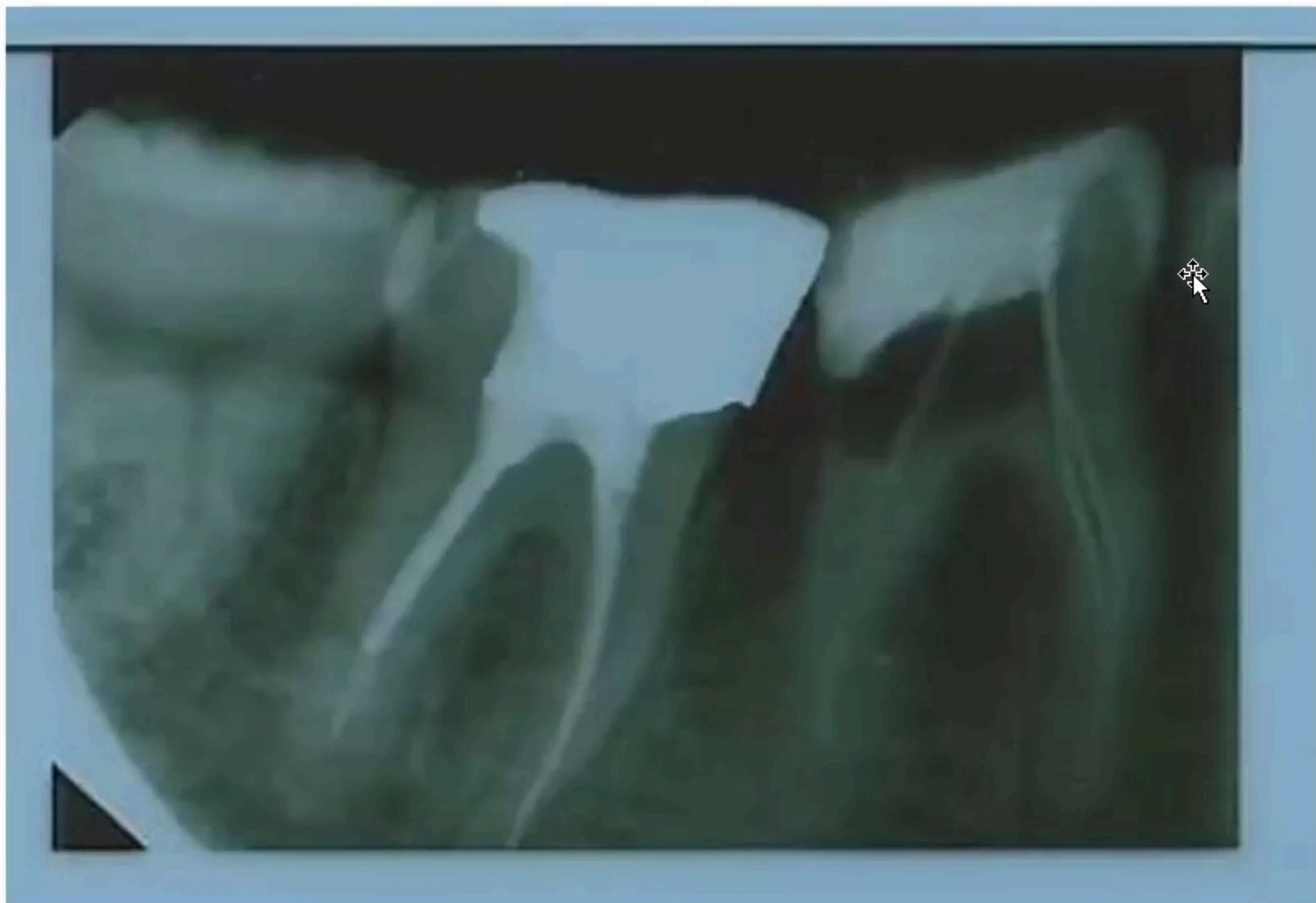
1. Incipient Carious Lesion with no cavitation
 - Fluoride application to reverse the lesion.

