IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 19, Issue 8 Ser.12 (August. 2020), PP 01-08
www.iosrjournals.org

Dental Caries

Dr. Sadhvi Gupta[1], Dr. Megha Mahajan[2], Ishita Khanna[3], Dr. Aasiya Yousuf[4], Dr. Anshu Gupta[5], Dr. Gursimran Singh Pabla[6], Dr. Divya Jakhar[7]

[1,6,7]*(Post Graduate Student, Department of Conservative Dentistry and Endodontics, Genesis Institute of Dental Sciences and Research, India)*

[2]*(Dental Practitioner, Nutrition and Diet expert, India)*

[3]*(Under Graduate Student, Genesis Institute of Dental Sciences and Research, India)*

[4]*(Post Graduate Student, Department of Pediatric and Preventive Dentistry, Genesis Institute of Dental Sciences and Research, India)*

[5]*(Post Graduate Student, Department of Conservative Dentistry and Endodontics, Maharishi Markandeshwar College of Dental Sciences, India)*

Abstract:
Dental caries is a unique chronic disease among human which is one of the most common important global oral health problems in the world today. It is the destruction of dental hard acellular tissue by acids by-products from the bacterial fermentation of dietary carbohydrates especially sucrose. It progresses slowly in most of the people which results from an ecological imbalance in the equilibrium between tooth minerals and oral biofilms which is characterized by microbial activity, resulting in fluctuations in plaque pH due to bacterial acid production, buffering action from saliva and the surrounding tooth structure. The microbial community of caries is diverse and contains many facultatively and obligately-anaerobic bacteria. S. mutans is the most primary associated with it.Dental disease has been associated with low self-esteem,adverse pregnancy outcomes, and increased risk of myocardial infarction, cardiovascular,respiratory, erectile, diabetes complications, cavernous sinus thrombosis and Ludwig angina which can be life threatening. Treatment, prevention, personal hygiene and dietary modification should be recommended for dental caries patients.

Key Word: *Dental caries, Histology, Etiology, Diagnosis, Prevention, Oral health.*

Date of Submission: 07-08-2020 Date of Acceptance: 21-08-2020

I. Introduction:

Teeth are tools that have evolved to ensure survival of species.[2] Survival of all higher forms of life, depend on ingestion of food, its digestion and absorption, which acts as fuel for various life processes.[1]

The word **caries** is derived from Latin, meaning "*rot or decay*".[3] It is similar to Greek word '*ker'*meaning death. Every individual, in his life-time has been effected by this disease. In Japanese, the caries is known by term called, mush-*ha* meaning hollow teeth.[4]

In ancient humans' caries was usually located at the cemento-enamel junction or in the cementum, whereas in modern man grooves and fissures are the most common sites of decay.[5]

Hundreds of dental research investigators for more than a century have studied various aspects of dental caries problems. Despite this extensive investigation, many aspects of etiology are still under observation and the efforts at prevention have been partially successful.[6]

The etiology of dental caries is generally agreed to be a complex problem complicated by many indirect factors, which obscure the direct cause or causes. There is no universally accepted opinion for the etiology of dental caries.[7]

DEFINITION OF CARIES:

Various authors have defined caries in the own way,

According to Sturdevant's:

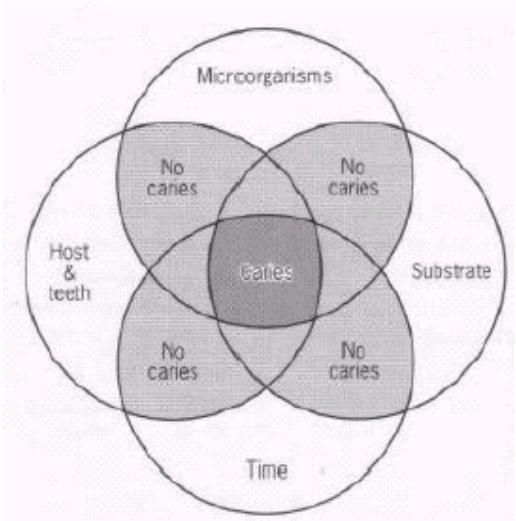
Dental caries is an infectious microbiologic disease of the teeth that results in localized dissolution and destruction of calcified tissues.[8]

Shafer, Hine,Levy has defined it as a "microbial disease of the calcified tissues of the teeth, characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth.[9]

