"ASSESSMENT OF LIP AND FINGER PRINT PATTERNS IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AND DENTAL CARIES IN LUCKNOW: A CROSS SECTIONAL STUDY"

DISSERTATION

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BABU BANARASI DAS UNIVERSITY,

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In the partial fulfilment of the requirements for the degree

of

MASTER OF DENTAL SURGERY

In

ORAL & MAXILLOFACIAL PATHOLOGY & ORAL MICROBIOLOGY

By

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(Faculty of Babu Banarasi Das University)

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I hereby declare that this dissertation entitled **"ASSESSMENT OF LIP AND FINGER PRINT PATTERNS IN PATIENTS WITH TYPE 2 DIABETES MELLITUS AND DENTAL CARIES IN LUCKNOW: A CROSS SECTIONAL STUDY"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. JIJI GEORGE,** Professor & head, and **Dr. ANKITA SINGH,** Reader as Co-Guide in Department of Oral & Maxillofacial Pathology & Oral Microbiology, Babu Banarasi Das College of Dental Sciences, Babu Banarasi Das University, Lucknow, Uttar Pradesh.

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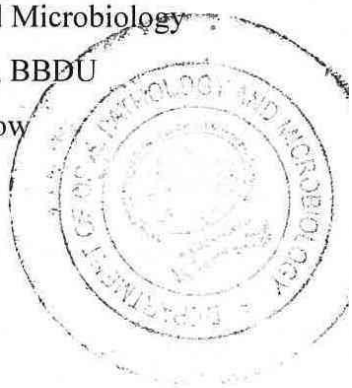

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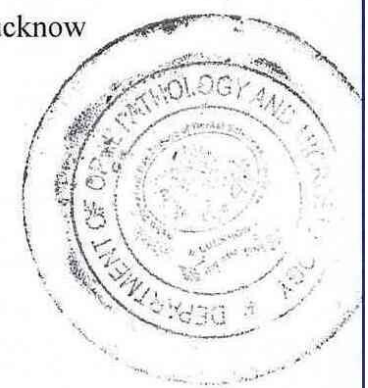

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"DEDICATED IN LOVING MEMORY OF MY MOTHER"

"I grew up watching my mother handle any obstacle life threw at her. I could never be weak because I have learned from the BEST".

"There is no realm vaster than God and there is no truth beyond God"

First and foremost, I bow my head to the **ALMIGHTY** for his divine grace on me and without whose blessings, this endeavor would have been impossible.

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I am infinitely obliged to express my feelings of pride for my most cherished treasure- **my parents** and **father-in-law** for their love and support and **my daughter** for her patience & understanding throughout the 3 years of my academic tenure. I present this thesis as an unassertive tribute to all their love, affection, dreams and blessing with which they have nurtured me.

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Every journey has to come to an end, but every end is a new beginning.

Dr. Dakshayani Vijay Patil

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ABSTRACT

Background and Objectives: Globally, the prevalence of diabetes and dental caries are soaring high in recent times. There is a constant effort in the scientific community to develop a reliable and economic early predictor which can serve the purpose of mass screening of genetically vulnerable populations. Hence, the present study was conducted to evaluate the different types of lip prints and finger prints in diabetes mellitus and dental caries and to see if dermatoglyphics and cheiloscopy can be used as factors for prediction and screening and also to assess correlation between the Diabetes mellitus and Dental caries if any.

Materials and Methods: Study subjects included 100 subjects [50 uncontrolled Type II diabetes mellitus patients and 50 healthy controls] in age group of 30-80 years among the population of Lucknow. Lip prints were obtained using lipstick and cellophane paper, analyzed and classified using Suzuki and Tsuchihashi's classification. Finger prints were obtained using inkpad, analyzed and classified using Henry's system of classification.

Results: We found loop type fingerprints and type IV lip prints associated with diabetic patients. Non-diabetics showed loop type fingerprints and type I lip prints. We found increased dental caries incidence (DMFT scores) in diabetics. Diabetic subjects with caries showed loop fingerprints which reiterated our earlier findings but did not correlate with type IV lip prints. Non-diabetics with caries showed arch fingerprints but did not correlate with type I lip prints. We found that DMFT scores that we used to assess caries did not correlate well with lip prints.

Conclusion: The results from our study strongly suggests that dermatoglyphics can be used as a non-invasive technique to mass screen for diabetes as well as dental caries as both diseases are predominantly associated with loop type fingerprints. Type IV lip prints could be used to screen for diabetes but no correlation of lip prints were seen in patients with caries.

INTRODUCTION

Diabetes Mellitus is a multi-factorial chronic systemic disease which is triggered by number of genetic and environmental factors. It is considered to be a slow and silent killer. India is known as the 'diabetic capital' of the world owing to increased blood glucose levels associated with our food habits and lifestyle. Maintaining good oral hygiene is of utmost importance for overall development and health of an individual. Prevalence of dental caries is also very high in the Indian population.

Lip prints and finger prints are unique and consistently stable features in lifetime of an individual. The word Cheiloscopy is derived from the Greek words, "cheilo" meaning lips and "skopein" meaning to see. Cheiloscopy is defined as a method of identification of a person based on characteristic arrangement of lines appearing on red part of lips. Dermatoglyphics is a Greek word which is derived from "derma" meaning skin and "glyphae" meaning carving. Dermatoglyphics is the study of palmar and plantar dermal ridge carvings on hands and feet.

Diabetes mellitus and dental caries can be detected using chair side methods which are not as cost effective when compared to routine lab diagnostic techniques which are in turn time consuming. Lip prints and finger prints are widely used for identification in forensics and can be recorded cost effectively with good patient compliance.

Hence we conceptualized this study to evaluate the different types of lip prints and finger prints in diabetes mellitus and dental caries and to see if they can be used as factors for prediction and screening and also to assess correlation between the most common diseases [Diabetes mellitus and Dental caries] if any.

AIM & OBJECTIVES

AIM:

To assess the correlation between Type II Diabetes Mellitus and Dental Caries if any using Dermatoglyphics and Cheiloscopy.

OBJECTIVES:

1. To assess the type of lip prints in Type II diabetes mellitus patients in the population of Lucknow.
2. To assess the type of lip prints in Non diabetic patients in the population of Lucknow.
3. To assess the type of finger prints in Type II diabetes mellitus patients in the population of Lucknow.
4. To assess the type of finger prints in Non diabetic patients in the population of Lucknow.
5. To assess type of lip prints in patients with dental caries in population of Lucknow.
6. To assess type of finger prints in patients with dental caries in population of Lucknow.
7. To assess if lip prints and finger prints can be used as early screening tool in Type II diabetes mellitus and dental caries.

REVIEW OF LITERATURE

DERMATOGLYPHICS

INTRODUCTION:-

"Methods of Physicians are like those of a detective, one seeking to explain disease, other a crime"

Dermatoglyphics, (from ancient Greek derma = skin, glyph = carving) is the study of configurations of epidermal ridges on the volar aspect of hands and feet.

HISTORY:-

Ancient Indians believed that the presence of ten whorls meant that a person was destined to be a "Chakravarti" or "an Emperor". Curiosity in the field of dermatoglyphics started in late centuries when Chinese used it as a basis for fortune telling[1]. In 1684, Nehemiah Grew, a physician pioneered in publishing about the epidermal ridges and their characteristic patterns when prints of fingertips were taken. These "innumerable little ridges of equal bigness and distance, and everywhere running parallel one with another," contain the pores of the sweat glands. Grew described them as "ellipticks" and "triangles." Grew's paper was then followed by the publication in Amsterdam of a brief account in Bidloo's (1685) Anatomia Humani Corporis. Purkinje (1823), described nine finger pattern types. The first systematic study was carried out by Galton (1892) on dermal patterns in families and racial groups[2]. It was further elaborated and improved by Sir Edward Richard Henry of Scotland Yard for identifying criminals[3]. Walker studied "The use of Dermal Configurations in the Diagnosis of Mongolism" (1957), attempted to quantify dermal configurations for use as a powerful diagnostic tool[4].

CLASSIFICATION OF DERMATOGLYPHICS ON FINGERTIPS:-

Cummins and Midlo classified various pattern types on fingertips as:

a. Arch

b. Loop

c. Whorl

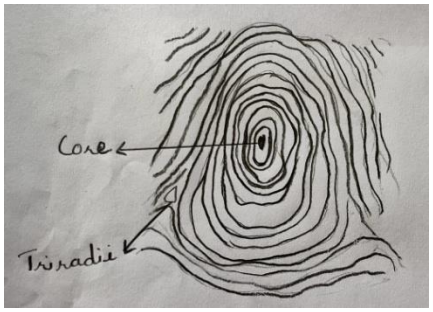
d. Composites

The palmar surface is divided into dermatoglyphic areas, which are hypothenar, thenar, and the four interdigital areas numbered I to IV. There are four digital triradii and one or two axial triradii (t)[5].

Uchida et al., classified fingerprints into arch, loop, and whorl. Ridge count was used as a dermatoglyphic indicator. Digital triradii a, b, c, d, and axial triradius "t" was described and position of "t" was described by measuring "atd" angle. They also described dermatoglyphic patterns in chromosomal abnormalities[6].

In 1971, Bali and Chaube described the formation of palmar creases and classified them as single radial base crease (SRBC), double radial base crease (DRBC), or triple radial base crease (TRBC)[7].

Triradius:- A triradius is located at the meeting point of three opposing ridge system.



[FIGURE 1: CORE AND TRI RADII]

Patterns:-

There are three main types of patterns:-

Arch→ The ridges pass from one margin of the digit to the other with a gentle, distally bowed sweep. There is no triradius.

Loop→ It possesses only one triradius. The ridges curve around only one extremity of the pattern, forming the head of the loop. From the opposite extremity of the pattern, ridges flow to the margin of the digit , this extremity of the pattern may thus be described as 'open'. According to this, loops may further be of two types:

• Ulnar loop – When the loop opens to the ulnar margin

• Radial loop – When the loop opens to the radial margin

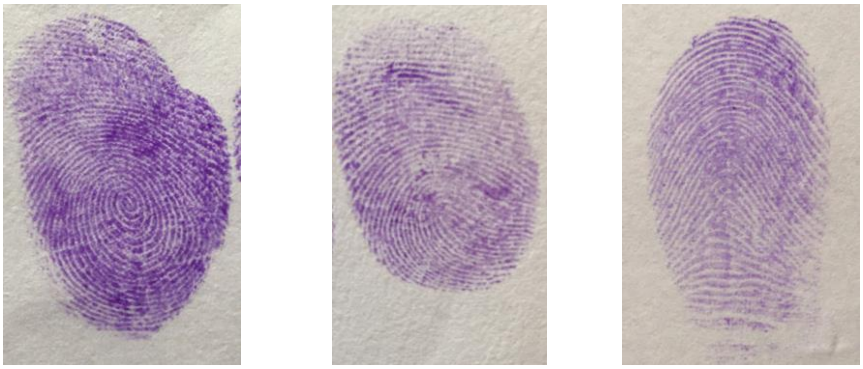
Whorl→ It is distinguished by concentric design. True whorls typically possess two triradii. There are also composite patterns in which two or more designs are combined in one pattern area. They have two or more triradii. They are included under whorls.

Open fields (O):- These are configurations in which the ridges are essentially straight, and therefore, form no patterns.

Vestiges (V):- They lack the sharp recurvature of ridges which distinguish true patterns. It is merely a local disarrangement of ridges.

Pattern intensity:- This is the number of triradii on all the ten fingers of an individual. The value ranges from 0 to 20.

Ridge count:- These are made from triradii point to point of the core[8].



WHORL **LOOP** **ARCH**

[FIGURE 2: DERMATOGLYPHIC PATTERNS]

EMBRYOLOGY:-

Volar pads develop on hands/feet at 7weeks I.U At 10 weeks I.U, the pads are reabsorbed into the palms and feet. Simultaneously Basal epidermis begins starts to fold owing to pressure from the growing skin. These folds are precursors of fingerprints, and their pattern depends on differential absorption of volar pads. If the volar pad remains, it will reveal a whorl. If the volar pad is partially absorbed, it forms a loop. If entirely absorbed, the pattern will be arch[9].

INHERITANCE OF FINGERPRINT PATTERNS:- Documented literature suggests that finger patterns are genetically inherited[6,10,11,12]. The homolateral differences in monozygotic twin pairs are a widely known fact. A progressive reduction in the degree of similarity is demonstrable but the totality of pattern characteristics is not transmitted[13]. The total ridge count is an inherited measured character. The diversity of ridge-count from finger-to-finger is also genetically inherited[6]. Other dependent factors are the presence of extra chromosomes and minor structural aberrations[10]. Environmental factors (or modifier genes) also play an important role[12] making fingerprints a multifactorial trait[13]. In 1967, Gibbs worked on general heritable aspects of dermatoglyphics and highlighted that individuals may be born without fingerprints, where tips of fingers are absolutely smooth[14]. In pilot

study conducted on neonates of low birth weight, higher frequency of Simian crease was found, both typical and transitional types[15].

USES:-

Dermotoglyphics is widely used as a diagnostic tool in a variety of inheritable diseases[16]. Currently, medical dermatoglyphics is associated with various conditions such as diabetes mellitus, hypertension, psychosis[17,18], alcohol embryopathy[19], epilepsy[20], congenital heart diseases[21], psoriasis[22], malignancy[23]. Borgaonkar et al. mentioned that chromosomal imbalance of any kind has an effect on the dermatoglyphic pattern, which is found in Down Syndrome[24]. David described Ridges-of-the End Syndrome in two families and the Nelson Syndrome, both of which are dermatoglyphic syndromes, probably inherited as autosomal dominant traits[25].

Forensic science is an important branch in the modern times, which heavily relies on the use of fingerprints, as they remain unchanged even after death. Three types of fingerprints are technically studied in addition to its morphological types such as, plastic impressions which are made in soft material like butter, soap, etc., visible prints made when fingers have been covered in blood, dirt, oil, paint, etc. and latent prints which are not visible to the human eye, hidden, unseen until treated. Automated fingerprint identification system scan is used in various fields.

Lesser known benefits of dermatoglyphics is multi-intelligence test (DMIT) for children/students and in the corporate sector. The test helps in identifying his/her inborn talents and weaknesses, help in the subject and educational stream selection. It helps to discover learning Style and teach children accordingly.

DMIT is done in three easy steps by DMIT software[26] .

- Step-1: Fingerprint Scan
- Step-2: Analyze fingerprints. Less than 35° ATD angle predicts the potential of a person as a born Athletes, Sharp Observer, Agile task performer and an angle of more than 46° is considered as slow learner
- Step-3: Counseling

CHEILOSCOPY

INTRODUCTION:-

The wrinkles and the grooves on the labial mucosa(called sulci labiorum) form a characteristic pattern called lip prints[27]. Cheiloscopy (from Greek words cheilos=lips,skopein=see) is the name given to the lip print studies. The importance of cheiloscopy is linked to the fact that lip prints are unique to one person, except in monozygotic twins. Like fingerprints and palatal rugae, lip grooves are permanent and unchangeable. It is possible to identify lip patterns as early as the sixth week of intrauterine life[28]. Like other biometric data, such as ear prints, elbow prints, rugae patterns, foot prints, and bite marks, every human lip print is a unique combination of grooves, wrinkles, lines, and creases with extremely fine details[29].

HISTORY:-

The biological phenomenon of systems of furrows on the red part of human lips was first noted by anthropologist R. Fischer in 1902[30,31]. In 1961, the first research of lip prints was carried out on subjects in Hungary. The examination started at the scene of crime, when lip traces were found on a glass door[32]. In Poland (1966) the interest in lip prints started, when accidentally a lip print was revealed on the window glass at the scene of burglary[33]. In August 1966, Dr. Martin Santos from Brazil presented his own classification of lip furrows and lines and described how these characteristic features can be used for identification[32].

