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Case Report

Long term clinical outcome of dental implants placed in a patient with aggressive periodontitis and Glucose-6-phosphate dehydrogenase (G6pd) deficiency

Reena Rodriguez[*], *Nico Hartmann*, *Dieter Weingart*

Department of Oral and Maxillofacial Surgery, Katharinen Hospital, Stuttgart, Germany

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ABSTRACT

Background and objectives: Aggressive Periodontitis is associated with higher risk of peri-implantitis and lower implant survival. Glucose-6-phosphate dehydrogenase (G6pd) deficiency is an enzymatic disorder which may produce hemolytic anemia in red blood cells. Since G6pd helps to maintain intracellular redox potential, a deficiency produces a weak interface at the marginal gingiva and additionally a reduced wound healing due to hemolysis. This case report discusses the clinical features and treatment protocol used to achieve osseointegration of dental implants in grafted and non grafted regions in a patient having aggressive periodontitis, congenital G6pd deficiency, refractory to conventional periodontal therapy.

Methods: Following extraction of the remaining teeth, 10 titanium implants (Straumann AG) were placed, starting with 4 implants in the mandible. After 5 years, augmentation of the extremely atrophic maxilla was performed. On one side the augmentation failed, however a total of 6 implants could be placed in the maxilla. The patient was followed up regularly for 15 years.

Results: The long term follow up (9 years in the maxilla and 15 years in the mandible) of the inserted implants showed 100% success rate.

Conclusion: Rehabilitation with dental implants in patients having aggressive periodontitis is a reasonable treatment option to restore maxillo-mandibular function and esthetics.

1. Introduction

Aggressive periodontitis is a rare but extremely progressive form of periodontitis that causes rapid destruction of the periodontal attachment apparatus and the supporting alveolar bone.[1,2] The disease distribution may be localized or generalized but two pertinent features of both forms are 1. Rapid attachment loss and bone destruction in an otherwise

* *Corresponding author.*
E-mail addresses: drreenajoseph@gmail.com, r.rodriguez@klinikum-stuttgart.de (R. Rodriguez).
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clinically healthy patient and 2. familial aggregation.[3] The primary etiological pathogens associated with aggressive periodontitis are Aggregatibacter actinomycetemcomitans and *Porphyromonas gingivalis*.[4,5] The majority of periodontal destruction however, is as a result of an inappropriate host response to the microorganism and their products.[6] The patients thus often present with limited microbial deposits that seem inconsistent with the severity of tissue destruction.

A systematic approach for the treatment of aggressive periodontitis is to arrest or slowdown the progression of the disease, preservation of the dentition in form and function, prevention of the disease recurrence and the regeneration of the periodontal apparatus. The restorative treatment of choice is often implant-supported prosthesis. Periodontal pathogens however may be transmitted from teeth to implants, implying that periodontal pockets may serve as reservoirs for bacterial colonization around implants.[7,8] The similarity in the microbial flora responsible for aggressive periodontitis and periimplantitis supports the view that periodontal pathogens may be associated with periimplant infections and failing implants.[9]

Glucose-6-phosphate dehydrogenase (G6pd) is the first and rate-limiting enzyme of the pentose phosphate pathway. It is indispensable to the maintenance of the cytosolic pool of NADPH and thus the cellular redox balance. The NADPH in turn maintains the supply of reduced glutathione in the cells that is used to counterbalance the free radicals that cause oxidative damage (Fig. 1). It causes disturbances in redox homeostasis and leads to dysregulation of cell growth and signaling and an altered susceptibility to infection making it an additional risk factor to the prognosis of periodontitis.[10]

G6pd deficiency is very frequent in Africa, Middle East, and Southeast Asia, but rare in Northern Europe (including Germany) or Northern America (except African Americans). Most of the G6pd deficient individuals are asymptomatic throughout their life. The illness generally manifests as acute hemolysis, which occurs when red blood cells undergo oxidative stress which may be by agents such as drugs, infection, or the ingestion of fava beans.[11] Glutathione is the primary antioxidant responsible for maintaining the reducing intracellular microenvironment that is essential for normal cellular function and viability.[12] Recent studies[13,14] report that G6pd had its maximum content in epithelium of marginal gingiva and lower contents in oral mucosa epithelium, crevicular epithelium and epithelial attachment. G6pd has an increased concentration from the basal to the superficial layers in attached and marginal gingival. It remains stable in all strata of oral mucosal epithelium and crevicular epithelium with a reduced activity towards the surface of the epithelial attachment. It is reasonable to speculate the G6pd deficiency produces a weak interface at marginal gingiva in addition to a reduced wound healing capacity due to hemolysis thus contributing to a more challenging clinical situation.

There have been no studies to date addressing the short term or long term success rate of dental implants placed in a G6pd deficient patient. The purpose of this case report is to present the long term clinical outcome of implants placed in a patient with generalised aggressive periodontitis and a known history of G6pd deficiency, which was refractory to conventional periodontal therapy.

2. Clinical report

A 23 year old male patient with generalised aggressive periodontitis associated with a known history of Glucose-6-phosphate dehydrogenase deficiency (G6pd) presented to the Department of Oral and Maxillofacial surgery, Katharinen Hospital, Stuttgart, Germany in October 1997. Prior to 1997, the patient underwent systematic periodontal therapy for the management of periodontitis in different university hospitals and private dental offices since 1988. The microbiological culture revealed a positive culture for actinomycetes comitants. Deep scaling and curettage and composite splinting to stabilize the teeth was done. Flap surgeries and the use of membranes for guided tissue regeneration had also been attempted. Tunnel technique flap surgery and periodontal plastic surgery was done in relation to teeth #24 and #26. However in spite of all these therapies the progression of the disease could not be reversed. The patient was then referred to the authors for a further surgical regenerative phase consisting of bone augmentation and the placement of dental implants. The disease progression had led to extensive generalised horizontal bone loss and intra bony defects in the premolar and molar areas. Generalized bone loss and loss of attachment was sustained in all teeth and this was clinically

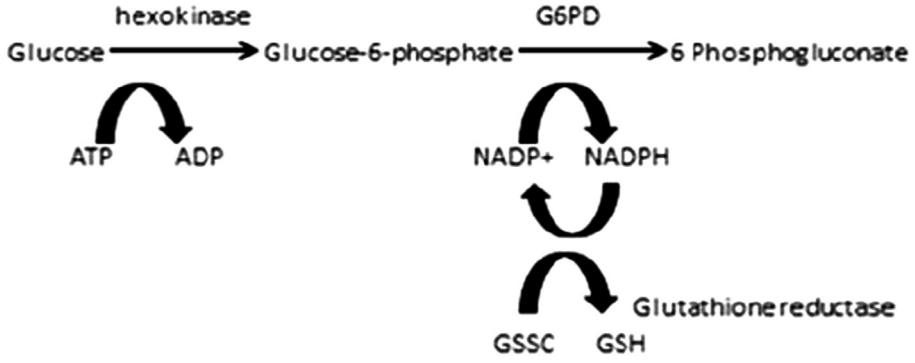

Fig. 1 – G6PD catalyzes NADP+ to its reduced form: NADPH, in the pentose phosphate pathway. (G6PD = glucose-6-phosphate dehydrogenase; ATP = adenosine triphosphate; ADP = adenosine diphosphate; NADP+ = nicotinamide adenine dinucleotide phosphate [oxidized form]; NADPH = reduced NADP; GSSG = oxidized glutathione; GSH = reduced glutathione.