WHO

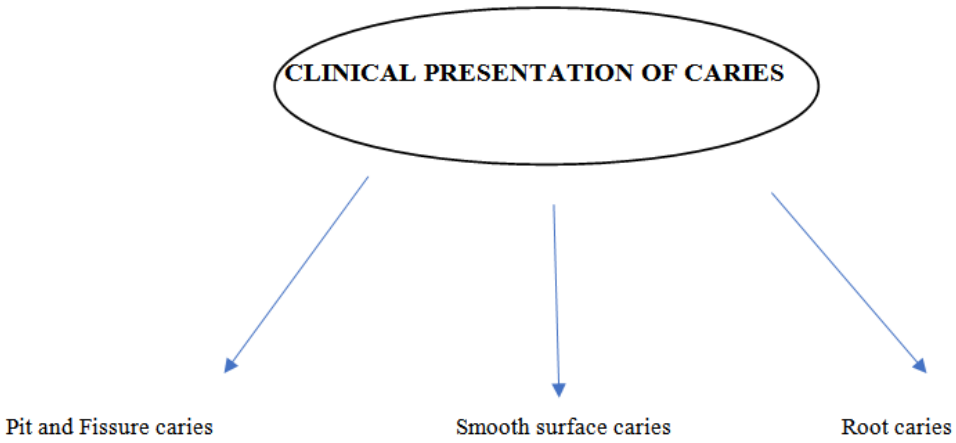
Dental caries is defined as a localized post eruptive, pathological process of external origin, involving softening of hard tooth tissues, and proceeding into the formation of cavity. [10]

ETIOLOGY OF DENTAL CARIES[14,15]	
PRIMARY FACTORS	**MODIFYING FACTORS**
➢ Tooth ➢ Dental Plaque ➢ Diet ➢ Time	➢ Saliva ➢ Systemic Health ➢ Sex ➢ Heredity ➢ Race ➢ Occupation



❖

❖ The characteristics of carious lesions vary according to the surface on which they develop:[28]



HISTOLOGY OF CARIES:[16,17]

➢ **Smooth surface caries**

The earliest manifestation of incipient caries of the enamel is the appearance of an area of decalcification which resembles a smooth chalky white area.

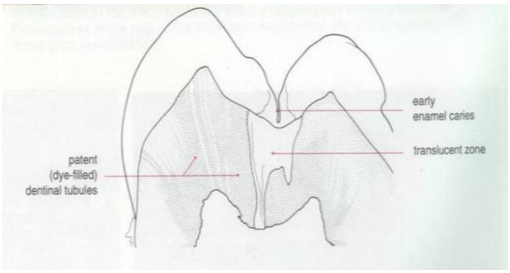
The first change seen is usually the loss of the interprismatic or interred substance of the enamel with increased prominence of the rods.

As the process advances and involves deeper structure, it forms a triangular/ cone shaped lesion with the apex towards the surface of the tooth.

ZONE 1:*TRANSLUCENT ZONE*

The translucent zone of enamel caries lies at the advancing front of the lesion and is the first recognizable zone of alteration from normal enamel. The zone appears translucent because the spaces or pores created in the tissue in this first stage of enamel caries are located at prism boundaries and other junctional sites.

Therefore, when the pores are filled with a medium like quinoline having the same refractive index as enamel, normal structural markings are no longer visible. They have a pore volume of 1% compared to about 0.1% for sound enamel. The fluoride content of translucent zone enamel was found to be increased relative to adjacent sound enamel. No evidence of protein loss is seen in this zone. Carious attack preferentially removes magnesium and carbonate rich mineral from the translucent zone and not organic material.



*Translucent zone:*In this section dye applied to the pulpal surface of the dentin has tracked. Outwards along the patent tubules. A translucent zone has begun to form but does not extend Through the full thickness of the dentin. The outer parts of those tubules are calcified and thus impermeable to the dye.