In the period 1968–1971, two Japanese scientists, Y. Tsuchihashi and T. Suzuki, examined 1364 persons at the Department of Forensic Odontology at Tokyo University. With that research, it was established that the arrangement of lines on the red part of human lips is individual and unique to each human being. In further research, the Japanese scientists examined the principles of the heredity of furrows on the red part of lips[34,35,36].

In 1974, Tsuchihashi carried out another study with greater number of participants as well as family groups. By comparing the lip prints of the twins with their parents, he found that they closely resembled one parent which adds strength to the theory of heredity of lip prints. He also found that following trauma to a lip, it resumed the groove pattern after healing[32]. In 1981, Cottone reported in his book outline Forensic Dentistry that cheiloscopy is one of the unique techniques used for person identification[35].

In 1982, a project was launched in the Forensic Institute of Warsaw University Criminal Law Department in cooperation with the former Forensic Institute of Milita in Warsaw with the aim of developing one cohesive cheiloscopy system practicable in forensic cheiloscopy. In 1985, the methods of finding and recovery of lip traces, recovering comparative material, and the techniques employed to carry out expertise have been introduced into the casework of fingerprint department of the Central Forensic Laboratory of Police in Warsaw, Poland[32]. In 1990, Kasprzak conducted research for 5 years in 1500 persons to indicate the practical use of lip prints[37,38].

In 1999, the Federal Bureau of Investigation (FBI) and the Illinois state police conspired that lip prints are unique like fingerprints and are useful for identification[38]. Alvarez et al (2000–2002) and Vahanwahal et al (2000) gave the explanation that vermilion borders of the lips have minor salivary glands and sebaceous gland secretions and moisturizing property, which makes the latent lip prints available at most of the crime scenes. In a study by Castello et al (2005) on luminous lip prints, he used luminescence as a special property for the search of invisible evidence in the scene of crime[39].

Study on postmortem changes of lip prints was carried out to find out the changes in anthropometric measurements of the lip region before and after fixation[40,41]. These studies were in agreement with the Japanese research and thus helped in concluding that the cheiloscopic studies can be implemented as an auxiliary method of identification. Prabhu et al (2012) stated that lip prints can be properly recorded without the use of any recording medium with the help of suitable nonporous surface[40].

Kundu S et al, mentioned that, lip print patterns are distinct for an individual. A statistically significant prevalence of curve and wavy form was seen in males and straight pattern in females. Khanapure et al (2014) summarized the view that the distribution of lip prints is unique for males and females and the association between geographic location and lip print was not statistically significant. Almuhaizia et al (2014) presented a published manuscript on gender determination using cheiloscopy in the pediatric population, in which they commented critically that no two lip print patterns matched each other, thus establishing the uniqueness of lip prints[39].

ANATOMY OF LIPS:-

The upper lip lies between the nose and oral cavity, whereas the lateral lips are separated from the cheeks by nasolabial grooves, extending from the nose and passing approximately 1 cm lateral to the angles of the mouth. These grooves can be easily observed while smiling. The philtrum present on the upper lip is an infranasal depression extending from the external nasal septum, separating the nostrils to the vermilion border which is the sharp demarcation between the colored edge of the lip and the surrounding skin. The lower lip lies between the mouth and the labiomental groove, which separates the lower lip from the chin. The upper and lower lips are continuous at the angles of the mouth or oral commissures. Sensory function for sensuality and sexuality is performed by a complex system of muscles and supporting structures which are denoted as classification of aging which states that:

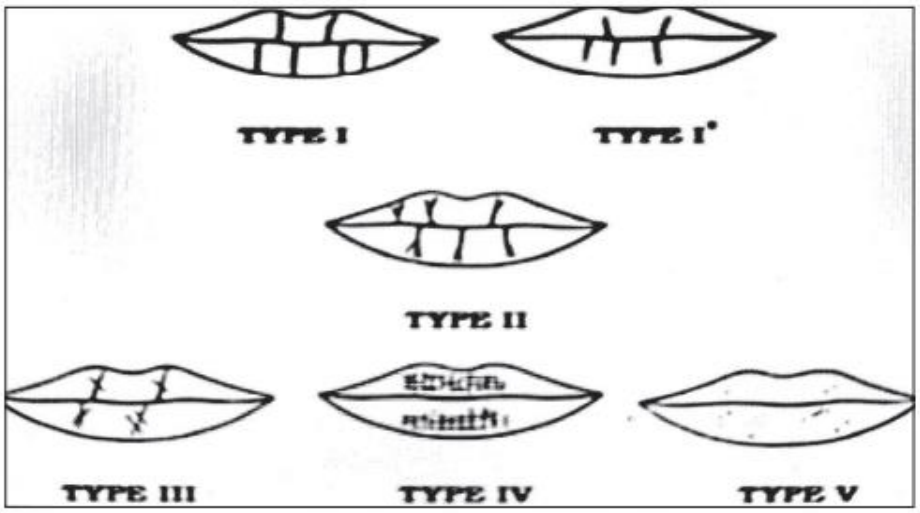
- Class 1 (nice shape and definition): These individuals have a nice vermillion and vermillion border but wish for enhancement.
- Class 2 (atrophic lips): These individuals have atrophic lips, which may be due to aging or heredity and are seeking augmentation to make them look more youthful.
- Class 3 (lip atrophy and vermillion disappearance): The perioral lines are observed at the edge of the white roll of the lips where the orbicularis oris is attached to the dermis with no interposed fatty layer. These lines typically start at the 30s and increase in length and depth with aging. They may be more apparent with increasing sun exposure, smoking, lifestyle changes, and genetic predisposition[42].

CLASSIFICATION[43]:-

1. Martin Santos Classification (1966):
   - **I.** Simple wrinkles
     - **a)** Straight lines
     - **b)** Angled lines
     - **c)** Sine-shaped curve
   - **II.** Compound wrinkles
     - **a)** Bifurcated
     - **b)** Trifurcated
     - **c)** Anomalous

2. Suzuki and Tsuchihashi Classification (1970):

- Type I: Clear-cut grooves running vertically across the lip
- Type I': Straight grooves which disappear half way instead of covering the entire breadth of the lip
- Type II: Fork grooves in their course
- Type III: Intersecting grooves
- Type IV: Reticulate grooves
- Type V: Undermined



[FIGURE 3: SUZUKI & TSUCHIHASHI CLASSIFICATION]

3. Raynaud's Classification:

a) Complete vertical
b) Incomplete vertical
c) Complete bifurcated
d) Incomplete bifurcated
e) Complete branched
f) Incomplete branched
g) Reticular pattern
h) X or coma form
i) Horizontal
j) Other forms (ellipse, triangle)

4. Afchar–Bayat Classification (1979):

- A1: Vertical and straight grooves, covering the whole lip
- A2: Vertical and straight grooves, but not covering the whole lip

- B1: Straight-branched grooves
- B2: Angulated-branched grooves
- C: Converging grooves
- D: Reticular pattern grooves
- E: Other grooves

5. The sex of the individual was determined as given by Vahanwala et al.

a) Type I and I' pattern dominant: Female

b) Type I and II pattern dominant: Female

c) Type III pattern dominant: Male

d) Type IV pattern: Male

e) Type V varied patterns: Male

6. Recently for the basis of the classification, only 10 mm portion of the middle part of the lower lip is used.

- Linear "L" – if the lines prevail
- Bifurcation "R" – if the bifurcation is dominant
- Reticular "S" – if the lines cross
- Undermined "N" – when no superiority can be established

7. The next step is, 23 types of individual features are described to establish individual features of patterns of the lines

S.No	Pattern of Lip line	Individual features	S.No	Pattern of Lip line	Individual features
01	An eye		15	Simple opening	
02	Hook		16	Closing top bifurcation	
03	Bridge		17	Pentagonal arrangement	
04	Line		18	Branch like top bifurcation	
05	Dot		19	Star like bifurcation	
06	Rectangle		20	Fence	
07	Triangle		21	Branch like bottom bifurcation	
08	Group of dots		22	Double fence	
09	Simple top bifurcation		23	Hexagonal arrangement	
10	Simple bottom bifurcation				
11	Double eye				
12	Crossing lines				
13	Crossing bottom bifurcation				
14	Delta like opening				

[FIGURE 4: INDIVIDUAL FEATURES OF PATTERNS OF LIP LINES]

METHODS FOR RECORDING OF LIP PRINTS:-

Lip Prints can be recorded in a number of ways:

1. Photographing the suspect's lips on a non-porous flat surface such as a mirror they can be photographed, enlarged and overlay tracings made of the grooves[44,45].
2. Applying lipstick, lip rouge, or other suitable transfer mediums to the lips and then having the individual press his or her lips to a piece of paper or cellophane tape or similar surface[30,44,46,47].
3. Using a finger printer, preferably a roller finger printer [46,48].
4. By having the subject impress his or her lips (without lipstick or other recording medium) against a suitable surface and then processing these prints with either conventional fingerprint developing powder or with a magna brush and magnetic powder[44].

- PHOTOGRAPHY:-

  Half-size photographs of the lips are taken with a Medical Camera and enlarged to double its size, thus obtaining life-size photographs. It should be ensured that the subjects are calm so as to get a clear photograph. The advantage of this method is that it removes the inaccuracy associated with the strength and direction of the pressure applied in taking lip prints by other methods.

- USING LIPSTICK:-

  After application of the lipstick, the subject is asked to rub his/her lips together to spread the lipstick uniformly. Prints are taken on a bond paper, supported by a flat cardboard piece. The centre portion of the lips are dabbed first by the paper and then pressing it uniformly to the right and left corners of the lips. Care is taken to avoid sliding of the lips to prevent smudging of the print.

- A FINGERPRINT ROLLER:-

  Used to apply the special paper (used in fingerprinting) over the lips. The print is then traced on to cellophane paper and examined under a magnifying glass.

- DENTAL IMPRESSION MATERIALS:-

  Can be used to make casts of the lips, in order to record and study the grooves on them. These materials are employed for a variety of uses such as making

study models for orthodontic treatment, casts for construction of maxillofacial prosthesis and dentures etc[49].

- RECORDING LATENT LIP PRINTS WITH FINGERPRINT POWDER:-

  The lips of the subject are first cleaned thoroughly using wet cotton with cleanser and then with sterile cotton. The lips are gently pressed together against a glass slab for 3-4 seconds. The print formed on the glass slab is developed by sprinkling the black fingerprint powder composed of charcoal, lampblack, and graphite. Gentle dusting using a special "Marabou" feather brush loaded with fingerprint powder is carried out. The excess powder is removed to visualize the hidden print and the print is then transferred to a white bond sheet with the help of a 2-inch-wide lifting tape[50].

- DIGITAL METHODS:-

  Similar to the use of scanners to record fingerprints, computers can be used to record lip prints as well. Digital images of lip prints can be stored in a computer that may be compared with prints obtained from a scene of crime. The prints can then be analyzed using software for cheiloscopy[49].

USES:-

1. Aid in forensic sex determination.
2. A tool in crime investigation.
3. An aid for personal identification.
4. Cheiloscopy is analogous to fingerprint analysis. Lip prints added evidence to a crime scene, and this is valuable, especially in cases of lacking other evidence, like fingerprints[51].
5. Lip Print as a Victim in Court. FBI has used lip prints as a means of positive identification only once[33].

DIABETES MELLITUS

DEFINITION:-

Diabetes mellitus is a group of metabolic diseases characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action or both. The abnormalities in carbohydrate, fat and protein metabolism that are found in Diabetes are due to deficient action of insulin on target tissues[52].

CLASSIFICATIONS:-

- According to Expert committee on diagnosis and classification of Diabetes[53]

Etiologic classification of diabetes mellitus

I. Type 1 diabetes (β-cell destruction, usually leading to absolute insulin deficiency)
   A. Immune mediated
   B. Idiopathic

II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance)

III. III. Other specific types
   A. Genetic defects of β-cell function
      1. Chromosome 12, HNF-1α (MODY3)
      2. Chromosome 7, glucokinase (MODY2)
      3. Chromosome 20, HNF-4α (MODY1)
      4. Chromosome 13, insulin promoter factor-1 (IPF-1; MODY4)
      5. Chromosome 17, HNF-1β (MODY5)
      6. Chromosome 2, *NeuroD1* (MODY6)
      7. Mitochondrial DNA
      8. Others
   B. Genetic defects in insulin action
      1. Type A insulin resistance
      2. Leprechaunism
      3. Rabson-Mendenhall syndrome
      4. Lipoatrophic diabetes
      5. Others

C. Diseases of the exocrine pancreas
    1. Pancreatitis
    2. Trauma/pancreatectomy
    3. Neoplasia
    4. Cystic fibrosis
    5. Hemochromatosis
    6. Fibrocalculous pancreatopathy
    7. Others
D. Endocrinopathies
    1. Acromegaly
    2. Cushing's syndrome
    3. Glucagonoma
    4. Pheochromocytoma
    5. Hyperthyroidism
    6. Somatostatinoma
    7. Aldosteronoma
    8. Others
E. Drug- or chemical-induced
    1. Vacor
    2. Pentamidine
    3. Nicotinic acid
    4. Glucocorticoids
    5. Thyroid hormone
    6. Diazoxide
    7. β-adrenergic agonists
    8. Thiazides
    9. Dilantin
    10. α-Interferon
    11. Others
F. Infections
    1. Congenital rubella
    2. Cytomegalovirus
    3. Others

G. Uncommon forms of immune-mediated diabetes
    1. "Stiff-man" syndrome
    2. Anti–insulin receptor antibodies
    3. Others
H. Other genetic syndromes sometimes associated with diabetes
    1. Down's syndrome
    2. Klinefelter's syndrome
    3. Turner's syndrome
    4. Wolfram's syndrome
    5. Friedreich's ataxia
    6. Huntington's chorea
    7. Laurence-Moon-Biedl syndrome
    8. Myotonic dystrophy
    9. Porphyria
    10. Prader-Willi syndrome
    11. Others

IV. Gestational diabetes mellitus (GDM)

- Diabetes mellitus is classified into:

1. Type 1, or insulin dependent diabetes mellitus (IDDM)
2. Type 2, or non-insulin dependent diabetes mellitus (NIDDM)
3. Gestational diabetes mellitus (GDM)
4. Other specific types of diabetes mellitus[54]

HISTORY:-

Ahmed AM suggested that clinical features similar to Diabetes mellitus were described 3000 years ago by the ancient Egyptians. Credit for the initial observation that Diabetes is not a single disorder rests with two Indian physicians- Chakarta and Susruta (600 B.C)- who differentiated two forms of the disease. The term "Diabetes" was first coined by Araetus of Cappadocia (81-133 A.D). Later, the word mellitus (honey sweet) was coined by Thomas Willis in 1675 after rediscovering the sweetness of urine and blood of patients (first noticed by the ancient Indians). It was only in 1776 that Dobson J first confirmed the presence of excess sugar in urine and blood as cause of their sweetness. In

modern time, the history of Diabetes coincided with the emergence of experimental medicine. An important milestone in the history of Diabetes is the establishment of the role of the liver in glycogenesis and the concept that Diabetes is due to excess glucose production was explained by Claude Bernard in 1857[55].

Harris MI in their review stated Diabetes mellitus to be clinically and genetically heterogenous group of disorders characterized by abnormally high levels of glucose in the blood. The hyperglycemia is due to deficiency of insulin secretion or to resistance of the body's cells to the action of insulin, or to a combination of these. Often there are also disturbances of carbohydrate, fat and protein.

