and often delayed. Despite their size and invasiveness, macroprolactinomas in postmenopausal women generally respond well to treatment with dopamine agonists.

EP_A134

CHARCOT ARTHROPATHY IN A CONTROLLED DIABETIC PATIENT: A CASE REPORT

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INTRODUCTION/BACKGROUND

Charcot arthropathy is a severe complication of diabetes which is often diagnosed late, characterized by a red, warm, and swollen foot with bone abnormalities on imaging. Most studies report elevated HbA1c as a risk factor in Charcot patients, but there are rare cases with normal HbA1c. If not promptly diagnosed and treated, the condition can lead to deformity, foot ulcers, amputation, and death.

CASE

A 54-year-old male came to M Djamil General Hospital with complaints of ulcers around the right ankle. The patient has a history of diabetes mellitus (13 years). We found deformity with ulcers and pus in the right ankle joint. We did several examinations to confirm the diagnosis. The laboratory results are random blood glucose 152 mg/dL; fasting blood glucose 65 mg/dL; two-hours postprandial glucose 111 mg/dL; HbA1c 7.0%. CT scan of the lower extremities found osteomyelitis of the tarsal bones with cellulitis; histopathology found chronic and acute inflammation with granulation tissue. The working diagnosis was Charcot arthropathy of the right distal tibia Brodsky Type 3A, and Type 2 Diabetes Mellitus. We performed immobilization, external fixation, sequestrectomy and boot casting and controlled glycemia with medical nutrition therapy and rapid acting insulin for perioperative management. We used antibiotics and analgesics to treat infection and pain. The results were good and the patient was advised to use ankle foot orthosis.

CONCLUSION

This is a rare case report of Charcot arthropathy in a patient with normal HbA1c. This condition may be associated with rapid HbA1c normalization, which can trigger acute episodes, and the duration of diabetes. Clinicians should assess glycemic history and neuropathic risk factors. Target HbA1c between 7.0 – 8.0% during treatment can facilitate wound healing without increasing mortality.

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SEVERE HYPOTHYROIDISM-INDUCED RHABDOMYOLYSIS IN THE ABSENCE OF A TRIGGERING FACTOR

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INTRODUCTION/BACKGROUND

Thyroid disorders are among the most common endocrine diseases globally, with hypothyroidism affecting approximately 3.4% of the Malaysian population. Muscle-related symptoms, such as fatigue, cramps, and myalgia are frequently observed in hypothyroidism and usually present with mild to moderate elevations of the muscle enzymes. However, rhabdomyolysis due to hypothyroidism, particularly in the absence of other apparent causes, is rare and is more frequently associated with Hashimoto's thyroiditis. The exact mechanism remains unclear, but it is hypothesized that hypothyroidism disrupts muscle metabolism, leading to prolonged oxidative damage and subsequently rhabdomyolysis.

CASE

We report a case of a 32-year-old male with no prior medical history who presented with one month of weight gain and lethargy, associated with facial puffiness for 2 weeks. He denied systemic symptoms, strenuous activity, trauma, alcohol use, or recent medications. No family history of thyroid or autoimmune disease was noted. Examination showed mild facial puffiness, no muscle weakness, and normal reflexes. Laboratory investigations revealed elevated creatinine kinase (CK) levels of 2,527 U/L (55-170), aspartate transaminase (AST) of 130.3 U/L (8-33), alanine transaminase (ALT) of 118.1 U/L (7-56) and acute kidney injury with urea 7.3 mmol/L (7-12), creatinine 182 μmol/L,