ZONE 2:*DARK ZONE*

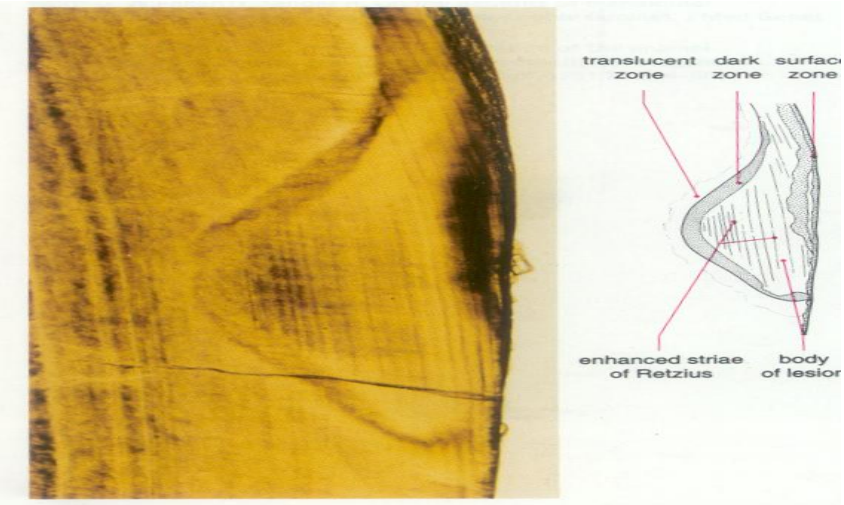
The dark zone is the second zone of alteration from normal enamel and lies just superficial to the translucent zone. It appears dark brown in ground sections examined by transmitted light after imbibition with quinoline Polarized light studies show that it has a pore volume of 2% to 4%.

Remineralization Experimental studies done by Silverstone et al (1977) supports the concept that the carious process in enamel is a dynamic one, with phases of demineralization alternating with phases of remineralization, rather than a more simple process of continuing dissolution.

The size of the dark zone is probably an indicator of the amount of reminerlization that has recently occured.

ZONE 3:*BODY OF THE LESION*

The body of the lesion is the largest portion of carious enamel in the small lesion.
It is the area positioned superficial to the dark zone and deep to the relatively unaffected surface layer.
It has pore volume,varying from 5% at the periphery to 25% at the center. The striae of Retzius are well marked in the body of the lesion indicating preferential mineral dissolution along the areas of relatively higher porosity.
The first penetration of caries enters the enamel surface via the striae of Retzius. The inter prismatic areas and these cross- striations provide access to the rod (prism) cores, which are then preferentially attacked.
Bacteria may be present in this zone if the pore size is large enough to permit their entry.



Ground section shows the body of the lesion, containing Enhanced striae ofretzius ,enclosed between the dark and peripheral translucent zones and the intact surface.

ZONE 4:SURFACE ZONE

It is relatively unaffected by the caries attack. It has lower pore volume than the body of the lesion (less than 5%) and a radiopacity comparable to unaffected adjacent enamel.

It has been hypothesized that hyper mineralization and increased fluoride content of the superficial enamel are responsible for the relative immunity of the enamel surface. However, removal of the hyper mineralized surface by polishing fails to prevent the reformation of a typical well mineralized surface over the carious lesion. Thus, the intact surface over incipient caries is a phenomenon of the caries demineralization process rather than any characteristics of the superficial enamel.

As the enamel lesion progresses conical shaped defects in the surface zone can be seen by SEM. These are the first sites where bacteria can enter into a carious lesion. Arresting thecaries process at this stage results in a hard surface that may at time be rough though cleanable.

- **Pit and fissure caries**

The carious process in pits and fissures does not differ in nature from smooth surface caries
Food stagnation and with bacterial decomposition at the base is expected.
Usually the enamel at the base of the fissure is relatively thin, so early dentineinvolvement frequently occurs.
The enamel rods flare laterally in the bottom of the pits and fissures. Caries follows the direction of the enamel rods and characteristically forms a triangular or cone shaped lesion with its apex at the outer surface and its base towards the DEJ.

CARIES OF THE DENTIN:

Caries of the dentin begins with the natural spread of the process along the dentino-enamel junction and the rapid involvement of great numbers of dentinal tubules, each of which acts as a tract leading to the dental pulp along which the microorganisms may travel at a variable speed, depending upon a number of factors. Thus, when lateral spread at the dentino-enamel junction occurs with involvement of underlying dentin, a cavity of considerable size may actually form with only slight clinically evident changes in the overlying enamel except to its undermining.

EARLY DENTINAL CHANGES:

The initial penetration of the dentin by cariesmay result in alterations in the dentin described as dentinal sclerosis, or transparent dentine. This dentinal sclerosis is a reaction of vital dentinal tubules and a vital pulp in which there is a calcification of the dentinal tubules that tends to seal them off against farther penetration by microorganisms. The formation of sclerotic dentin is minimal in rapidly advancing caries and is most prominent in slow chronic caries.