During 18th and 19th century, a less clinically symptomatic variety of disorder, identified by heavy glycosuria, often detected in later life and commonly associated with overweight rather than wasting was noted which is recognized as Type II Diabetes mellitus.

In the mid 1930s Himsworth HP proposed that there were atleast two clinical types of Diabetes mellitus, insulin sensitive and insulin insensitive, the former being due to insulin deficiency. Confirmation of his clinical observation came with Bornstein J and Lawrence RD development of bioassay for insulin and when radioimmunoassay for insulin became available a decade later their observations were confirmed. The widespread acceptance of the terms juvenile onset and maturity onset DM at this time affirmed the concept that there were atleast two major forms of this disease.

The 20th century, when screening programs for DM commenced, it became apparent that there were many people who could be classified as having DM but who were in general 'asymptomatic'. It has became apparently subsequently that the term Diabetes mellitus covers a wide spectrum of diseases, from those with acute and sometimes explosive onset to asymptomatic people whose disease is discovered by screening. During the last two decades of the 20th century, research has led to the recognition that DM is a syndrome and comprises of a heterogeneous collection of disorders and that the different types of Diabetes mellitus have different etiologies, although their pathologic effects after onset of disease may be similar[56].

DIABETES MELLITUS TYPE II:-

Shareef BT, Ang KT, Naik VR stated majority of Diabetes is of Type II or Non-Insulin Dependent Diabetes Mellitus(NIDDM). Type II Diabetes occurs usually in patients over 40yrs of age and is strongly familial, though the genetic markers are yet to be identified. It is usually associated with obesity[57].

Davidson SS, Boon NA stated that type II Diabetes commonly occurs in subjects who are obese and insulin resistant but these two factors alone are insufficient to cause Diabetes unless accompanied by impaired beta cell function[58].

Das SK said that type II Diabetes mellitus encompasses a diverse set of diseases marked by elevated levels of plasma glucose[59].

Braunwald E, Fauci S, Kasper DL et al defined type II Diabetes as a heterogeneous group of disorders usually characterized by variable degrees of insulin resistance, impaired insulin secretion and increased glucose production[60].

Expert committee on Diagnosis and classification of Diabetes mellitus concluded that it is term used for individuals who have insulin resistance and usually have relative (rather than absolute) insulin deficiency[61].

CLINICAL FEATURES:-

Five clinical features independently associated with Diabetes mellitus type II are thirst, weight loss, skin infections, fasting glucose less than 5.6mmol/L38 and random glucose level less than 5.6mmol/L15[62].

Type II Diabetes has become increasingly common among children aged 6-11years and adolescents aged 12-19 years especially in obese and overweight children[63].

In newly diagnosed type II Diabetic patients abnormal thirst, frequent urination, weight loss, genital itching, stomatitis, visual disturbances, fatigue, confusion and balanitis were common, of short pre-diagnosed duration and associated with glycaemic level[64].

Diabetic retinopathy is the most specific of diabetic complications. Others include neuropathy, nephropathy, macrovascular diseases and increase tendency for infections[60]. By compiling clinical presentation of type II diabetes mellitus in children and adolescents, obesity is commonly seen. Ketoacidosis is seen in less than 33% of cases and associated disorders that can be seen are acanthosis nigricans, polycystic ovarian syndrome and metabolic syndrome[65].

This disease affects overweight or obese individuals. Most are over 40 years of age but type II is now increasingly seen among children. It has gradual and insidious onset. The diagnosis is made incidently in almost one third of cases. Common presentations are with genital candidiasis (particularly in women) or urinary tract or skin infections. Ketoacidosis is rare but may occur in the setting of concurrent acute stress. Relative insulin deficiency, combined with stress induced increases in counter regulatory hormones, can raise glucose blood level but is not profound enough to allow lipolysis and ketogenesis to precede unrestrained[66].

PATHOGENESIS:-

Shah P compiled pathogenesis of NIDDM and discussed about insulin deficiency and insulin resistance.

Insulin secretory dynamics: In the analysis of insulin secretory dynamics in NIDDM, it is important to distinguish between impaired beta cell sercretory function and decreased absolute circulating insulin levels. In NIDDM fasting (basal) insulin levels are normal or increased. A characteristic pathophysiologic feature of NIDDM is the loss of the first or early phase insulin response to intravenous glucose. This loss of first phase insulin response to intravenous glucose is restored by insulin therapy, salicylates and alpha adrenergic blockers. Second phase insulin response is normal or low. Glucose augments beta cell response to non glucose stimuli and based on this the changes in the acute insulin response to arginine or isoproterenol can be expressed as a function of increasing plasma glucose, thus yielding a glucose potential slope. In NIDDM this glucose potentiation of beta cell functions is reduced.

Another very early lesion that has recently been described in NIDDM is the loss of the normal pulsatile insulin secretory response. Normal individuals in the fasting stage exhibit regular pulses of insulin secretion at afrequency of about 12-15 minutes. But first degree relatives of NIDDM subjects with minimum glucose intolerance exhibit no regular oscillatory activity in insulin secretion. Greater the fasting plasma glucose, greater is magnitude of beta cell dysfunction in NIDDM.

Islet pathology: Islet morphological changes in NIDDM are non specific and non diagnostic. The most important pathologic lesion is insular hyalinization (islet amyloidosis). Insulin fibrosis manifests as intra and inter acinar fibrosis, arterio-sclerosis and fatty atrophy of the pancreas. Islet hypertrophy and insular regeneration

may be observed in the early stages. Margination of granules and degranulation of beta cells are physiologic changes associated with active insulin secretion.

Gross pathological changes in pancreas in NIDDM include reduction in weight, up to 50% of normal & accentuation of lobular markings.

Insulin resistance: It is a metabolic state in which normal concentration of insulin produces a less than normal biologic response. It can involve any of the multiple metabolic effects of insulin.

Causes of insulin resistance:

- Abnormal beta cell product
  - ✓ Abnormal insulin molecule
  - ✓ Incomplete conversion of proinsulin molecule
- Circulating insulin antagonists
  - ✓ Elevated levels of counter regulatory hormones. eg→ growth hormone, cortisol, glucagon
  - ✓ Antiinsulin antibodies
  - ✓ Antiinsulin receptor antibodies
- Target tissue defects
  - ✓ Insulin receptor defects
  - ✓ Post receptor defect[67]

Insulin secretion: Although basal insulin levels in type II diabetic may be normal, several different varieties of abnormalities have been identified in insulin secretion after stimulation of the beta cells. Many patients who do not respond to glucose intravenously will secrete insulin during oral glucose tolerance testing.

With mild to moderate degrees of glucose intolerance, insulin secretion is elevated above normal, peaking when plasma glucose value at 2 hours after glucose load, reaches 200mg/dl. With more severe degrees of glucose intolerance, the secretion of insulin in response to the oral glucose load is reduced, generating a curve for beta cell function reminiscent of the “starling curve” for cardiac function.

Insulin resistance: The finding of normal or elevated plasma levels of insulin in patients with type II diabetes suggests the presence of some form of insulin resistance or reduced insulin sensitivity. This is confirmed by direct measurements of the effects of exogenous insulin in these patients.

The resistance to insulin present in patient with NIDDM can occur as a result of defects at several levels in the action of insulin. The first step in the action of insulin is the binding of insulin to its receptor on the plasma membrane of the cell. This is high molecular weight membrane glycoprotein (mol wt 350,000) which binds insulin and transmits some form of signal to initiate the changes in cellular metabolism associated with effects of insulin. It is suggested that higher the basal insulin concentration, lower the receptor concentration[68].

ORAL MANIFESTATIONS:-

A number of oral conditions have been associated with Diabetes mellitus, particularly in patients with poor disease control.

Krasteva A, Panov V et al compiled oral manifestations in patients with Diabetes mellitus as burning mouth syndrome, xerostomia, decreased salivary secretion, multiple carious lesions and caries in unusual places (root caries), enlarged gingival tissues, bleeding easily upon manipulation, periodontal disease, multiple periodontal abscess, oral candidiasis, ulcers and irritational fibromas, lichen planus and lichenoid reactions, erythema migrans, diabetic sialadenosis, altered oral micro flora and faster alveolar bone resorption. They also suggested that there is prevalence of other fungal infections which manifest as rhomboid glossitis, denture stomatitis and angular chelitis[69].

Mahima VG, Raina A, Patil K suggested that knowledge of wide spectrum of the oral markers of Diabetes mellitus is imperative for the oral health care providers as they frequently encounter individuals with undetected, untreated or poorly controlled disease who present more often with oral manifestations that may indicate the underlined undiagnosed disease. They said that several studies showed an increased incidence of dental caries in Diabetic due to increased glucose level in saliva, altered plaque micro flora and reduced salivary flow as well as poor control of Diabetes. Current evidences however suggests decreased caries incidence in the well controlled diabetic due to dietary restrictions, observance of meticulous oral hygiene and regular dental follow up.

According to them diabetes can lead to gingivitis, periodontitis, salivary gland abnormalities such as xerostomia, sialorrhoea, oral mucosal diseases like oral lichen

planus, oral fungal infections and recurrent aphthous stomatitis. Mucormycosis, a potential fatal infection occurs in individuals in poorly controlled diabetes. Other manifestations include taste disorders like hypogeusia, burning mouth syndrome and stomatopyrosis, oral infection and delayed wound healing[70].

Girtan M, Zurac S, Staniceanu F, Bastian A et al suggested that oral complications of diabetes have huge impact on comfort of patient due to both disease related pain and teeth decay. Oral manifestations include candidiasis, lichen planus, recurrent aphthous stomatitis, gingivitis, salivary disorders, xerostomia, burning sensation, taste changes, glossodynia and neurosensory disorders. There is also higher incidence of oral atrophy, oral leukoplakia, abnormal exfoliative cytology and squamous cell carcinoma. Diabetic patients frequently present gingivorrhagia and chronic marginal periodontitis with alteration of desmodont, cementosis, mobilization and eventually teeth expulsion. Subsequent hyperplastic lesions of the oral mucosa with further evolution towards precancerous/ malignant lesions may occur[71].

Kidambi S, Patel SB said that periodontal disease seems to be associated with atherosclerotic cardiovascular disease. Poorer the control of DM, greater is the risk of developing periodontal disease. Periodontal disease has been proposed as the sixth complication of DM, the other five complications are retinopathy, neuropathy, nephropathy, cardiovascular disease and peripheral vascular disease[72].

Lamster IB, Lalla E et al concluded that although there is no specific association between dental caries and xerostomia with diabetes yet a number of oral mucosal diseases like oral lichen planus and recurrent aphthous stomatitis have been reported in diabetic patients. Periodontal disease is a recognized and well documented complication of diabetes. Other features include burning mouth syndrome and dysphagia[73].

Maeley BL, Ocampo GL suggested that degree of glycaemic control is an important variable in the relationship between diabetes and periodontal disease, with a higher prevalence and severity of gingival inflammation and periodontal destruction being seen in those with poor control. Diabetes increases the risk of alveolar bone loss and attachment loss. Periodontal diseases are inflammatory in nature and in patients with

severe periodontitis, the death rate from ischaemic heart disease and diabetic nephropathy are higher[74].

Herring ME, Shah SK suggested that diabetes leads to a number of conditions that can be revealed on examination of mouth. These might include ulcers, atrophic glossitis, candida infections, periodontitis, severe gingivitis and leukoplakic lesions[75]. Chuang SF, Sung JM, Kuo SC, Huang JJ, Lee SY examined the difference in oral manifestations and dental conditions between diabetic and non diabetic hemodialysis patients. They concluded that diabetic patients had more severe subjective symptoms including dry mouth, taste change and mucosal pain. Patients with poor glycaemic control presented with a higher incidence of tongue pain and tongue coating. Diabetic patients tend to be denture wearers and at higher risk of fungal infections[76].

DENTAL CARIES

INTRODUCTION:-

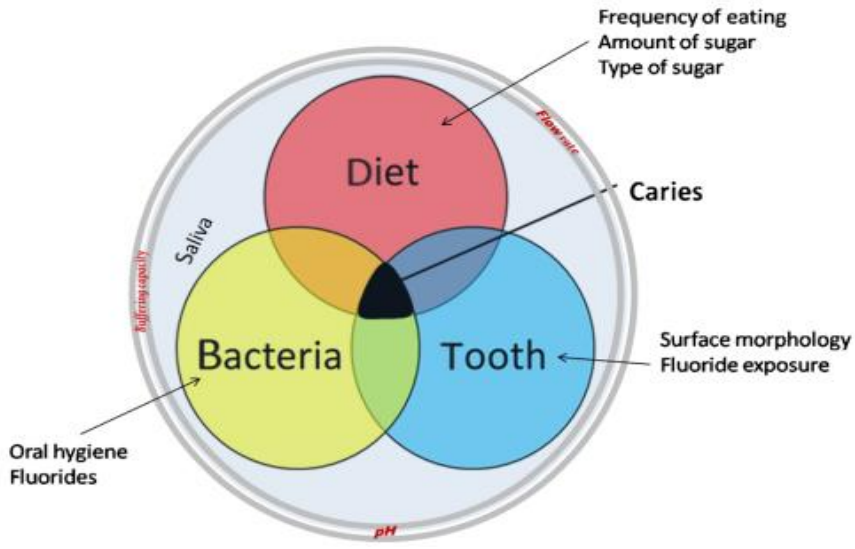
Caries in Latin means, 'rotten'. For a nonprofessional, caries meant a hole in the tooth and for the dental professionals, it meant destruction of the tooth structure in the form of cavitation[77]. Dental caries is one of the most common preventable childhood diseases; people are susceptible to the disease throughout their lifetime[78,79]. It is the primary cause of oral pain and tooth loss[80]. It can be arrested and potentially reversed in its early stages, but is often not self-limiting and without proper care, caries can progress until the tooth is destroyed[81].

DEFINITION:-

Dental caries is the localised destruction of susceptible dental hard tissues by acidic by-products from bacterial fermentation of dietary carbohydrates[82].

The terms dental caries or caries can be used to identify both the caries process and the carious lesion (cavitated or non-cavitated) that is formed as a result of that process[83,84]. The cavity, or decayed surface, is the sequela of the disease process and is a sign of fairly advanced disease[85]. Dental caries is a continuum of disease states of increasing severity and tooth destruction that ranges from sub-clinical sub-surface changes at the molecular level to lesions with dentinal involvement, either with an intact surface or obvious cavitation[86].

