Adult E-Poster

Upon diagnosis of osteoporosis, warfarin was replaced with rivaroxaban for anticoagulation and vitamin D replacement and calcium supplements were started, while no anti-osteoporosis medications were initiated. Annual BMD was done, and the latest imaging showed an improvement of 2.4% in the femoral neck compared to the previous year. Apart from the previously noted vertebral compression fractures, no new fractures were appreciated during follow-up. BMD monitoring will continue every 2 years.

CONCLUSION

Osteoporosis in the young should be thoroughly investigated and managing the underlying condition is key to proper treatment.

EP_A103

LIPOPROTEIN X-MEDIATED PSEUDOHYPONATREMIA IN A PATIENT WITH TYPE 2 DIABETES

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INTRODUCTION/BACKGROUND

Pseudohyponatremia is a lab abnormality commonly caused by hypertriglyceridemia, hyperglycemia or hypergammaglobulinemia. Lipoprotein X (LpX) is an abnormal lipoprotein that most commonly appears in the plasma of patients with cholestasis. LpX mediated pseudohyponatremia is rare but has been described in the literature. We report a patient with type 2 diabetes mellitus (T2DM) and LpX-mediated pseudohyponatremia due to severe cholestatic hepatitis.

CASE

A 31-year-old female was admitted with newly diagnosed T2DM and severe DKA secondary to bilateral calf abscesses. She was treated with insulin and intravenous cefazolin as intraoperative tissue culture grew MSSA. Three days after starting cefazolin she developed progressively worsening severe cholestasis [peak total bilirubin (TB) 245 umol/L(reference interval (RI) <17), conjugated bilirubin 175 umol/L (RI <6), peak ALP 1027 U/L (RI 45-129), with normal to marginally elevated transaminases] with negative viral and autoimmune serologies including AMA. Malignancy, biliary stones, and extra-hepatic cholestasis were excluded by imaging including CECT liver. Liver biopsy showed non-caseating granulomatous hepatitis, consistent with drug-induced liver injury secondary to cefazolin.

Concurrently, she developed hyponatremia despite adequate glycemic control on insulin therapy, that was established to be secondary to severe hypercholesterolemia [nadir serum sodium (sNa) 125 mmol/L (RI 136-145), serum osmolality 308 mmol/kg (RI 275-295), total cholesterol (TC) 30.6 mmol/L (RI <5.2), triglyceride 5.3 mmol/L]. Serum protein electrophoresis showed a supernumerary peak between albumin and alpha-1 region, suggestive of the presence of LpX. Cefazolin was discontinued and she was given a course of ursodeoxycholic acid (UDCA) for three months. Subsequently, TB and ALP dramatically improved, TC gradually declined and serum sodium became normal. During her most recent follow-up, her liver panel and serum sodium remained normal. TC, triglyceride, and LDL, while markedly improved, remained slightly elevated, compatible with her diagnosis of metabolic syndrome.

CONCLUSION

Recognition of the relationship of cholestasis, elevated LpX and pseudohyponatremia is important to avoid mismanagement of hyponatremia. Electrophresis confirms the diagnosis of LpX and diagnosed patients should subsequently be monitored for hyperviscosity secondary to hypercholesterolemia.

EP_A104

A CASE OF LATE-ONSET HYPOPARATHYROIDISM FOLLOWING RECURRENT ANTERIOR NECK SURGERY RESULTING IN RHABDOMYOLYSIS

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INTRODUCTION/BACKGROUND

Hypoparathyroidism is a known complication of anterior neck surgery, with 1.5% becoming permanent. Delayed-onset hypoparathyroidism can manifest years postoperatively due to progressive scar tissue formation. It is often overlooked, causing complications. We present such a patient complicated by rhabdomyolysis and renal failure.