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**كمساعدة لغرسات الأسنان الفورية الداعمة للأطقم الفوقية**  
**فى الفك السفلى لمرضى السكر**

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**Platelet-Rich Plasma as an Adjunct  
for Immediate Implants Supporting Mandibular  
Overdentures in Diabetic Patients**

**Thesis**

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Ten male diabetic patients having only mandibular canine or premolar on each side were selected. Immediate implants were inserted and platelet – rich plasma was introduced with only one implant. After three months the implants were loaded by over denture. It was found that bone density was increased around both implants and more increase was with platelet-rich plasma before loading. Bone height was decreased and the peri-implant probing depth was increased around both implants.

أختير عشرة مرضى البول السكري لديهم فقط ناب أوضاعك في كل ناحية من الفك السفلي وتم غرس الدعامتين الفورية في نفس المكان ووضعت البلازما الغنية بالصفائح قبل غرس واحدة فقط من الدعامتين مباشرة. ثم حُملت الأطقم بعد ثلاثة أشهر. وجد أن كثافة العظم تزداد حول الدعامتين وكانت الزيادة أكبر مع البلازما الغنية بالصفائح وذلك في فترة ما قبل التحميل. أما ارتفاع العظم نقص حول الدعامتين وكذلك زاد عمق الجسر بالمسبر حولهما.

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## Key words:

Platelet-rich plasma- immediate implants- overdentures- diabetic patients

## **REVIEW OF LITERATURE**

### **Diabetes Mellitus**

Diabetes mellitus (D.M) is metabolic disorder with wide variation in its prevalence rates among different populations and within the same population (**Traisman, 1980**).

It is a complex, multifactorial, genetically derived, endocrinal, chronic disease of metabolic deregulations' mainly of carbohydrate metabolism. It results from either insufficient insulin secretion from the  $\beta$  cells of the pancreas or from decreased tissue response to the circulating insulin (target-insulin resistance). This is attributed to weakness or decreased number of insulin receptors by (down regulation mechanism) or from both. It leads to increased blood glucose level and excretion of sugar in urine (hyperglycemia). Impairment in lipid and protein metabolism may also be encountered as a common characteristic in diabetes mellitus (**Siperstein, 1975, Salvi et al., 1997 and Nathan, 1993**).

The classic symptoms of the disease are polyurea, ketonurea, rapid weight loss and gross elevation of plasma glucose level above its renal threshold. Depending on the severity of metabolic abnormality, the disease may be asymptomatic or may progress to ketoacidosis and coma (**Albert and Zimmet, 1998 and Kuzuya et al., 2002**).

Retinopathy, nephropathy and micro-angiopathy, which produces thickening of capillary basement membranes in the blood vessels throughout the body are some of complications that may occur (**Kinane et al., 2001**).

Based on the stage, onset of the disease and degree of its severity, a variety of descriptive terms were formerly used to classify diabetes. Ideal, the classification of diabetes should be based on its etiology and pathogenesis (**Atkinson and Maclaren, 1990 and Brenner, 2003**).

The most common classification of diabetes mellitus was presented by the **Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, American Diabetes Association (A.D.A) (2000)** and by the **World Health Organization (W.H.O), Diabetes Mellitus (2002)**.

**The A.D.A. classification (2000):**

This classification was based on the pathophysiology of the disease rather than its treatment approaches.

- **Type 1:** formerly; insulin-dependant.
- **Type 2:** formerly; non-insulin-dependant.
- **Gestational (GDM):** is defined as any degree of glucose intolerance and was first recognized during pregnancy.
- **Other specific types :**
  - Genetic defect in  $\beta$  cell function.
  - Genetic defect in insulin action.
  - Pancreatic disease or injuries.
  - Endocrinopathies.
  - Drug or chemically induced.
  - Infections.
  - Part of other genetic syndromes.
  - Uncommon forms of immune mediated diabetes.

**The W.H.O. classification (2002):**

- **Type I:** Insulin dependant diabetes mellitus (IDDM).
- **Type II:** non insulin dependant diabetes mellitus (NIDDM).
- **Type III:** Gestational diabetes mellitus (GDM).

**Type I diabetes (IDDM) :**

It was formerly called juvenile-onset diabetes because it manifests in childhood and adolescence, yet it may occur at any age. This type is characterized by destruction of the  $\beta$  cell of pancreas which leads to absolute insulin deficiency and systemic ketosis or acidosis may occur. It constitutes 5-15% of diabetic patients. Its onset is abrupt and the course is unstable and is difficult to control (**Rees et al., 1992 and Smith, 1987**).

**IDDM is divided into:****a) Type I A/ immune mediated:**

It is more common than type I B. In this type, one or more immune response is in linkage with disequilibrium in human leucocytic antigen (HLA). This may increase the susceptibility to  $\beta$ -cell to be damaged by interaction of environmental factors with specific cell membrane antigen. (**Christopher et al., 1992**).

Acquired environmental factors such as viruses and chemicals may superimpose on genetic factors and lead to cell mediated destruction of  $\beta$ -cells (**Ronald and Weir, 1994**).

**b) Type I B/idiopathic:**

These patients usually have associated endocrinal disease such as Hashimoto's thyroiditis, Grave's disease, and primary gonadal failure. A non endocrinal disease may also be encountered such as pernicious anemia, celiac disease and myasthenia gravis (**Locatto et al., 1993**).

**Type II diabetes (NIDDM):**

It was formerly called maturity onset diabetes because it often occurs in midlife or later. It is a condition of impaired insulin function rather than insulin deficiency. It results from defects in the insulin