ETIOLOGY OF DENTAL CARIES:-

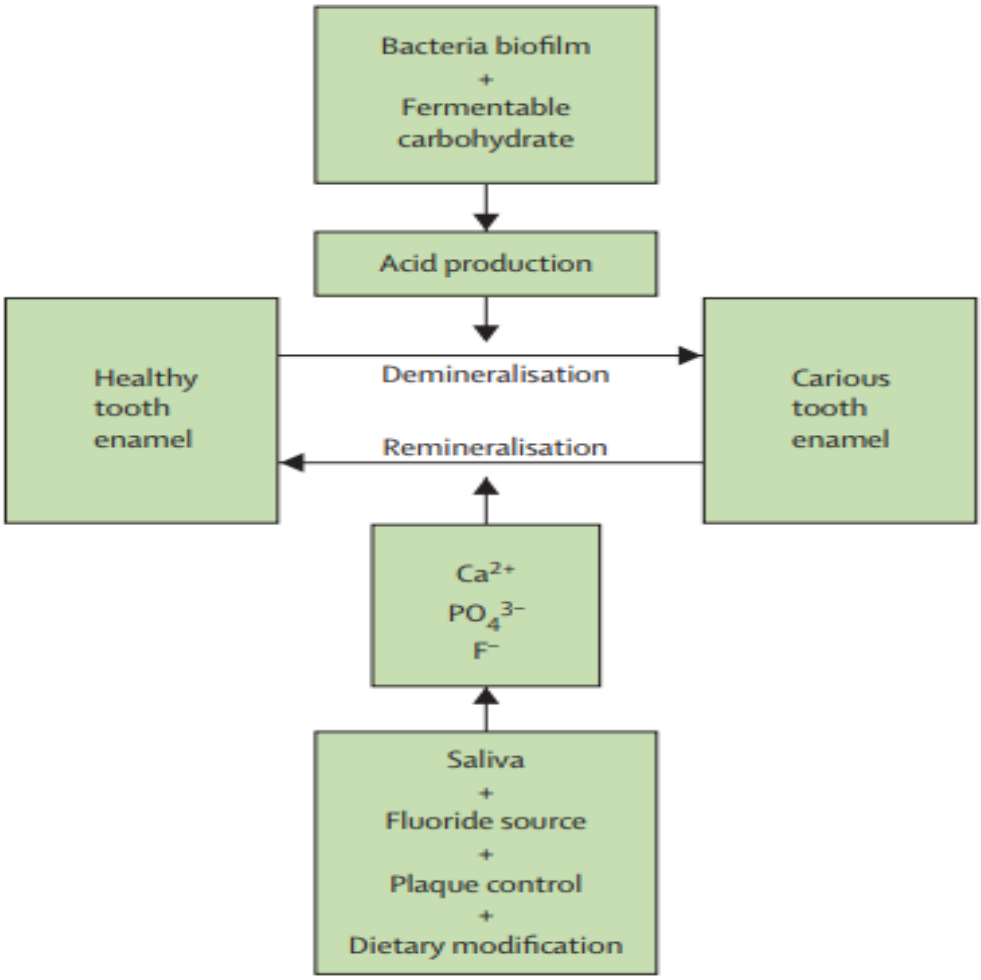


[FIGURE 5: ETIOLOGY OF DENTAL CARIES][87]

OTHER CAUSATIVE FACTORS:-

Other contributing factors in dental caries causation are salivary flow rate, buffering capacity, i.e., ability of saliva to neutralize acids and maintain its pH and availability of some protective enzymes and molecules in saliva[88].

PATHOGENESIS:-

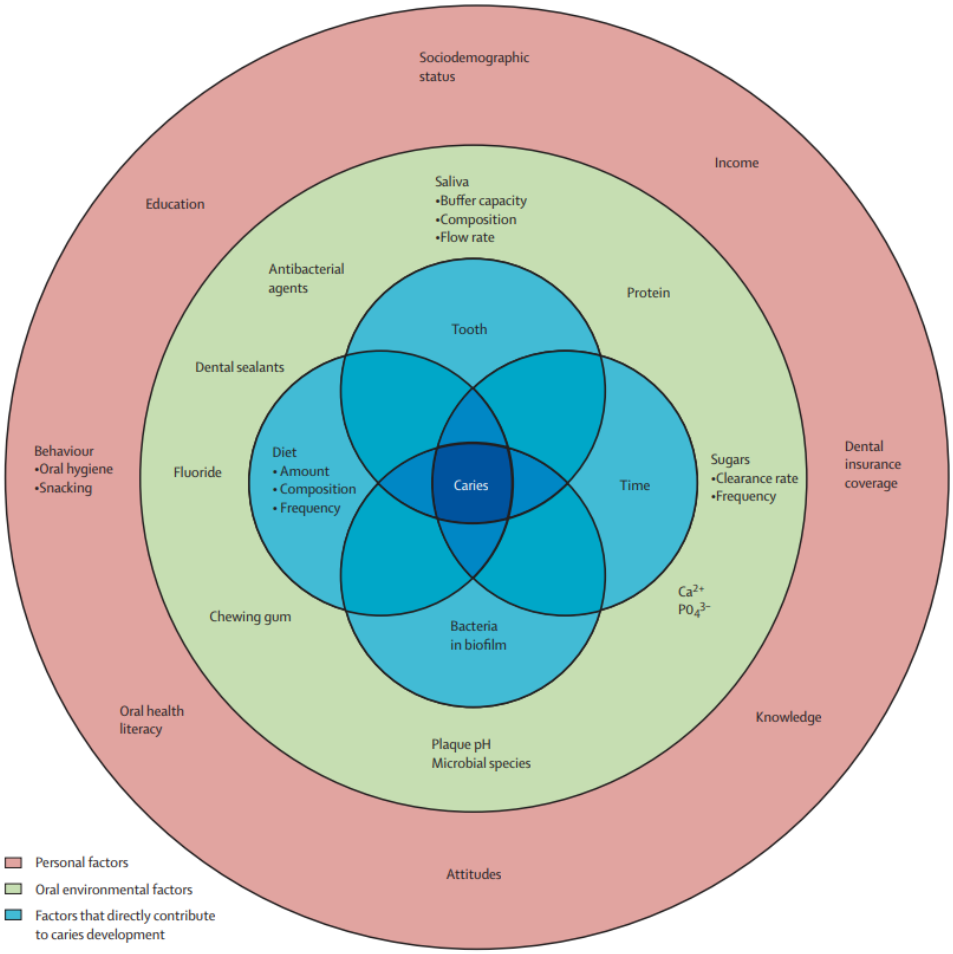


[FIGURE 6: PATHOGENESIS OF CARIES][89]

RISK FACTORS:-

A person's risk of caries can vary with time since many risk factors are changeable. Physical and biological risk factors for enamel or root caries include inadequate salivary flow and composition, high numbers of cariogenic bacteria, insufficient fluoride exposure, gingival recession, immunological components, need for special health care, and genetic factors[90,91,92,93,94].

FACTORS INVOLVED IN CARIES DEVELOPMENT:-



[FIGURE 7: FEATURES INVOLVED IN CARIES DEVELOPMENT][89]

HISTOPATHOLOGY:-

Zones of enamel caries→

- translucent zone
- dark zone
- body of the lesion and
- surface layer

Zones of dentin caries→

- Zone 1: Zone of fatty degeneration of odontoblast process
- Zone 2: Zone of dentinal sclerosis characterized by deposition of calcium salts in dentinal tubules
- Zone 3: Zone of decalcification of dentin, a narrow zone, preceding bacterial invasion

- Zone 4: Zone of bacterial invasion of decalcified but intact dentin
- Zone 5: Zone of decomposed dentin[95]

DIFFERENTIAL DIAGNOSIS - FLUOROSIS, ENAMEL AND DENTIN IMPERFECTAS, HYPOPLASIA:-

Initial lesion of dental caries may resemble an enamel hypoplastic spot or fluorosis. However, caries can be differentiated by wetting the surface with saliva or water. The hypoplastic or fluorosis spots in enamel are due to increase in opacity and it will remain the same after wetting the surface but in case of dental caries the spot is visible due to initial demineralization and porosity of enamel (loss of enamel translucency) and on wetting the surface the pores are filled with water causing disappearance of the white spot. The location of initial caries is normally near the gingival line (cervical) and hypoplasia and fluorosis is in the middle part of crown[87].

PREVENTION:-

- Oral health needs to be linked with general health.
- Cooperation and training of medical professionals, Anganwadi, and social workers required in emphasizing and promoting oral health care. Also, they can be trained to diagnose early carious lesions.
- **It has been reported that the four most prevalent noncommunicable diseases, cardiovascular diseases, diabetes, cancer, and chronic obstructive pulmonary diseases, have the same risk factors as oral diseases and can be controlled by lifestyle changes[96]. Thus, lifestyle changes can be promoted by dental and medical professionals.**
- School teachers can play a crucial role in awareness and implementation of preventive strategies, especially focusing on good dietary and brushing habits, and in promoting the importance of milk teeth.
- Social workers should be engaged in monitoring oral health especially in the underprivileged and special children.
- Government support would be required in planning and implementing the strategies.
- Timely surveillance of fluoride levels in water needs to be done.
- Fluoride toothpaste, gels, and varnishes that have been clearly documented as effective preventive measures toward dental caries should be practiced[97,98,99].

- Schools should be targeted for water fluoridation or sealant applications.
- When any survey is conducted, one should try to train and educate the mother or at least one family member for the detection of carious lesions or signs of early demineralization.

WHY DENTAL CARIES NEEDS ATTENTION?

- High Prevalence and Incidence:-

  In a recent publication[100] that secondarily analyzed the data provided in a publication in Lancet in 2017, it was stated that as compared to South Asians, Indians had a higher incidence of dental caries and more females suffered from this problem as compared to males.

- Unfavorable Sequelae of Dental Caries:-

  Untreated dental caries can lead to sequelae like severe pain, abscess, loss of the tooth, swelling, trismus, and systemic manifestations like fever and lymphadenopathy[101].

- Oral foci of infection:-

  A variety of situations exist in the oral cavity which are at least theoretical sources of infection and which may set up distant metastases. These include infected periapical lesions such as the periapical granuloma, cysts, or abscesses, teeth with infected root canals, periodontal disease with special reference to tooth extraction or manipulation[95].

- Increased Risk for Hospitalization

- Impact on Quality of Life:-

  Dental caries also detrimentally affect the quality of life as it can lead to problems in chewing, communication, disturbed sleep, and social interaction. This affects both children and adults.

- Rejection from Military Services:-

Tooth cavity and missing tooth have been reported to be the most common causes for not getting selected during medical screening prior to joining military services.

- Disparities in Caries Distribution:-

  It has been found that the prevalence of caries is not uniform throughout the subgroups of a country. Dental caries is more prevalent in poor and low socioeconomic groups.

- Economic Impact of Dental Caries:-

The demand of restorative treatment in the developing countries is higher than the resources available for public health programs. These funds are available only for emergency services like severe pain or trauma. If preventive and restorative procedures are carried out, the costs of treatment in children alone would exceed the total healthcare budget for children[101].

MATERIALS AND METHODS

The present cross sectional study was conducted in the Department of Oral Pathology and Microbiology, Babu Banarasi Das College of Dental Sciences, BBD University, Lucknow.

Study subjects included 100 subjects [50 uncontrolled Type II diabetes mellitus patients and 50 healthy controls] in age group of 30-80 years among the population of Lucknow. This age group was selected, as Type II diabetes mellitus is commonly seen in subjects above 30 years of age.

Eligibility Criteria:-

Inclusion criteria:

- Patients with known history of diabetes, dental caries and non diabetic individuals

Exclusion Criteria:

- Uncooperative, mentally and physically handicapped patients
- Patients with developmental anomalies, skin disorders, trauma or any pathology to fingertips, lips
- Those allergic to lipstick, ink pad, cellophane tape

Sampling Method:- Random Sampling

Materials and Equipments :-

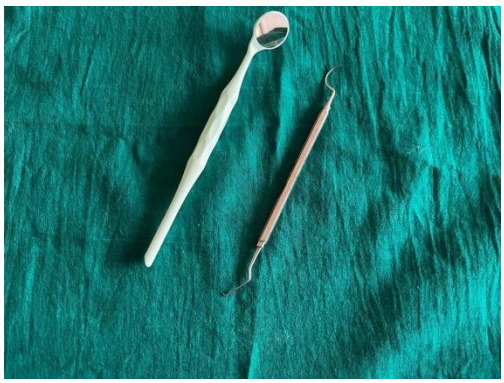
- Ear bud, lipstick and cellophane paper for recording lip prints

[FIGURE 8: EAR-BUD, LIPSTICK, CELLOPHANE FOR RECORDING LIP PRINT]

- Stamp pad [ink pad] with duplicating ink of blue colour for recording fingerprints

[FIGURE 9: INK PAD FOR RECORDING FINGERPRINT]

- Explorer tip and mouth mirror for assessment of dental caries



[FIGURE 10: EXPLORER TIP AND MOUTH MIRROR]

- Accu-check active glucometer kit for random blood sugar check

[FIGURE 11: GLUCOMETER KIT]

- Magnifying lens

[FIGURE 12: MAGNIFYING LENS]

Methodology:-

Methodology for lip prints analysis:

- Lips were first cleaned thoroughly and outlining of lips were done using sharp lip liner pencil
- Using one end of ear bud, lipstick was applied uniformly on upper lip starting from midline and spreading laterally
- Same procedure was repeated for lower lips
- Individual was asked to spread the lipstick evenly by gently rubbing his/her lips together
- At the relaxed lip position impression was taken on glued portion of cellophane tape by dabbing in centre first and then pressing comfortably near corners of lip
- Tape was carefully lifted from lip without smudging of print and impression will be stuck on plain paper
- By using cotton and Vaseline lipstick was removed from lips
- Magnifying lens was used to analyse lip prints by Suzuki and Tsuchihashi's classification

Methodology for finger print analysis:

- Patients hands were cleaned and dried
- Fingers of both hands were pressed on stamp pads and prints were transferred to a plain paper

- Then paper was allowed to dry
- By using magnifying lens finger prints were analysed by Henry's system of classification

Methodology for DMFT index:

- Prevalence of dental caries were obtained by calculating the number of decayed [D], missing [M], filled [F], teeth [T]
- Sum of 3 figures DMF were taken as the DMFT value; which was then recorded on paper

Methodology for random blood sugar testing:

- Using aseptic conditions finger was pricked by lancing device. Blood drop was collected to centre of green field
- Test results appear on display within few seconds; the value after beep was recorded

The obtained reading from above parameters were calculated and subjected to statistical analysis. The obtained statistical results are discussed by correlating with published English scientific literature.

OBSERVATIONS AND RESULTS

The present a cross-sectional study assess lip and finger print patterns in patients with Type II diabetes mellitus and dental caries in population of Lucknow city. Total 100, 50 non diabetic subjects and 50 age and gender matched Type II diabetes mellitus patients were recruited. The outcome measures of the study were lip and finger print patterns (or types), and dental carries (DMFT scores).

The objective of the study was (i) to compare the outcome measures (lip print pattern, finger print pattern and DMFT score) between two groups (non diabetic and diabetic), and (ii) to correlate the lip print pattern and finger print pattern with DMFT score in within and between two study groups.

Continuous data were summarised in Mean ± SE (standard error of the mean) whereas discrete (categorical) in number (n) and percentage (%). Continuous two independent groups were compared by independent Student's t test. Continuous groups were also compared by two factor (Groups x Lip or Finger print type) analysis of variance (ANOVA) and the significance of mean difference within (intra) and between (inter) the groups was done by Tukey's HSD (honestly significant difference) post hoc test after ascertaining normality by Shapiro-Wilk's test and homogeneity of variance between groups by Levene's test. Categorical groups were compared by chi-square (χ^2) test. The early diagnostic (sensitivity and specificity) of lip print patterns, finger print patterns and DMFT scores in determination of case (diabetic) and controls (non diabetic) was assessed using receiver operating characteristic (ROC) curve analysis. A two-tailed (α=2) $P < 0.05$ was considered statistically significant. Analyses were performed on SPSS software (Windows version 22.0).

Outcome measure

I. Lip print pattern

The lip print patterns (Type I, Type II, Type III and Type IV) of two groups (non diabetic and diabetic) is summarised in Table 1 and also depicted in Graph. 1. In non diabetic group, 19 (38.0%) subjects were with Type I, 9 (18.0%) with Type II, 13 (26.0%) with Type III and 9 (18.0%) with Type IV lip print patterns. In contrast, in diabetic group, it were 1 (2.0%), 13 (26.0%), 6 (12.0%) and 30 (60.0%) respectively.

