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diabetic ketoacidosis and tooth Loss (Case Study)

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1.0 Description

A 47-year-old man was presented to our hospital with nausea , vomting ,malaise , generalized weakness , fatigue and acute abdominal pain . past hx: polyuria , polydipsia and weight loss for two month before .-ev S&S no fever, no diarrhea normal pulse . Physical examination : cvs: s1 s2 + 0, chest: clear no added sound , abdomen: diffuse abdominal pain and tender abdomen , extremities: no lower limb edema , ENT: normal ,orofacial : edema, erythema ,

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tenderness, halitosis . Hx of bleeding during. Brush and tooth loss(E. & N. 2011).

2.0 Laboratory

2.1 fasting plasma glucose

A- 389 mg/dL

B- Metabolic acidosis with an elevated anion gap

C- HbA1c, 10.8%

2.2 serum ketone levels

4.9 mmol/L

2.3 CRP

0.88 mg/dL

3.0 Treatment

Treated with IV NS and regular insulin injections was immediately initiated, the symptoms disappeared within few days as DKA resolved. The patient received follow-up treatment with oral metformin and non-surgical odontotherapy with personal oral hygiene. Haematological findings revealed the improvement of glycaemic control and systemic inflammation at two months (HbA1c, 7.7% and CRP, 0.22 mg/dL) and 4 months (HbA1c, 6.4% and CRP, 0.09 mg/dL) after treatment. The patient maintained appropriate glycaemic control with normal HbA1c levels for the succeeding year (HbA1c, 6.3% and 6% at 7 and 11 months after treatment initiation, respectively(B. et al. 2005).

Periodontitis is a chronic inflammatory disorder by putative pathogens such as Porphyromonas gingivalis and Tannerella forsythia.4–6 In comparison with monocytes from non-diabetic individuals, those from patients with DM when challenged with lipopolysaccharides derived from the outer membrane

component of these pathogens produce significantly greater concentrations of tumour necrosis factor-α, interleukin-1β and prostaglandin E2 and decreased expression of receptor activator of NF-κB ligand in the nearby alveolar bone.4–6 These are implicated in the worsening of periodontitis and a net increase in tooth loss in patients with DM. The patient was not aware that he had DM until the first visit to our department. Although the evidence for periodontal disease as a predictor of incident DM is conflicting, some study had offered a clinical approach that can be easily used in dental care settings.7 Their simple algorithm comprising only two dental parameters—the number of missing teeth (≥4 missing teeth) and percentage of deep periodontal pockets (≥26% deep pockets)—was effective in identifying 73% of patients with unrecognised pre-diabetes or DM. Approximately 35% of adults in Japan visit a dentist at least once a year and >90% of individuals with periodontitis may be candidates for diabetes screening, according to the guidelines set by the American Diabetes Association. The patient underwent odontotherapy at a dental clinic more than a few years before the hospitalisation. Although it remains unclear whether the patient had been suffering from severe periodontal disease and had several missing teeth, his oral symptoms may be indicative of the presence of glucose intolerance. The serum CRP levels of the patient gradually decreased in parallel with the improvement in his oral hygiene confirmed using serial gross findings and the information provided by the patient, which was likely to precipitate proper glycaemic control by eliminating insulin resistance(E. et al. 2006).

The tooth lost during DKA was representative of the bidirectional relationship between diabetes and periodontal disease. The two diseases are interrelated and may amplify one another. This case shows the importance of substantial examination for oral health that may be associated with unrecognised glucose intolerance.

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