Adult E-Poster

EP_A115

PARANEOPLASTIC HYPOGLYCEMIA IN HEPATOCELLULAR CARCINOMA: A REPORT OF TWO CASES

https://doi.org/10.15605/jafes.040.S1.123

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INTRODUCTION/BACKGROUND

Hepatocellular carcinoma (HCC) is the second most common cause of non-islet cell tumor hypoglycemia (NICTH), with a reported prevalence varying from 4% to 27%. It can also be an early presentation of HCC, even before the tumour is large enough to be detected on imaging. We report two cases of NICTH in patients with viral hepatitis-associated HCC.

CASE

Case 1: A 64-year-old male presented with severe hypoglycemia (glucose 1.8 mmol/L) during an admission for spontaneous bacterial peritonitis. He was newly diagnosed with Child-Pugh C liver disease from chronic hepatitis C. Hypoglycemia persisted despite resolution of sepsis, requiring continuous dextrose 50% infusion. Physical examination revealed a hard mass in the right upper quadrant. Laboratory findings included elevated AFP at >830 IU/mL, low albumin at 24 g/L, INR of 1.5, bilirubin of 25 μmol/L, and platelet count of 180 x10^9/L. CT imaging showed a cirrhotic liver with a segment II mass (4.2 x 4.0 x 4.0 cm). Laboratories done during hypoglycemia showed low levels of insulin at <0.5 mIU/L (normal 3–25), low C-peptide at 46 pmol/L (298–2350), low IGF-1 at 10.5 ng/mL (42–179), and HbA1c of <3.4% (Hb 9.6 g/dL). IGF-2 testing was unavailable.

Case 2: A 48-year-old female with chronic hepatitis B and a large HCC (14 x 10 x 9 cm) and elevated AFP at 8,925 IU/mL. She presented with symptomatic hypo-glycemia (glucose 2.5 mmol/L). Prolonged fasting revealed hypoglycemia at 4 hours (glucose 2.1 mmol/L), with insulin of <2 μIU/mL, C-peptide of <33 pmol/L, IGF-1 of 17 ng/mL (normal 90–249), and cortisol at 509 nmol/L. The presence of inappropriately low insulin and low c-peptide during an episode of hypoglycemia favors the diagnosis of NICTH.

Both cases were managed with oral prednisolone 1 mg/kg which allowed for successful weaning from dextrose. Despite transient stabilization, both patients succumbed within one month.

CONCLUSION

Paraneoplastic hypoglycemia should be considered in HCC patients with unexplained hypoglycemia. Early recognition and glucocorticoid therapy may improve short-term outcomes, though prognosis remains poor.

EP_A116

PSEUDOACROMEGALY: A CASE OF SEVERE INSULIN RESISTANCE WITH ACROMEGALIC FEATURES AND LOW IGF-1

https://doi.org/10.15605/jafes.040.S1.124

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INTRODUCTION/BACKGROUND

Severe insulin resistance (SIR) is typically associated with well-established metabolic conditions such as central obesity, glucose intolerance, hypertension, dyslipidaemia and metabolic dysfunction-associated fatty liver disease (MAFLD). We present a case of SIR with prominent dermatologic and acral manifestations mimicking acromegaly, highlighting the importance of recognizing skin and soft tissue changes as possible indicators of underlying insulin resistance.

CASE

A 35-year-old male with a history of obesity (weight 128.7 kg, height 1.71 m, BMI 44 kg/m^2), hypertension (on amlodipine) and hyperlipidaemia (on atorvastatin) was referred for evaluation of hypokalemia (2.35 mmol/L). Clinical examination revealed acromegalic features, including macroglossia, rhinophyma, spade-like hands and marked dermatologic findings with acanthosis nigricans, pachyderma, multiple epidermoid cysts and dense axillary skin tags. Visual fields were normal. Anterior pituitary function was normal except for low IGF-1 at 78 ng/mL (normal: 88.3–246 ng/mL). Liver function tests revealed mild abnormalities of bilirubin (51 μmol/L), aspartate aminotransferase 71 U/L (normal: <32 U/L), alanine aminotransferase 23 U/L (normal: <33 U/L) and alkaline phosphatase 136 U/L (normal: 40–129 U/L). The patient denied alcohol use. Fasting insulin was 647 pmol/L (normal: 17.8-173 pmol/L). A diagnosis of pseudoacromegaly secondary to SIR was considered, supported by a significantly elevated HOMA-IR of 27.3 (normal: 0.5-1.4). HbA1c was 8.5%. He was initiated on metformin and lifestyle modification.