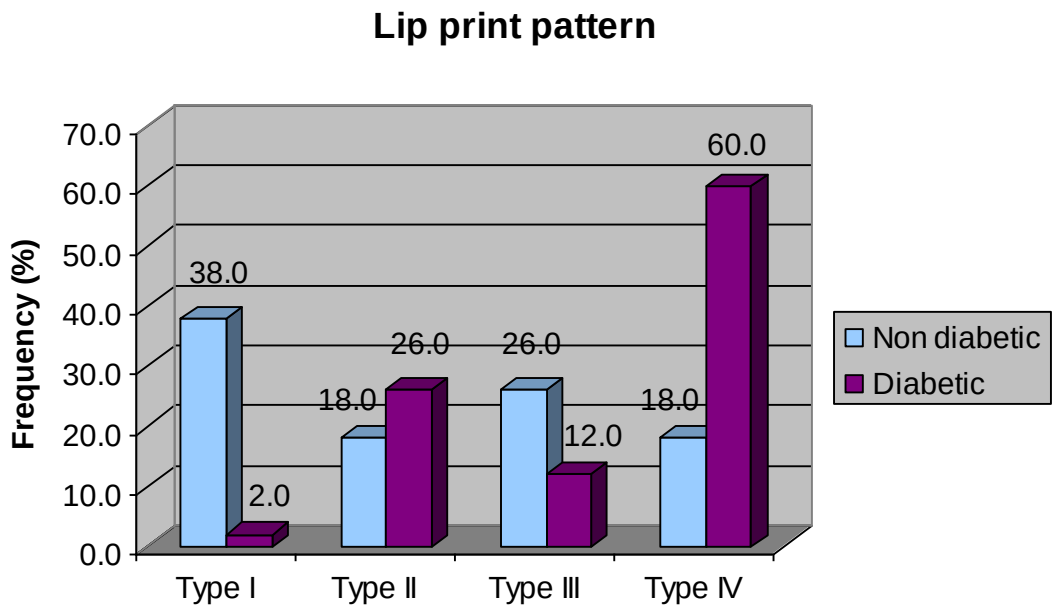
The frequency (%) of both Type I and Type III lip print patterns were higher in non diabetic subjects as compared to diabetic patients. In contrast, the frequency of both Type II and especially Type IV lip print pattern was comparatively higher in diabetic group as compared to non diabetic group.

Comparing the frequency of lip print patterns of two groups, χ^2 test showed significantly ($P < 0.001$) different lip print pattern between the two groups (χ^2=30.81, $P < 0.001$). The both Type I and Type IV lip print patterns make the difference significant which were 36.0% lower and 42.0% higher respectively in diabetic patients as compared to non diabetic subjects.

Table 1: Frequency distribution and comparison of lip print patterns of two groups

Lip print pattern	**Non diabetic (n=50) (%)**	**Diabetic (n=50) (%)**	**χ^2 value**	***P* Value**
Type I	19 (38.0)	1 (2.0)	30.81	< 0.001
Type II	9 (18.0)	13 (26.0)		
Type III	13 (26.0)	6 (12.0)		
Type IV	9 (18.0)	30 (60.0)		

The lip print patterns of two groups were summarised in number (n) and percentage (%) and compared by χ^2 test (χ^2 value).



Graph. 1. Frequency distribution of lip print patterns of two groups.

II. Finger print pattern

The finger print patterns (arch, loop and whorl) of two groups (non diabetic and diabetic) is summarised in Table 2 and also shown in Graph. 2. In non diabetic group, 3 (6.0%) had arch type, 31 (62.0%) loop type and 16 (32.0%) whorl type finger print patterns. In contrast, in diabetic group, it were 2 (4.0%), 29 (58.0%) and 19 (38.0%) respectively.

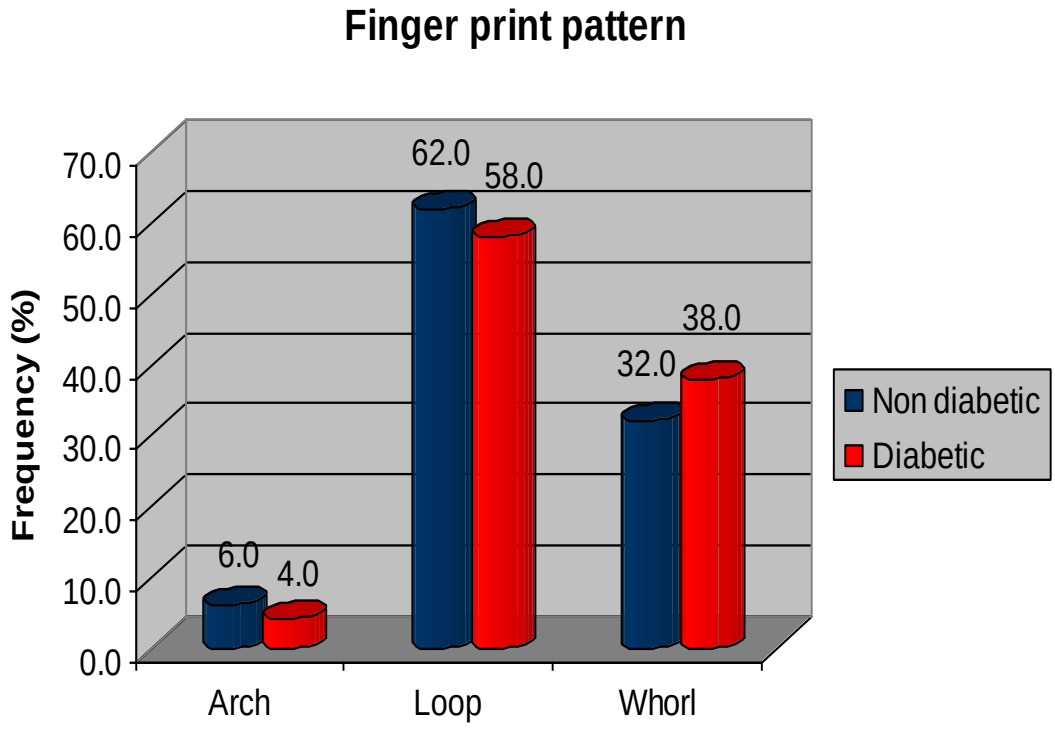
The frequency (%) of both loop and whorl type finger print patterns were slightly higher in non diabetic subjects as compared to diabetic patients. In contrast, the frequency of loop type finger print pattern was slightly higher in diabetic group as compared to non diabetic group.

Comparing the frequency of finger print patterns of two groups, χ^2 test showed similar ($P > 0.05$) finger print pattern between the two groups ($\chi^2=0.52$, $P = 0.770$) i.e. did not differ significantly.

Table 2: Frequency distribution and comparison of finger print patterns of two groups

Finger print pattern	**Non diabetic (n=50) (%)**	**Diabetic (n=50) (%)**	**χ^2 value**	***P* Value**
Arch	3 (6.0)	2 (4.0)	0.52	0.770
Loop	31 (62.0)	29 (58.0)		
Whorl	16 (32.0)	19 (38.0)		

The finger print patterns of two groups were summarised in number (n) and percentage (%) and compared by χ^2 test (χ^2 value).



Graph. 2. Frequency distribution of finger print patterns of two groups.

III. DMFT score

The DMFT score of two groups (non diabetic and diabetic) is summarised in Table 3 and also shown in Graph. 3. The DMFT score in non diabetic group ranged from 0 to 24 with mean (± SE) 3.66 ± 0.56 and median 3 whereas in diabetic group, it ranged from 0 to 28 with mean (± SE) 6.82 ± 0.81 and median 6.

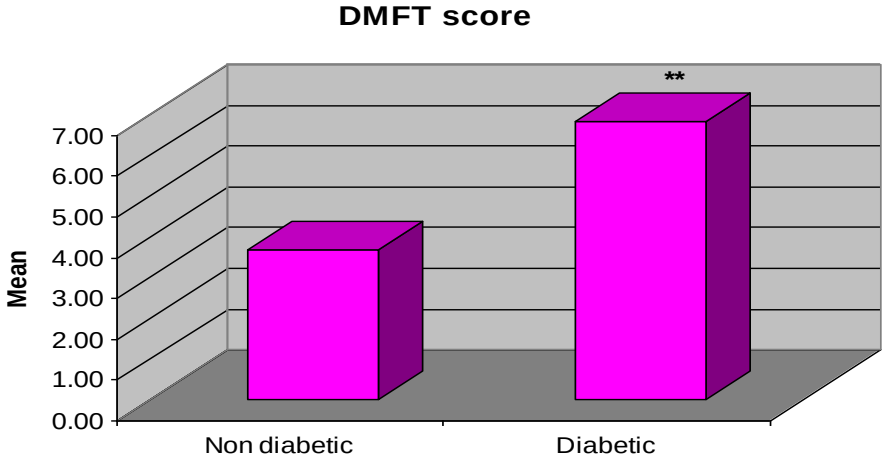
The mean DMFT score was comparatively higher in diabetic group as compared to non diabetic group.

Comparing the mean DMFT score of two groups, Student's t test showed significantly ($P < 0.01$) different and higher (46.3%) DMFT score in diabetic group as compared to non diabetic group (3.66 ± 0.56 vs. 6.82 ± 0.81, mean difference=3.16 ± 0.98, 95% CI of difference=1.21 to 5.11, t=3.21, $P = 0.002$).

Table 3: Distribution and comparison of DMFT score of two groups

Non diabetic (n=50) (%)	**Diabetic (n=50) (%)**	**Mean difference (95% CI)**	**t value**	***P* value**
3.66 ± 0.56 (0 to 24)	6.82 ± 0.81 (0 to 28)	3.16 ± 0.98 (1.21 to 5.11)	3.21	0.002

The DMFT score of two groups were summarised in Mean ± SE and range (min to max) and compared by Student's t test (t value). **CI:** confidence interval.



**$P < 0.01$- as compared to Non diabetic

Graph. 3. Distribution and comparison of mean DMFT score of two groups.

Correlation

I. DMFT score and Lip print patterns

The correlation (i.e. comparison) of lip print patterns (Type I, Type II, Type III and Type IV) with DMFT score of two groups (non diabetic and diabetic) is summarised in Table 4 and also shown in Graph. 4 and 5. In non diabetic group, the mean DMFT score was highest in Type III followed by Type IV, Type II and Type I, the least (Type I < Type II < Type IV < Type III). In contrast, in diabetic group, it was highest in Type III followed by Type II, Type IV and Type I, the least (Type I < Type IV < Type II < Type III). Further, in all four lip print patterns, the mean DMFT score was higher in diabetic group as compared to non diabetic group.

For each group, comparing the difference in mean DMFT score between lip print patterns (i.e. intra group), Tukey test showed similar ($P > 0.05$) DMFT score among all four lip print patterns in both groups i.e. did not differ significantly (Table 5 and Graph. 4).

Similarly, for each lip print pattern, comparing the difference in mean DMFT score between groups (i.e. inter group), Tukey test further showed similar ($P > 0.05$) DMFT score between two groups at all lip print patterns i.e. also not differ significantly (Table 4 and Graph. 5).

In conclusion, DMFT score did not correlate well with lip print patterns in both non diabetic subjects and diabetic patients.

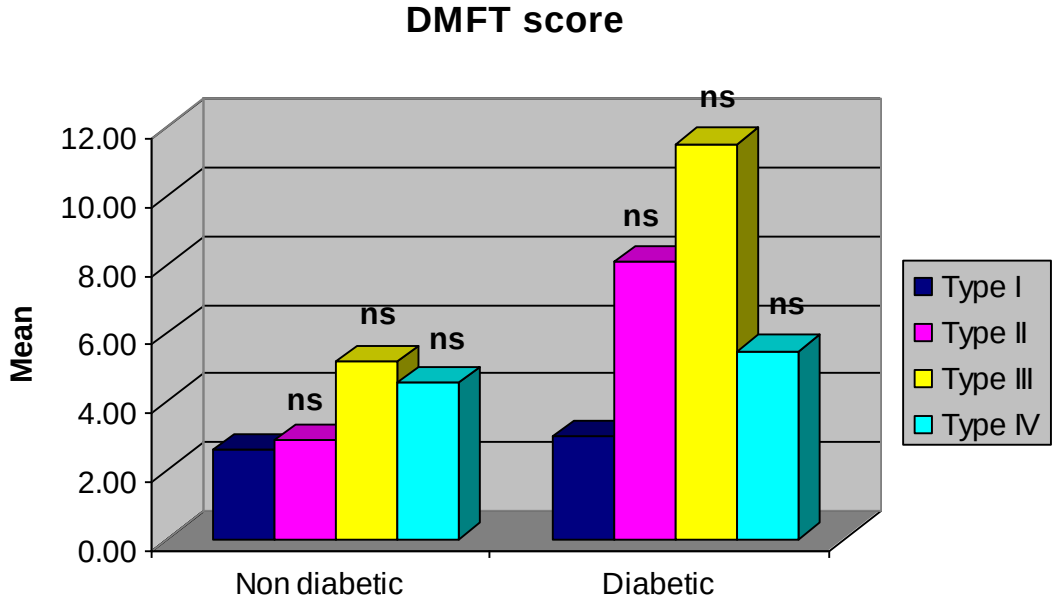
Table 4: Correlation of lip print patterns with DMFT score of two groups

Lip print pattern	DMFT score					*P* Value
	Non diabetic (n=50)		Diabetic (n=50)		Mean difference	
	N	Mean ± SE	n	Mean ± SE		
Type I	19	2.58 ± 0.62	1	3.00 ± 0.00	0.42	1.000
Type II	9	2.89 ± 0.82	13	8.08 ± 1.11	5.19	0.203
Type III	13	5.15 ± 1.72	6	11.50 ± 3.53	6.35	0.135
Type IV	9	4.56 ± 0.97	30	5.47 ± 0.96	0.91	1.000

The DMFT score according to lip print patterns of two groups were summarised in Mean ± SE and compared by Tukey post hoc test (*P* value).

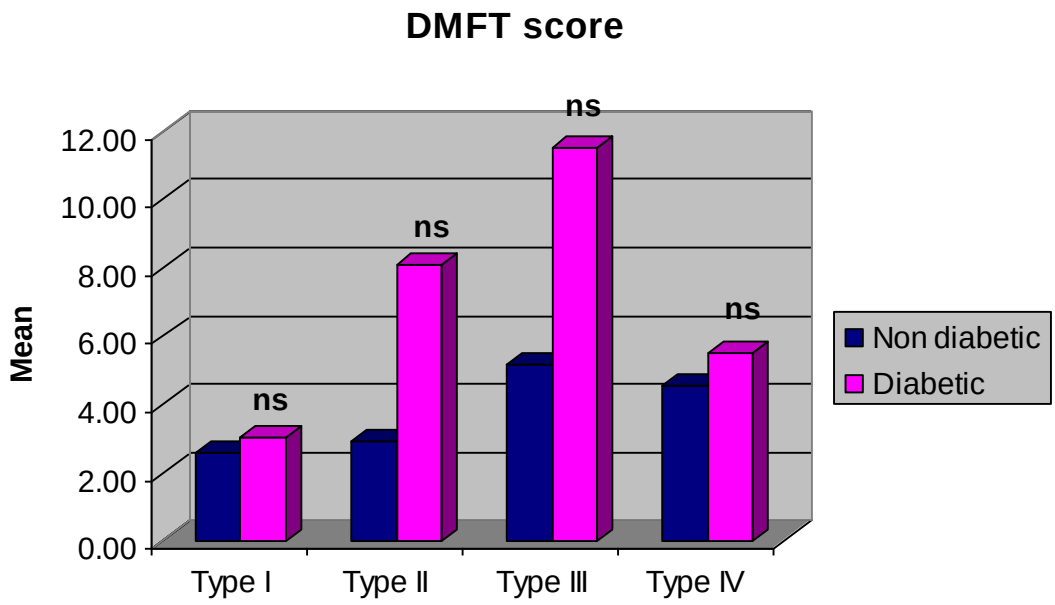
Table 5: For each group, comparison (*P* value) of difference in mean DMFT score between lip print patterns by Tukey post hoc test

Comparison- Lip print pattern	Non diabetic		Diabetic	
	Mean difference	*P* value	Mean difference	*P* value
Type I vs. Type II	0.31	1.000	5.08	0.969
Type I vs. Type III	2.57	0.804	8.50	0.716
Type I vs. Type IV	1.98	0.969	2.47	1.000
Type II vs. Type III	2.26	0.956	3.42	0.828
Type II vs. Type IV	1.67	0.995	2.61	0.718
Type III vs. Type IV	0.59	1.000	6.03	0.099



[ns]*P* > 0.05- as compared to Type I

Graph. 4. For each group, comparisons of difference in mean DMFT score between lip print patterns.