The appearance of fatty degeneration of Tomes' dentinal fibers, with the deposition of fat globules in these processes, precedes even the early sclerotic dentinal changes. In the earliest stages of caries when only a few tubules are involved, microorganisms may be found penetrating these tubules before there is any clinical evidence of the carious process. These have been termed as "*pioneer bacteria*".

Proteolytic organisms would appear to predominate in deeper caries of the dentin, than the acidogenic forms which are more prominent in early caries. The observation that the morphologic type of the bacteria in deep carious dentin is different from that of the bacteria in initial caries substantiates the hypothesis, that initiation and progression of dental caries are two distinct processes and must be differentiated. The evidence indicates that the organisms responsible for the initiation of caries are subsequently replaced by others as the environmental conditions occasioned by the advancing carious lesion are altered.

ADVANCED DENTINAL CHANGES:

The decalcification of the walls of the individual tubules leads to their confluence, although the general structure of theorganic matrix is maintained for some time. A thickening and swelling of the sheath of Neumann may sometimes be noted at irregular intervals along the course of involved dentinal tubules, in addition to the increase in diameter of the dentinal tubules due to packing of the tubules by microorganisms. Tiny "*liquefaction foci,"* described by Miller, are formed by focal coalescence and breakdown of a few dentinal tubules.

The destruction of dentin through a process of decalcification followed by proteolysis occurs at numerous focal areas which eventually coalesce to form a necrotic mass of dentin of a leathery consistency. Clefts are rather common in this softened dentin, although they are rare in chronic caries, since the formation of a great deal of softened necrotic dentin is unusual. These clefts extend at right angles to the dentinal tubules and appear to be due to extension of the carious process along the lateral branches of the tubules or along the matrix fibers which run in this direction. These clefts parallel the contour lines of the dentin, which are due to alternating resting periods during the calcification of the dentin. The clefts account for the manner in which carious dentin often can be excavated by peeling away thin layers with hand instruments.

As the carious lesion progresses, various zones of carious dentin may be distinguished which grossly tend to assume the shape of a triangle with the apex towards the pulp and the base towards the enamel. Beginning pulpally at the advancing edge of the lesion adjacent to the normal dentin.

Caries advancement in dentin proceeds through three changes

1) weak organic acids demineralize the dentin.
2) organic material of the dentin, collagen degenerates and dissolves.
3) The loss of structural integrity is followed by invasion of bacteria.

❖ **DIFFERENT ZONES:**

ZONE 1: ***NORMAL DENTIN***

- Deepest area is normal dentin which has tubules with odontoblastic process that are smooth and no crystals in the lumen.
- The inter tubular dentin has normal cross banded collagen and normal dense apatite crystals.
- No bacteria in the tubules.
- Stimulation of dentin (eg: by osmotic gradient, a bur, a dragging instrument or air blow) produces a sharp pain.

ZONE 2:*SUBTRANSPARENT DENTIN*

- Zone of demineralization of the inter tubular dentin and initial formation of very fine crystals in the tubule lumen at the advancing front.
- Odontoblastic process damage is evident.
- No bacteria are found in this zone.
- Stimulation of dentin produces pain.
- Dentin is capable of remineralization

ZONE 3:*TRANSPARENT DENTIN*

- This dentin is softer than normal dentin and shows further loss of mineral from the inter tubular dentin and many large crystals in the lumen of the tubules.
- Stimulation produces pain.
- Intact collagen can serve as a template for the remineralization of the inter tubular dentin and thus this region is capable of self repair , provided the pulp remains vital.

ZONE 4:*TURBID DENTIN*

- Zone of bacterial invasion and is marked by widening and distortion of the dentinal tubules which are filled with bacteria.
- There is very little mineral present and the collagen is irreversibly denatured.
- Dentin in this zone will not undergo self repair .This zone cannot be remineralized and must be removed before restoration.

ZONE 5:*INFECTED DENTIN*
- Outer most decomposed dentin that is teeming with bacteria.
- No recognizable structure to the dentin and collagen ,and minerals are absent.
- Removal of infected dentin is essential to sound, successful restorative procedure as well as prevention of spread of infections.