2. Removal of carious lesion and filling to:

- Repair the damage
- Restore physical appearance/ aesthetic
- Restore function

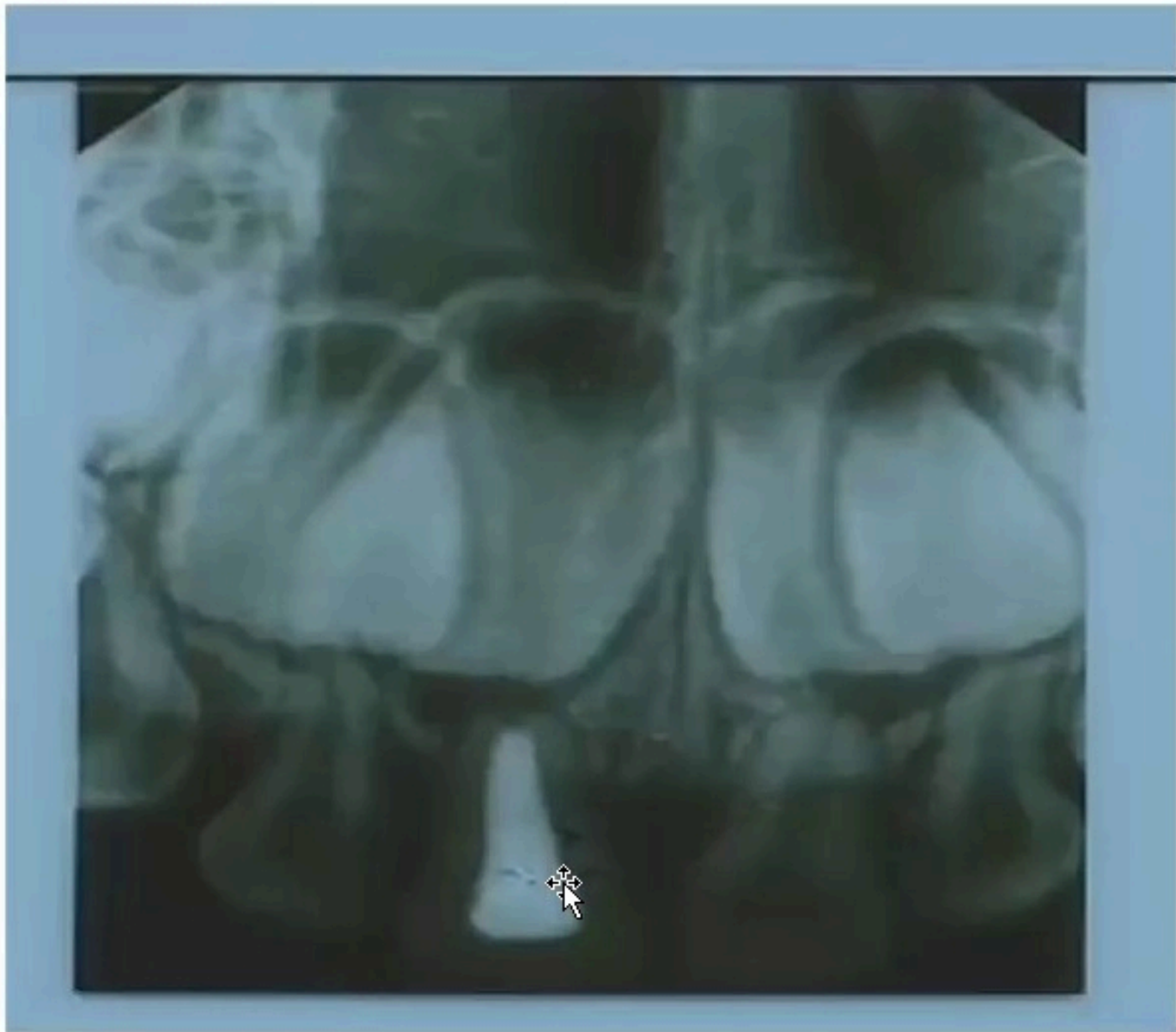


3. Root Canal Therapy and Filling where there is pulpal involvement



ROOT CANAL FILLED PREMOLARS

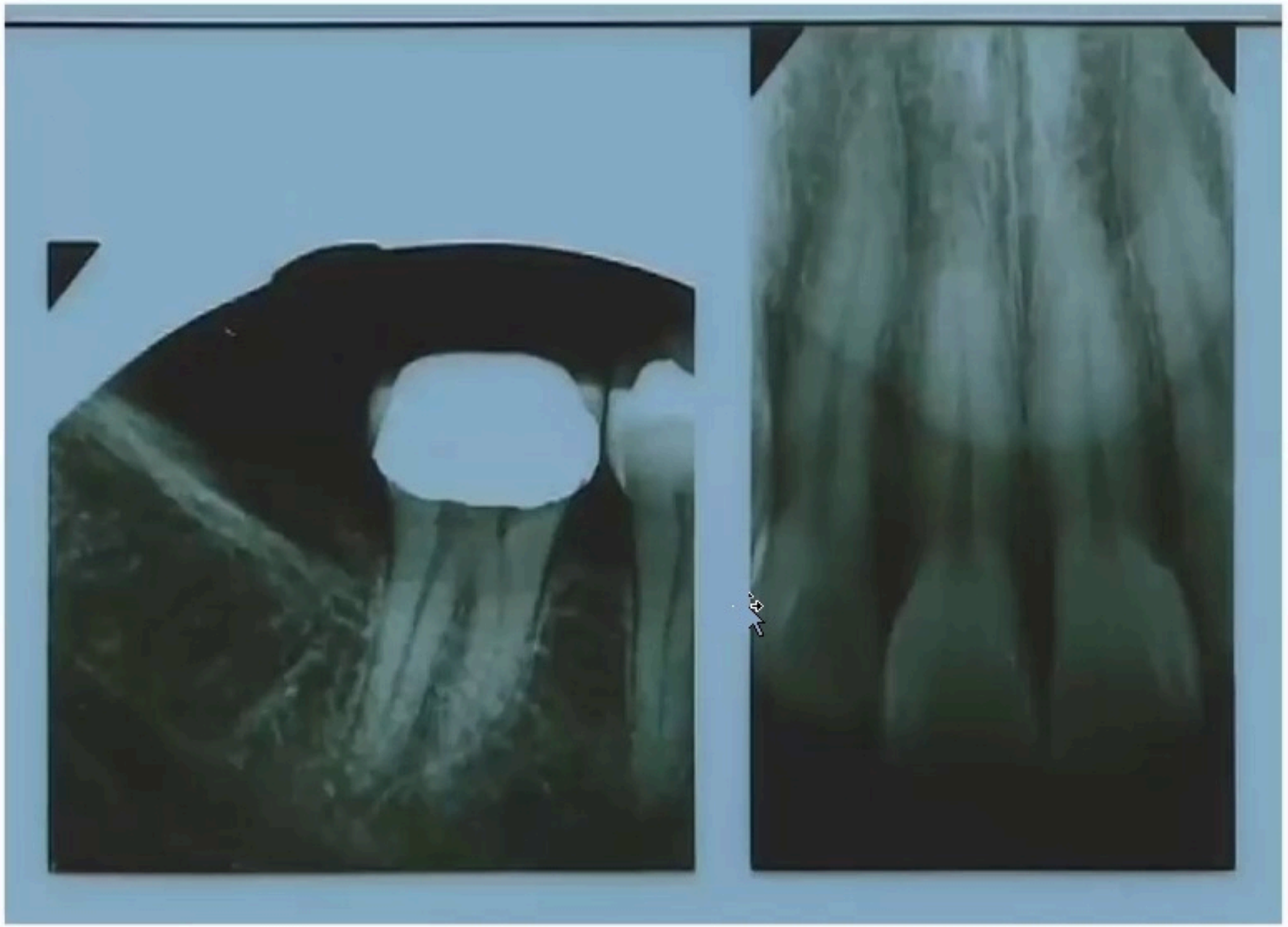




4. Surgical intervention where there is associated pathological lesion e.g. periapical abscess with bone loss

5. Crowning of decayed tooth

Fabrication of Artificial crown and fixation where there is marked destruction of the natural crown by dental caries



6.Tooth Extraction

When other methods of treatment are not possible, not accessible, not affordable, not available or on patient demand

PREVENTION OF DENTAL CARIES

Basis of Prevention: Aetiologic model is well established.