$^{ns}P > 0.05$- as compared to Non diabetic

Graph. 5. For each lip print pattern, comparisons of difference in mean DMFT score between two groups.

II. DMFT score and Finger print patterns

The correlation (i.e. comparison) of finger print patterns (arch, loop and whorl) with DMFT score of two groups (non diabetic and diabetic) is summarised in Table 6 and also shown in Graph. 6 and 7. In non diabetic group, the mean DMFT score was highest in arch followed by whorl and loop, the least (loop < whorl < arch). In contrast, in diabetic group, it was highest in loop followed by whorl and arch, the least (arch < whorl < loop). Further, in both loop and whorl, the mean DMFT score was higher in diabetic group as compared to non diabetic group but in arch, it was higher in non diabetic group as compared to diabetic group.

For each group, comparing the difference in mean DMFT score between finger print patterns (i.e. intra group), Tukey test showed similar ($P > 0.05$) DMFT score among all three finger print patterns in both groups i.e. did not differ significantly (Table 7 and Graph. 6).

Similarly, for each finger print pattern, comparing the difference in mean DMFT score between groups (i.e. inter group), Tukey test showed significantly ($P < 0.01$) different and higher (58.4%) DMFT score in loop of diabetic patients as compared to non diabetic subjects (Table 6 and Graph. 7). However, in both arch and whorl, it did not differ ($P > 0.05$) between the two groups i.e. found to be statistically the same.

In conclusion, DMFT score correlate well with finger print patterns especially loop type between non diabetic subjects and diabetic patients.

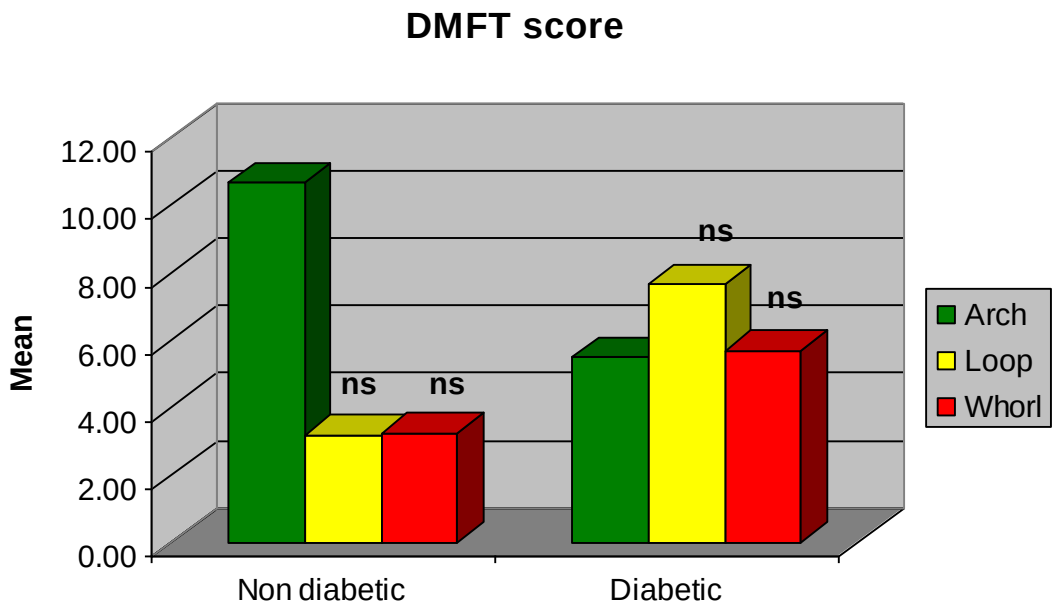
Table 6: Correlation of finger print patterns with DMFT score of two groups

Finger print pattern	**DMFT score**					***P* value**
	Non diabetic (n=50)		**Diabetic (n=50)**		**Mean difference**	
	N	**Mean ± SE**	**N**	**Mean ± SE**		
Arch	3	10.67 ± 6.67	2	5.50 ± 0.50	5.17	0.845
Loop	31	3.19 ± 0.55	29	7.66 ± 1.21	4.47	0.007
Whorl	16	3.25 ± 0.53	19	5.68 ± 1.03	2.43	0.668

The DMFT score according to finger print patterns of two groups were summarised in Mean ± SE and compared by Tukey post hoc test (*P* value).

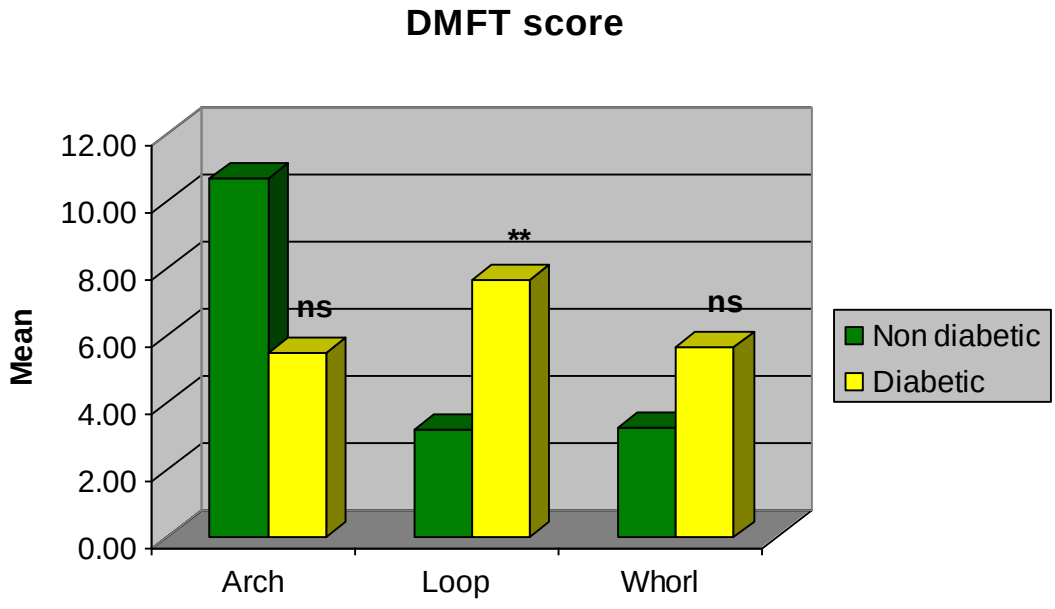
Table 7: For each group, comparison (*P* value) of difference in mean DMFT score between finger print patterns by Tukey post hoc test

Comparison- Finger print pattern	**Non diabetic**		**Diabetic**	
	Mean difference	***P* value**	**Mean difference**	***P* Value**
Arch vs. Loop	7.48	0.114	2.16	0.990
Arch vs. Whorl	7.42	0.148	0.18	1.000
Loop vs. Whorl	0.06	1.000	1.98	0.732



$^{ns}P > 0.05$- as compared to Arch

Graph. 6. For each group, comparisons of difference in mean DMFT score between finger print patterns.



$^{ns}P > 0.05$ or $^{**}P < 0.01$- as compared to Non diabetic

Graph. 7. For each finger print pattern, comparisons of difference in mean DMFT score between two groups.

Early screening or diagnosis

To assess whether lip print patterns, finger print pattern and DMFT scores may discriminates subjects of two groups (non diabetic and diabetic), ROC curve analysis was done and summarised in Table 8 and also shown in Graph. 8-10, respectively. The ROC curve analysis showed significant diagnostic of both lip print patterns (AUC=0.755, Z=5.26, $P < 0.001$) and DMFT score (AUC=0.693, Z=3.68, $P < 0.001$) but insignificant diagnostic of finger print patterns (AUC=0.535, Z=0.61, $P = 0.545$) in discriminations of subjects of two groups.

Further, the lip print patterns > Type III discriminating the subjects of two groups with high sensitivity 60.00% (95% CI: 45.2-73.6) and high specificity 82.00% (95% CI: 68.6-91.4) and with 76.9% positive predictive value and 67.2% negative predictive value.

Similarly, the DMFT score at cut-off value of >5 also discriminating the subjects of two groups with high sensitivity 54.00% (95% CI: 39.3-68.2) and high specificity 82.00% (95% CI: 68.6-91.4) and with 75.0% positive predictive value and 64.1% negative predictive value.

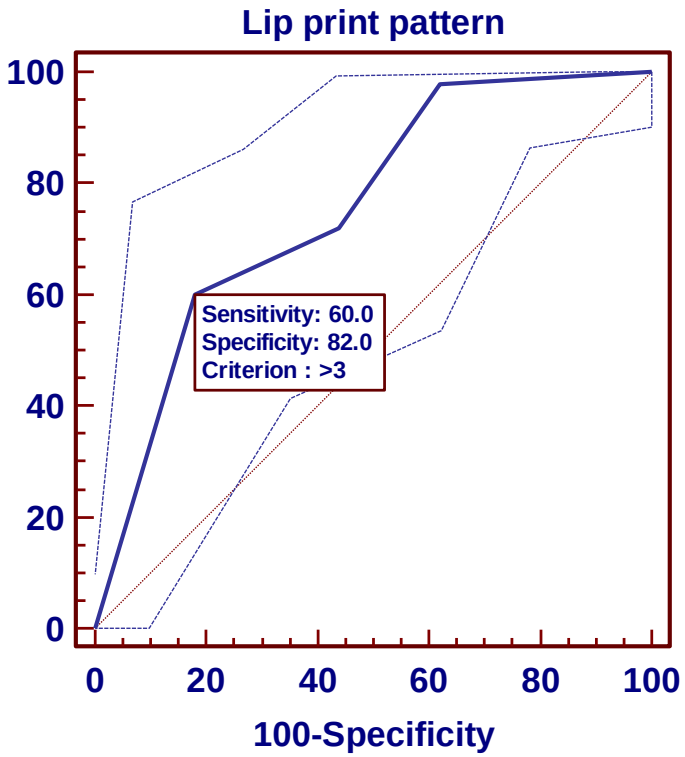
Further, among outcome measures (lip print patterns, finger print pattern and DMFT scores), the early screening/diagnostic of lip print patterns was found to be the highest followed by DMFT scores and finger print patterns, the least.

In conclusion, lip print patterns and DMFT scores can be used as early screening/diagnosis in type II diabetic mellitus with dental caries.

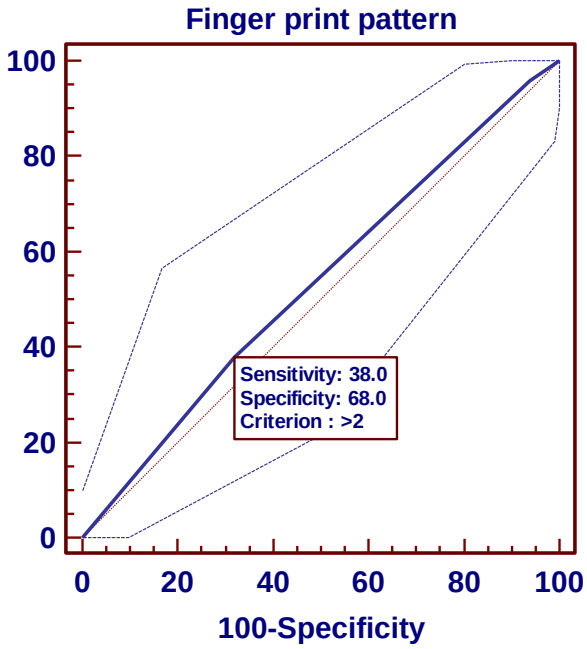
Table 8: Diagnostic accuracy of lip print patterns, finger print patterns and DMFT scores in discrimination of non diabetic subjects and type II diabetes mellitus patients using ROC curve analysis

Variable	Cut off value	Sensitivity (95% CI)	Specificity (95% CI)	+PV	-PV
Lip print patterns	>3[#]	60.00 (45.2-73.6)	82.00 (68.6-91.4)	76.9	67.2
Finger print patterns	>2[ε]	38.00 (24.7-52.8)	68.00 (53.3-80.5)	54.3	52.3
DMFT score	>5	54.00 (39.3-68.2)	82.00 (68.6-91.4)	75.0	64.1

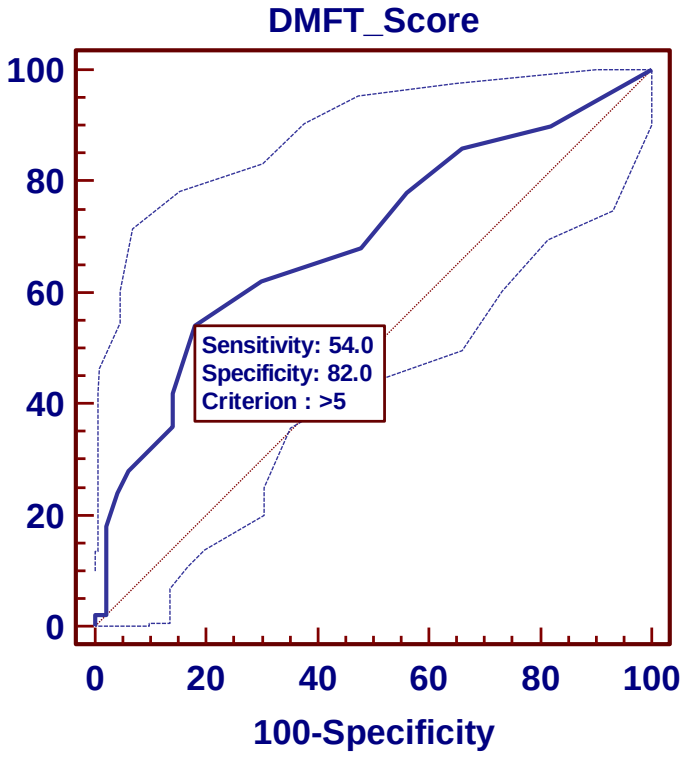
CI: confidence interval, **+PV:** positive predictive value, **-PV:** negative predictive value. **Lip print patterns >3[≠]**: >Type III and Type IV, and **Finger print patterns >2[ε]:** >Loop and whorl types.



Graph. 8. Diagnostic accuracy of lip print patterns in non diabetic subjects and type II diabetes mellitus patients using ROC curve analysis.



Graph. 9. Diagnostic accuracy of finger print patterns in non diabetic subjects and type II diabetes mellitus patients using ROC curve analysis.



Graph. 10. Diagnostic accuracy of DMFT scores in non diabetic subjects and type II diabetes mellitus patients using ROC curve analysis.