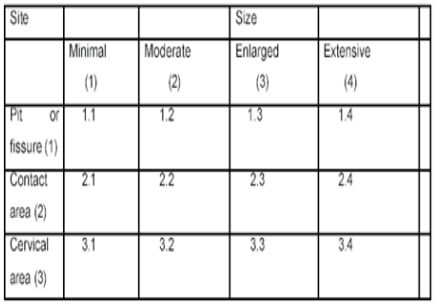
INFECTED DENTIN	AFFECTED DENTIN
1)Softened demineralized dentin invaded with bacteria	Softened demineralized dentin not yet invaded by bacteria
2) Collagen is irreversibly denatured	Collagen cross linking remains
3) Cannot be remineralized	Acts as a template for reminralization
4) Soft necrotic tissue followed by dry leathery dentin. Flakes away with instrument	Softer than normal dentin , discolored but does not flake easily
5)Dyes – 1% Acid red in propylene glycol .Stains only irreversible denatured collagen.	Does not stain.

ARRESTED CARIES:
Characterized by surface: hard, leathery or eburnated
Color: darkly pigmented
Pain: none
The arrested lesion has more homogeneous appearance than the active lesion.
With arrest of the dentinal caries, sclerosis of the affected tubules renders them, impermeable to acids and other irritants of cariogenic bacteria. Pulp will have time to repair by formation of secondary dentine.

ROOT CARIES:
Caries of cementum.
Four lesions which affect the root surface of a tooth
- Abrasion
- Erosion
- Idiopative resorption
- Caries

CLASSIFICATION OF DENTAL CARIES:[11,12]
Dental caries can be classified in several ways. Most of these are based on the clinical, radiographic or histologic appearance of the carious lesion.

Based on the location of the caries	• Pit and fissure caries • Smooth surface caries • Root surface caries
Based on speed of caries progression	• Acute or rampant caries • Chronic caries • Arrested caries
Based on whether it is a new or recurrent carious lesion	• Initial or primary caries • Recurrent or secondary caries
Based on the extent of caries	• Incipient caries • Cavitated caries
Based on the pathway of caries spread within the tooth	• Forward caries • Backward caries
Based on the number of tooth surfaces involved	• Simple caries • Compound caries • Complex caries
Based on restoration and treatment design (G.V. Black's classification)13	• Class I caries • Class II caries • Class III caries • Class IV caries • Class V caries • Class VI caries

Based on whether caries is completely removed or not during treatment	• Residual caries
Based on the age of the patient	• Nursing bottle caries • Adolescent caries • Senile caries
Based on the tooth surface to be restored	• **O** Occlusal surface • **M** Mesial surface • **D** Distal surface • **F** Facial surface • **B** Buccal surface • **L** Lingual surface
Graham Mount classification29	

Site			Size	
	Minimal (1)	Moderate (2)	Enlarged (3)	Extensive (4)
Pit or fissure (1)	1.1	1.2	1.3	1.4
Contact area (2)	2.1	2.2	2.3	2.4
Cervical area (3)	3.1	3.2	3.3	3.4

DIAGNOSIS OF DENTAL CARIES:[12,18,19,20,21]

Diagnosis of caries implies deciding whether a lesion is active, progressing rapidly or slowly, or whether the lesion is already arrested. Without this information, a logical decision about treatment is impossible.

- **Prerequisites for early diagnosis:**

1. Good lighting
2. Clean teeth
3. A three-in-one syringe so that teeth can be viewed both wet and dry.
4. Sharp eyes.
5. Blunt probes.
6. Reproducible bite-wing radiographs.

Methods of caries detection:[30]

In vivo (clinically)	**In vitro: (for research purposes)**
• Visual examination • Tactile examination • Radiographs– conventional, digital and xeroradiography • Fibreoptic transillumination • Optical methods – Fluorescence, light scattering • Electronic resistance measurements • Ultrasonics • Dyes • Visible luminescent spectroscopy • Optical caries monitor • Laser luminescence • Endoscopic methods • Diagnodent	• Chemical analysis • Cross-sectional microhardness • Polarized light microscopy • Traditional transverse microradiography • Microphobe analysis • Iodine absorbitometry • Longitudinal microradiography • Surface microhardness.

PREVENTION OF DENTAL CARIES:[22,23,24,25]

- **Strategies of prevention**

- Substrate limitation.
- Microflora modification
- Plaque inhibition
- Stimulation of salivary flow
- Modifying the tooth substance
- Preventive restorations
- Patient education

OPERATIVE MANAGEMENT OF CARIES:[26,27]

For cavitated carious lesions appropriate restorations must be placed to restore the integrity of the tooth. There are five basic reasons to place restorations when cavitation occurs due to caries:

- To remove infected dentin
- To protect the pulp and avoid pain
- To remove the habitat for cariogenic bacteria.
- To facilitate plaque control
- To restore the esthetics and integrity of the tooth

The choice of restorative material for a cavitated carious lesion depends on:

- Specific area of the tooth to be restored
- Properties of the restorative material
- Needs of the patient
- Economic status of the patient

II. Conclusion

Dental caries remains a commonly encountered clinical problem in routine dental practice. Presently, we have a clearer understanding about the etiology of the disease. Technological advancements have improved our diagnostic skills. The earlier methods of merely replacing diseased tooth with restorations is gradually giving way to the enlightened approach of caries risk assessment, prevention and minimal intervention.
The public who avoid dental treatment due to the fear of the drill will therefore be better served in the future.