Justification/ Rationale for Prevention

1. High Prevalence Rate
2. Local Consequences of the disease
 - Source of pain involving intra and extra oral tissues
 - Loss of function
 - Loss of aesthetics
 - Tooth mortality
 - Life threatening mandibular and maxillary infections

3. Consequences on general Health

- Generalised pain and suffering where there is a systemic spread of the infection
- Malnutrition, poor general health and lowered immunity leading to vulnerability to other diseases.
- Adverse pregnancy outcomes including Low Birth weight
- Respiratory diseases

..General Health

- Cardiovascular diseases: increases risk of heart disease by almost 3 times
- May lead to life threatening infection of the Brain
- May lead to gangrenous oral facial infections such as noma, Cellulitis such as Ludwings Angina and infection of the mediastinum which can lead to death

4. Economic impact. Leads to demand for prevention by

- Public
- Governments
- Non Governmental Organizations

5. Curative Dentistry creates a vicious circle of increasing treatment needs.

- Poor
 - Accessibility
 - Affordability
- High cost
 - Individuals
 - Families
 - Governments

METHODS OF PREVENTION

A) Communities Based Methods

1.0 Use of Fluorides

The most effective means of Dental caries prevention in community based programmes.

1.1 Mechanism of Fluoride action

- i. Reduction in susceptibility of tooth enamel to dissolution by acids
- ii. Interference with plaque bacterial metabolism and growth.
- iii. Enhancement of remineralization
 - Repair of early carious lesions.

1.2 Methods of Fluoride use.

- a) Fluoridation of Public water supplies.
-----**controversial**
- b) Fluoridation of school water supplies.
- c) Fluoridation of salt
- d) Fluoridation of milk. U.K., Chile, China, Russia--- school milk programs for ages up to 6 yrs

1.3 Limitations in Kenya

- i. Variation in distribution of Fluorides
- ii. Requires piped water supplies. 78% have no piped water (86% for rural areas).
- iii. Lack of adequate data on dietary sources of Fluorides
- iv. Lack of official policy
- v. Logistics and variation in consumption patterns e.g. in milk.

2.0 Promotion of Health diet/ Control of Cariogenic diet

a) **Food modification:** Substitution of sucrose with non-cariogenic sugar sweeteners e.g. Xylitol, sorbitol.

b) **Legislation and Regulation**

Aim. Control of production, labelling, advertising, marketing. Influence consumption patterns.

c) **Public Health Education**

Aim: Inculcate better dietary habits.

d) **Ban/ Restrictions**

Sale of cariogenic foods to vulnerable groups – school children.

2.1 Limitations of control of Cariogenic Diet

- i. National economic considerations
- ii. Monetary interest of powerful groups of manufacturers
- iii. Biological needs

iv. Nature and safety of cariogenic sugar substitutes

- Tastes and preferences
- May be rejected.

B) Individual Based Methods

1. Use of Fluorides

a) Supervised F^- use (in Children)

- i. F^- mouth rinsing in schools based programs (≥ 7 yrs old)

- ii. Fluoride tablets. Children \leq 13 yrs
 - iii. Brushing with Fluoride solutions, gels.
- b) Professionally applied Topical Fluorides

2. Prophylaxis – Professional plaque removal 3-6 Months.

c) Individual Self Care

- i. Fluoride tooth pastes in oral hygiene measures
- ii. Fluoride Mouth rinses ≥ 7 yrs of age
- iii. Dietary control by individual
- iv. Self performed mechanical oral hygiene measures (brushing, flossing, use of interdental brush).

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