DISCUSSION

India is known as the 'diabetic capital' of the world owing to increased blood sugar levels associated with our food habits and lifestyle[102]. Diabetes Mellitus is a multi-factorial chronic systemic disease which is triggered by number of genetic and environmental factors. It is considered to be a slow and silent killer. Also in the civilized world with refined dietary habits, dental caries and other oral diseases are common and found in majority of present population. There is a definite correlation between high blood sugar levels found in diabetes mellitus patients and increased prevalence of dental caries in them[103]. Despite of being an epidemic; diagnosis and treatment of diabetes mellitus remains expensive.

As a disease of the civilized, dental caries is common among Indians and it warrants intensive dental procedures; the cost of which is high making it unaffordable to a major chunk of the population. Considering high cost involved in the treatment of both diabetes mellitus and dental caries our study can give a midway to correlate them by rendering their early and effective diagnosis by cost effective means; indirectly reducing the burden on healthcare system.

Genetic markers such as lip prints and fingerprints are unique to each individual and remain constant throughout life, thereby helping us predict the probable future occurrence of diabetes mellitus & dental caries at an early age. Lip prints and finger prints are widely used for identification in forensics and can be recorded cost effectively with good patient compliance.

Hence we conceptualized this study to evaluate the different types of lip prints and finger prints in diabetes mellitus and dental caries and to assess if they can be used as predictive factors for screening and also to assess correlation between the most common diseases [Diabetes mellitus and Dental caries] if any.

EMBRYOLOGY OF FINGER PRINTS: A fingerprint is an impression left by the friction ridges of human fingers. The embryological development of finger prints can be traced back to volar pads that develop on hands/feet at 7 weeks I.U from the

thumb to the little finger; also corresponding to the timeline of tooth formation. Volar pads are transient mesenchymal elevations situated below the epidermis on the palmar surface of the hands and soles of the human fetus. As a result of the volar pads' slowing growth, their contour becomes progressively less distinct as compared to the more rapidly growing epidermis; the process defined as "regression[104]. At 10 weeks I.U, as the pads are differentially reabsorbed into the palms, basal epidermis starts to fold under pressure from the growing skin. If the volar pad regresses symmetrically, it will form a whorl. Volar pads on partial asymmetrical absorption, results in a loop and if entirely absorbed, the pattern will be arch[105]**.** The ridge patterns are completed by 12^{th}–14^{th} week of gestation, i.e. simultaneous to completion of tooth formation in intra-embryonic life. Primary genetic determination and development secondary to flexion function have been suggested as the mechanisms underlying crease development[106]. Hence, each fingerprint is a variation of 3 broad categories: ARCH, LOOP and WHORL. Fingerprint patterns are so unique to an individual that even identical twins don't share them[107]. Dermatoglyphic patterns stay stable from womb to tomb.

Development of nerves also play a remarkable role in development of epithelial ridges. Neurovascular bundles are seen in the developing dermis by 6 weeks EGA and innervates the overlying epidermis by 9 weeks EGA followed by merkel cells in the epidermis by the 10^{th} week. Developing nerves may interact with epidermal cells to stimulate clustered interactions that blend in the early stages of ridge development[108,109]. Literature search reveals that innervation at the sites of ridge formation immediately preceding the appearance of friction ridges and suggest that innervation could be the trigger mechanism for the onset of proliferation[108,110,111].

Several researchers postulate that the patterning of the capillary–nerve pairs at the epidermal- dermal junction is the direct cause of primary ridge alignment[108,111,112,113]**.** The presence of nerves and capillaries in the dermis before friction ridge formation may be necessary for friction ridge proliferation. Friction ridge formation would benefit from being in communication with the central nervous system or the endocrine and exocrine (hormone) systems. According to these models, hormones circulate first through newly formed capillaries just before ridge formation in the epidermis, offering another potential factor in the genesis of ridge formation[109].

Ridge pattern distributions are thought to follow "developmental fields", i.e, groups of fingers may share similar patterns[114,115,116]. Later discoveries confirmed the neurological relation of spinal cord sections C–6, C–7, and C–8 to innervation of the fingers[117]. Kahn et al (2001) reported a large ridge-count difference between C–8-controlled fingers 4 and 5; which may predict a larger waist-to-thigh ratio and, therefore, an increased risk of major chronic diseases such as heart disease, cancer, and diabetes[118].

We found it interesting to note that embryologically the chronology of development of teeth, lips and fingerprints are simultaneous (6-9 weeks I.U); and both develop from the ectoderm; perhaps the reason for their abnormalities to be related too. The genetic and environmental factors responsible for dental caries may also cause peculiarities in the dermatoglyphic patterns[119]. Altered dermatoglyphic patterns were seen in Ellis–van Creveld syndrome[120], in dental caries[121] and in hypohydrotic ectodermal dysplasia[122] which reiterates the importance of chromosomal alterations and perhaps altered nerve innervations in their genesis. Dermatoglyphics can be used in the detection of Genetic disorders like Klinefelter's syndrome, Trisomy, Turner, Schizophrenia, Down syndrome, Non-genetic like Epilepsy, Heart diseases, Bronchial Asthma, Leprosy, TB, Diabetes, Hyper tension, Cancer, Dental disorders like Cleft lip and palate (CL/P), Dental caries, Periodontal diseases and Potentially malignant disorders and oral carcinomas[123].

EMBRYOLOGY OF Diabetes Mellitus: Diabetes mellitus is a disorder in which body does not produce enough insulin or normally respond to it, causing blood glucose levels to be abnormally high. It is of 2 types: Diabetes mellitus type I and Diabetes mellitus type II. The beta cells of islets of Langerhans situated on the pancreas, produces insulin that plays a key role in pathogenesis of diabetes mellitus. Beta cells start to develop at 12 weeks I.U [124] with their maximum growth at 4 days after birth and then negligible after the 10th day of life. Type II Diabetes Mellitus with its long latency before diagnosis causes several long term complications in the major organ systems[125]. The pre-diabetic stage, which spans through several years, is comparatively shorter in Indians[126]. Therefore dermatoglyphics in Type II Diabetes Mellitus can bridge the gap between predisposition, the pre-diabetic stage and the diagnosis. Type II Diabetes Mellitus has been described as a geneticist's nightmare

since several genetic factors have been linked to the disease, spanning more than 70 genes at multiple loci, with familial tendency, polygenic mode of inheritance, phenotypic expression modified by environmental factors throughout the lifespan[127]. It is therefore difficult to predict its occurrence by a specific genetic test. We strongly believe the early growth process of beta islets and its strong relation to development of a branched neural network has a huge role to play in their development; which is similar to that of finger ridges. Since the islet cells, the epidermal ridges and associated nerves are ectodermal in origin and are initiated simultaneously, any metabolic insult to the growing fetus will affect all these structures.

Epidermal ridges of the fingers are supplied by the branchial plexus consisting of radial, ulnar, median nerves which are part of the peripheral nervous system; whereas, the pancreatic islets are supplied by Vagus nerve which is a cranial nerve. Two nerves that completely originate from the roots of branchial plexus are dorsal scapular nerve (from C5) and the long thoracic nerve (from C5, C6 and C7). C5 root also contributes to the phrenic nerve which therefore is indirectly a part of the branchial plexus though it doesn't supply the finger ridges[128]. Though unrelated by origin, vagus and phrenic nerves share the same course till the diaphragm. We believe this proximity may have a role in the correlation between diabetes mellitus and specific dermatoglyphic patterns.

Development of lip prints: Lip prints are patterns that are recorded from the surface of lips owing to surface elevations and depressions. Though we couldn't find any literature about the embryological basis of their development, we believe the elevations correspond to relatively increased thickness of epithelial cells and depressions correspond to higher CT cores whose arrangement may be dependent on capillary-nerve innervation patterns during embryogenesis as lip is a highly innervated zone.

Development of dental caries: In addition to the well-known factors causing dental caries, like fermentable sugars, tooth surface, time and microbiota, genetic susceptibility of caries is also important. The role of BCOR and BCORL1 (2x-linked genes with sequence similarity) in pit & fissure and smooth surface caries has been documented[129]. Mutations of the BCOR gene prevents the production of functional proteins, which disrupts the prenatal development of organs, as seen in

oculo-facio-cardio-dental syndrome which presents with rhizomegaly of teeth, oligodontia, supernumerary teeth, malformed permanent teeth, malocclusion, root dilacerations, macrodontia, enamel defects, and cleft palate. Thus, BCOR may play a role in the enamel, palate & rugae development. Enamel is usually the first structure which gets affected by caries. The epithelium of fingers develops during the same intrauterine period as the development of enamel and other craniofacial structures such as lips and palate; hence, both genetic and environmental factors affecting one can affect the other. Thus, fingerprints, lip prints, and palatal rugae patterns can be used for detecting and preventing caries at an early age[130].

CORRELATION OF FINGERPRINTS AND DIABETES MELLITUS: In the present study, we compared predominant fingerprint patterns in diabetic and non-diabetic individuals. The frequency of loop pattern was slightly higher in the diabetic group. As we have already discussed, loop pattern results from asymmetric volar pad regression. We believe this gives us the predictive edge because genetic alterations in diabetes and asymmetry in volar pad regression may reveal a correlation at the embryological level. The frequency of both loop and whorl patterns were slightly higher in non-diabetic subjects. The fingerprint pattern between the two groups did not differ significantly as our study sample was not big enough. Our result coincides with various documented research. Mouneshkumar CD et.al assessed cheiloscopic and dermatoglyphic patterns in hypertension and type II diabetes and found the ulnar loop pattern to be the most frequent in both genders[131]. Manjusha P et al assessed the predictive role of cheiloscopy and dermatoglyphics on type II diabetes mellitus and found that loop pattern was higher in both diabetic and nondiabetics[132]. However Jeddy N et al[133], Ramhari Sathawane et al[134] and Srivastava S et al[135] found a higher frequency of whorl pattern in diabetics and non diabetics. Whorls are a result of symmetric volar pad regression. However, we found no studies that reported predominant arch type pattern in diabetics. Arches form when the entire volar pad is absorbed. So, does it imply that both whorls and loops can be early diagnostic markers for diabetes; and is there a possibility that these patterns follow a gradient manner in relation to severity of diabetes where whorls correlate with milder version of the disease and loops represent a severe version? We believe this is a scope for future research. If we succeed in this correlation, we can possibly predict development of diabetes at an early age thereby acting early to prevent diabetic

complications in the later years; thereby reducing the healthcare burden of a developing country like India.

CORRELATION OF LIP PRINTS AND DIABETES MELLITUS: Lip prints are analogous to fingerprints and used for personal identification as they are unique and remain stable for life. We compared lip print patterns among diabetic and non-diabetic individuals and found the frequency of Type II and especially Type IV lip print patterns comparatively higher among diabetics, whereas Type I and Type III patterns were higher in non-diabetic subjects. Type IV pattern is the reticular type which perhaps can be correlated to some asymmetry whereas type I pattern is regular vertical lines. So, we can see the asymmetry in Diabetes Mellitus cases which is absent in Non Diabetic group. Similar results were seen in studies conducted by Manjusha P et al, Jeddy N et al, Ramhari Sathawane et al and Ramnathan V et al where Type IV pattern of lip prints showed a greater association with Type II Diabetes Mellitus subjects[132,133,134,136]. Our study reiterates the role of application of cheiloscopy as a potential biomarker in the early diagnosis of type II diabetes mellitus to be used in mass screening.

CORRELATION OF FINGERPRINTS AND DENTAL CARIES: Between 6th and 7th week of intrauterine life, the ridged skin and teeth develop from ectoderm which suggests that the genetic information stored in the genome is dissipated during this period, and any disturbance affecting odontogenesis may be simultaneously reflected through changes in dermatoglyphic patterns or viceversa[137]**.** Diabetic subjects with dental caries (Mean DMFT) showed a predominant loop pattern fingerprints followed by whorl and arch whereas in the non diabetic group with caries, arch pattern followed by whorl and loop was more prevalent. DMFT score correlated well with fingerprint patterns. Few studies have emphasized the increased loop pattern of fingerprints among dental caries (Maitrayee et al, Shetty SS et al, Nallanchakrava S et al, Deepti A et al, Madhusudam K et al)[138,139,140,141,142]. Also, several studies reported an increased DMFT in diabetes mellitus (Latti BR et al, Malvania EA et al and Sridevi N et al) which reiterates the fact that diabetic mouths presented with increased caries[143,144,145].

CORRELATION OF LIP PRINTS AND DENTAL CARIES:-

In diabetic group, the mean DMFT score or caries incidence was highest in patients with Type III lip prints followed by Type II, Type IV and Type I (Type I < Type IV < Type II < Type III). In non-diabetic group, the mean DMFT score was highest in Type III followed by Type IV, Type II and Type I, the least (Type I < Type II < Type IV < Type III). Hence Lip print patterns did not correlate well with dental caries.

CONCLUSIONS

We found loop type fingerprints and type IV lip prints associated with diabetic patients. Both Loop type fingerprint and type IV lip print represent asymmetry (in volar pad reabsorption pattern and in lip surface elevations respectively) and is seen associated-with, in diabetics. Non-diabetics showed loop type fingerprints and type I lip prints. We found increased dental caries incidence (DMFT scores) in diabetics. Diabetic subjects with caries showed loop fingerprints which reiterated our earlier findings but did not correlate with type IV lip prints. Non-diabetics with caries showed arch fingerprints but did not correlate with type I lip prints. We found that DMFT scores that we used to assess caries did not correlate well with lip prints.

The results from our study strongly suggests that dermatoglyphics can be used as a non-invasive technique to mass screen for diabetes as well as dental caries as both diseases are predominantly associated with loop type fingerprints. Type IV lip prints could be used to screen for diabetes but no correlation of lip prints were seen in patients with caries.

Correlation can help prediction, which can be helpful to provide preventive and interceptive treatment options. For India, it might prove to be a noninvasive, inexpensive and effective tool for predicting Diabetes Mellitus & Dental Caries. Further extensive research and studies are warranted in order to determine, ascertain and to evaluate the significance of the aforementioned findings.

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APPENDICES

ANNEXURE – I

BABU BANARASI DAS COLLEGE OF DENTAL SCIENCES (FACULTY OF BBD UNIVERSITY), LUCKNOW

INSTITUTIONAL RESEARCH COMMITTEE APPROVAL

The project titled **"Assessment of lip and finger print patterns in patients with type 2 diabetes mellitus and dental caries in Lucknow: A cross sectional study"** submitted by **Dr Dakshayani Vijay Patil** Post graduate student from the **Department of Oral Pathology & Microbiology** as part of MDS Curriculum for the academic year 2020-2023 with the accompanying proforma was reviewed by the Institutional Research Committee present on **12th October 2021** at BBDCODS.

The Committee has granted approval on the scientific content of the project. The proposal may now be reviewed by the Institutional Ethics Committee for granting ethical approval.

Prof. Vandana A Pant
Co-Chairperson

Prof. B. Rajkumar
Chairperson

ANNEXURE – II

Babu Banarasi Das University
Babu Banarasi Das College of Dental Sciences,
BBD City, Faizabad Road, Lucknow – 226028 (INDIA)

Dr. Lakshmi Bala
Professor and Head Biochemistry and
Member-Secretary, Institutional Ethics Committee

Communication of the Decision of the IXth Institutional Ethics Sub-Committee

IEC Code: 01 **BBDCODS/04/2022**

Title of the Project: Assessment of lip and finger print patterns in patients with type 2 diabetes mellitus and dental caries in Lucknow: A cross sectional study.

Principal Investigator: Dr Dakshayani Vijay Patil **Department:** Oral Pathology & Microbiology

Name and Address of the Institution: BBD College of Dental Sciences Lucknow.