References:

[1]. Ungar PS. Mammal teeth: origin, evolution, and diversity. JHU Press; 2010 Oct 1.
[2]. Chour GV, Chour RG. Diet counselling–A primordial level of prevention of dental caries. J Dent Med Sci. 2014;13(1):64-70.
[3]. Hema BS, Goenka S, Verma P. Overview on concepts of dental caries Hema BS, Saloni Goenka2, Poorva Verma3.
[4]. Nikiforuk G. 3 Etiology of Dental Caries—A Review of Early Theories and Current Concepts. InUnderstanding Dental Caries 1985 (Vol. 1, pp. 60-82). Karger Publishers.
[5]. Garg N, Garg A. Textbook of operative dentistry. Boydell & Brewer Ltd; 2010.
[6]. Brodsky RH. Factors in the etiology and arrest of dental caries. Journal of the American Dental Association. 1933 Aug 1;20(8):1440-58.
[7]. Babu NA, Anitha N, Malathi L. Dental Caries as an Ill Effect of Long Term Medication. Indian Journal of Public Health Research & Development. 2019 Nov 1;10(11).
[8]. Ritter AV. Sturdevant's art & science of operative dentistry-e-book. Elsevier Health Sciences; 2017 Dec 20.
[9]. Galhotra V, Sofat A, Dua H, Rohila S. Anticariogenic And Cariostatic Potential Of Components Of Diet: A Review. Indian Journal of Dental Sciences. 2014 Oct 1;6(4).
[10]. Fejerskov O, Kidd E, editors. Dental caries: the disease and its clinical management. John Wiley & Sons; 2009 Mar 16.
[11]. Understanding Dental CariesEtiology &Mechanism ,Basic and Clinical aspect *Gorden Nikiforuk*
[12]. Textbook of Cariology edited by *Anders Thylstrup, Old Fegorskov*
[13]. Sturdevant's Art and Science of Operative Dentistry.4th edition
[14]. The Dental Clinics Of North America, Cariology*James s. Wefel,PhD& Kevin J Donle DDS,MS*
[15]. Cariology , 3rd edition *Ernest Newburn. DMD,PhD*
[16]. A Text book of Oral Pathology , 4th edition .*William .G. Shafer.*
[17]. Dentine caries excavation: a review of current clinical techniques. *A. Banerjee ,T.F Watson ,and E.A.M Kidd*
[18]. Diagnosis and risk prediction of Dental Caries .Per Axelsson DDS PhD
[19]. Clinical Trends In The Diagnosis And The Treatment Of Dental Caries.*Steven Steinberg DDS*
[20]. Dental caries diagnoses *Stookey et al. DCNA, 1999.*
[21]. Caries Diagnosis. *Angmar- Mansson. Et al.*J.Dent. Edu.62, 1998.
[22]. Nutrition, Growth and Metabolism of MicroorganismsDemosthenes *Pappagianis, MD*
[23]. Dental Caries Is Preventable Infectious Disease: *Mayooram Balakrishnan* Aust Dent Journal: 45(4) 2000
[24]. Genetic Modalities in Caries Prevention *PonamBogra Et Al* JIDA Feb 2003.
[25]. Caries Prevention In Children - The Indian Challenge*Beena Rani Goel*
[26]. Modern concepts of Operative Dentistryedited by *PrebenHorsted, Ivan A Mjor*
[27]. The ART Of Restoring Teeth: *Elly Grossman*Http://Www.Mrc.Ac.Za
[28]. Essentials of Preventive and Community Dentistry*Soben Peter.*
[29]. A new classification for dentistry*Mount GJ,* Quint Int 1997,28. pg-301-303
[30]. A colour atlas of operative dentistry.*J R Grundy*

Dr. Sadhvi Gupta, et. al. "Dental Caries*." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 19(8), 2020, pp. 01-08.