Type of Submission: New, MDS Project Protocol

Dear Dr Dakshayani Vijay Patil,

The Institutional Ethics Sub-Committee meeting comprising following four members was held on 07th April, 2022.

1.	Dr. Lakshmi Bala Member Secretary	Prof. and Head, Department of Biochemistry, BBDCODS, Lucknow
2.	Dr. Amrit Tandan Member	Prof. & Head, Department of Prosthodontics and Crown & Bridge, BBDCODS, Lucknow
3.	Dr. Rana Pratap Maurya Member	Reader, Department of Orthodontics, BBDCODS, Lucknow
4.	Dr. Akanksha Bhatt Member	Reader, Department of Conservative Dentistry & Endodontics, BBDCODS, Lucknow

The committee reviewed and discussed your submitted documents of the current MDS Project Protocol in the meeting.
The comments were communicated to PI thereafter it was revised.

Decisions: The committee approved the above protocol from ethics point of view.

Forwarded by:

Lakshmi Bala

(Dr. Lakshmi Bala)
Member-Secretary
IEC

Member-Secretary
Institutional Ethic Committee
BBD College of Dental Sciences
BBD University
Faizabad Road, Lucknow-226028

(Dr. Puneet Ahuja)
Principal
BBDCODS

PRINCIPAL
Babu Banarasi Das College of Dental Sciences
(Babu Banarasi Das University)
BBD City, Faizabad Road, Lucknow-226028

ANNEXURE – III

Babu Banarasi Das College of Dental Sciences

(Babu Banarasi Das University)
BBD City, Faizabad Road, Lucknow – 227105 (INDIA)

Consent Form (English)

Title of the Study ……..

Study Number……..
Subject's Full Name…….
Date of Birth/Age ……
Address of the Subject……..
Phone no. and e-mail address……
Qualification ……
Occupation: Student / Self Employed / Service / Housewife/ Other (Please tick as appropriate)
Annual income of the Subject……
Name and of the nominees(s) and his relation to the subject…… (For the purpose of compensation in case of trial related death).

1. I confirm that I have read and understood the Participant Information Document dated ……..for the above study and have had the opportunity to ask questions. **OR** I have been explained the nature of the study by the Investigator and had the opportunity to ask questions.
2. I understand that my participation in the study is voluntary and given with free will without any duress and that I am free to withdraw at any time, without giving any reason and without my medical care or legal rights being affected.
3. I understand that the sponsor of the project, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. However, I understand that my Identity will not be revealed in any information released to third parties or published.
4. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).
5. I permit the use of stored sample (tooth/tissue/blood) for future research. **Yes [ ]** **No [ ]**

6. I agree to participate in the above study. I have been explained about the complications and side effects, if any, and have fully understood them. I have also read and understood the participant/volunteer's Information document given to me.

Representative:……..
Signatory's Name……. Date …….
Signature of the Investigator…… Date……..
Study Investigator's Name..... Date……..
Signature of the witness…… Date……..
Name of the witness……
Received a signed copy of the PID and duly filled consent form
Signature/thumb impression of the subject or legally Date……..

Acceptable representative

ANNEXURE – IV

Babu Banarasi Das College of Dental Sciences
(Babu Banarasi Das University)
BBD City, Faizabad Road, Lucknow – 227105 (INDIA)

सहमति पत्र

अध्ययन शीर्षक.....
अध्ययन संख्या.....
प्रतिभागी के पूर्ण नाम.....
जन्म तिथि / आयु.....
प्रतिभागी का पता .....
फोन नं. और ई-मेल पता .....
योग्यता .....
व्यवसाय: छात्र / स्व कार्यरत / सेवा / ग्रहिणी .....
अन्य (उचित रुप मे टिक करें) .....
प्रतिभागी की वार्षिक आय .....
प्रत्याशीयो के नाम और प्रतिभागी से संबंध...(परीक्षण से संबंधित मौत के मामले मे मुआवजे के प्रयोजन के लिए)

1. मेरी पुष्टि है कि मैने अध्ययन हेतु सुचना पत्र दिनांक ..... को पढ व समझ लिया तथा मुझे प्रश्न पुछने या मुझे अध्ययन अन्वेषक ने सभी तथ्यों को समझा दिया है तथा मुझे प्रश्न पुछने के समान अवसर प्रदान किए गये।

2. मैंने यहॉ समझ लिया कि अध्ययन में मेरी भागीदारी पूर्णतः स्वैच्छिक है और किसी भी दबाव के बिना स्वतंत्र इच्छा के साथ दिया है किसी भी समय किसी भी कारण के बिना , मेरे इलाज या कानूनी अधिकारो को प्रभावित किए बिना , अध्ययन में भाग न लेने के लिए स्वतंत्र हुँ ।

3. मैंने यह समझ लिया है कि अध्ययन के प्रायोजक , प्रायोजक की तरफ से काम करने वाले लोग, आचार समिति और नियामक अधिकारियों को मेरे स्वास्थ्य रिकार्ड को वर्तमान अध्ययन या आगे के अध्ययन के सन्दर्भ देखने के लिए मेरी अनुमति की जरूरत नही है, चाहे मैने इस अध्ययन से नाम वापस ले लिया है। हॉलाकि मै यह समझता हुँ कि मेरी पहचान को किसी भी तीसरे पक्ष या प्रकाशित माध्यम में नही दी जायेगी।

4. मै इससे सहमत हूँ कि कोई भी डेटा या परिणाम जो इस अध्ययन से प्राप्त होता है उसका वैज्ञानिक उद्देश्य (ओं) के उपयोग के लिए मेरी तरफ से कोई प्रतिबंध नही है।
5. भविष्य के अनुसंधान के लिए भंडारित नमूना (ऊतक/रक्त) पर अध्ययन के लिए अपनी सहमति देता हुँ।

हॉ [ ] नही [ ] अनउपयुक्त [ ]

ANNEXURE – V

Observations

I. Non diabetic

Subjects	Age (yrs)	Sex	Lip Print Type	Finger print type	Overall DMFT Score	Total DMFT Score
1	33	F	Type II	Loop	1+0+0=1	1
2	44	F	Type II	Whorl	2+3+0=5	5
3	45	F	Type IV	Whorl	5+0+0=5	5
4	36	F	Type IV	Whorl	4+0+0=4	4
5	42	M	Type III	Loop	0	0
6	30	F	Type IV	Arch	4+0+0=4	4
7	65	F	Type II	Whorl	5+3+0=8	8
8	68	M	Type I	Loop	2+3+0=5	5
9	33	F	Type III	Whorl	2+1+0=3	3
10	38	F	Type IV	Loop	1+4+3=8	8
11	32	F	Type IV	Whorl	1+0+0=1	1
12	55	F	Type I	Loop	1+5+0=6	6
13	37	F	Type I	Loop	1+0+0=1	1
14	42	F	Type IV	Loop	6+4+0=10	10
15	50	F	Type III	Arch	4+20+0=24	24
16	42	F	Type I	Loop	0	0
17	32	F	Type II	Loop	4+0+0=4	4
18	78	M	Type II	Loop	0	0
19	38	F	Type III	Loop	2+0+0=2	2
20	35	M	Type IV	Whorl	1+0+3=4	4
21	38	M	Type III	Loop	1+2+5=8	8
22	31	M	Type IV	Loop	1+0+0=1	1
23	40	F	Type I	Loop	1+1+0=2	2
24	45	F	Type III	Loop	3+6+0=9	9
25	33	F	Type I	Arch	2+0+2=4	4
26	40	F	Type III	Loop	1+0+2=3	3

27	38	M	Type IV	Whorl	0+2+2=4	4
28	32	F	Type I	Loop	1+0+5=6	6
29	34	M	Type I	Loop	0	0
30	32	M	Type III	Loop	0+0+4=4	4
31	42	M	Type I	Loop	6+0+2=8	8
32	38	M	Type I	Loop	0	0
33	34	F	Type II	Whorl	0+0+3=3	3
34	32	F	Type III	Whorl	2+0+0=2	2
35	33	F	Type I	Whorl	0	0
36	31	F	Type I	Loop	0	0
37	35	F	Type I	Whorl	4+1+0=5	5
38	42	F	Type I	Loop	3+0+2=5	5
39	51	M	Type III	Whorl	3+1+0=4	4
40	31	F	Type III	Loop	0+0+1=1	1
41	33	F	Type I	Whorl	1+0+0=1	1
42	32	F	Type I	Loop	0	0
43	42	F	Type I	Whorl	0	0
44	37	F	Type II	Loop	2+0+0=2	2
45	42	F	Type I	Loop	2+1+2=5	5
46	36	M	Type I	Loop	1+0+0=1	1
47	33	F	Type III	Loop	2+0+2=4	4
48	38	M	Type II	Loop	1+0+0=1	1
49	48	F	Type III	Whorl	0+3+0=3	3
50	41	F	Type II	Loop	2+0+0=2	2

II. Diabetic

Subjects	Age (yrs)	Sex	Lip Print Type	Finger print type	Overall DMFT Score	Total DMFT Score
1	68	F	Type II	Loop	8+8+0=16	16
2	37	M	Type IV	Whorl	0	0
3	62	F	Type IV	Loop	4+2+0=6	6
4	58	M	Type IV	Whorl	0+2+0=2	2
5	70	M	Type IV	Loop	5+2+12=19	19
6	41	M	Type IV	Whorl	0	0
7	55	F	Type IV	Whorl	1+0+0=1	1
8	60	F	Type III	Loop	7+3+3=13	13
9	33	F	Type IV	Loop	3+0+0=3	3
10	45	M	Type IV	Whorl	2+0+0=2	2
11	45	F	Type IV	Loop	2+0+0=2	2
12	52	F	Type IV	Loop	0	0
13	66	M	Type IV	Loop	1+1+2=4	4
14	58	F	Type IV	Whorl	0+0+1=1	1
15	69	M	Type IV	Whorl	0+4+13=17	17
16	65	F	Type IV	Whorl	8+4+0=12	12
17	45	F	Type IV	Loop	1+2+0=3	3
18	41	F	Type II	Whorl	4+4+0=8	8
19	70	M	Type III	Loop	28	28
20	65	F	Type II	Loop	7+5+0=12	12
21	45	F	Type II	Loop	4+3+2=9	9
22	38	F	Type IV	Whorl	4+0+2=6	6
23	40	F	Type IV	Loop	2+0+0=2	2
24	32	F	Type IV	Whorl	5+0+0=5	5
25	42	F	Type IV	Loop	4+0+0=4	4
26	43	M	Type III	Arch	4+0+1=5	5
27	39	M	Type IV	Loop	2+0+4=6	6

28	53	F	Type III	Loop	6+2+0=8	8
29	56	F	Type III	Loop	2+0+3=5	5
30	68	F	Type II	Loop	8+0+3=11	11
31	56	M	Type II	Whorl	8+1+0=9	9
32	53	F	Type IV	Loop	6+2+0=8	8
33	49	M	Type IV	Loop	0	0
34	56	F	Type II	Loop	10+0+0=10	10
35	66	F	Type IV	Whorl	0+3+0=3	3
36	48	M	Type I	Whorl	0+0+3=3	3
37	59	F	Type IV	Loop	0+0+4=4	4
38	54	M	Type IV	Whorl	4+3+0=7	7
39	58	M	Type IV	Whorl	2+0+5=7	7
40	56	M	Type IV	Whorl	8+2+0=10	10
41	54	M	Type II	Loop	0+0+3=3	3
42	43	M	Type IV	Loop	3+2+0=5	5
43	49	M	Type II	Whorl	7+1+0=8	8
44	64	F	Type II	Loop	0	0
45	58	M	Type II	Arch	2+4+0=6	6
46	52	F	Type IV	Loop	6+0+0=6	6
47	60	F	Type II	Whorl	3+0+4=7	7
48	40	F	Type II	Loop	6+0+0=6	6
49	48	M	Type IV	Loop	6+3+10=19	19
50	65	M	Type III	Loop	6+4+0=10	10

ANNEXURE – VI

Formula used for the analysis

Arithmetic Mean

The most widely used measure of central tendency is arithmetic mean, usually referred to simply as the mean, calculated as

$$\bar{X} = \frac{\sum_{i=1}^{n} X_i}{n}$$

Standard deviation and standard error

The standard deviation (SD) is the positive square root of the variance, and calculated as

$$SD = \sqrt{\frac{\sum X_i^2 - \frac{(\sum X_i)^2}{n}}{n-1}}$$

and SE (standard error of the mean) is calculated as

$$SE = \frac{SD}{\sqrt{n}}$$

where, n= no. of observations

Minimum and Maximum

Minimum and maximum are the minimum and maximum values respectively in the measure data and range may be dented as below

Range = Min to Max

and also evaluated by subtracting minimum value from maximum value as below

Range = Maximum value-Minimum value

Median

The median is generally defined as the middle measurement in an ordered set of data. That is, there are just as many observations larger than the median as there are smaller. The median (M) of a sample of data may be found by first arranging the measurements in order of magnitude (preferably ascending). For even and odd number of measurements, the median is evaluated as

M= [(n+1)/2]th observation- odd number

M= [n(n+1)/2]th observation – even number

Student's t Test

Student's t-test was used to calculate the differences between the means of two groups

$$t = \frac{\bar{X}_1 - \bar{X}_2}{SE}$$

where,

$$SE = \sqrt{S^2 X \left(\frac{1}{n_1} + \frac{1}{n_2} \right)}$$

S^2 is the pooled variance and n_1 and n_2 are number of observations in group 1 and 2 respectively. The degrees of freedom (DF) is calculated as

DF = n1 + n2 – 2

Chi-square test

The chi-square (χ^2) test is used to compare the categorical data as

$$\chi^2 = \Sigma\Sigma \frac{(Fij - fij)^2}{fij}$$

where, F_{ij} is the observed frequency while f_{ij} the expected frequency. The degrees of freedom (DF) is calculated as

DF= (r-1) (c-1)

Analysis of Variance

Analysis of variance (ANOVA) is used when we compare more than two groups simultaneously. The purpose of one-way ANOVA is to find out whether data from several groups have a common mean. That is, to determine whether the groups are actually different in the measured characteristic. One way ANOVA is a simple special case of the linear model. For more than two independent groups, simple parametric ANOVA is used when variables under consideration follows Continuous exercise group distribution and groups variances are homogeneous otherwise non parametric alternative Kruskal-Wallis (H) ANOVA by ranks is used. The one way ANOVA form of the model is

$$Y_{ij} = \alpha_{.j} + \varepsilon_{ij}$$

where;

- Y_{ij} is a matrix of observations in which each column represents a different group.
- $\alpha_{.j}$ is a matrix whose columns are the group means (the "dot j" notation means that α applies to all rows of the j^{th} column i.e. the value α_{ij} is the same for all i).
- ε_{ij} is a matrix of random disturbances.

The model posits that the columns of Y are a constant plus a random disturbance. We want to know if the constants are all the same.

Tukey Test

After performing ANOVA, Tukey HSD (honestly significant difference) post hoc test is generally used to calculate differences between group means as

where,

$$q = \frac{\bar{X}_1 - \bar{X}_2}{SE}$$

$$SE = \sqrt{\frac{S^2}{2}\left(\frac{1}{n_1} + \frac{1}{n_2}\right)}$$

S^2 is the error mean square from the analysis of variance and n_1 and n_2 are number of data in group 1 and 2 respectively.

Level of significance "*P*" is the probability signifies level of significance. The mentioned *P* in the text indicates the following:

P > 0.05- Not significant (ns)

P < 0.05- Just significant (*)

P < 0.01- Moderate significant (**)

P < 0.001- Highly significant (***)

ANNEXURE – VII

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