CASE

We report a case of a 14-year-old male with no known premorbid conditions who presented with presyncope and a 1-month history of headache. On examination, he had severe hypertension (242/167 mmHg), tachycardia (127 bpm), and grade IV hypertensive retinopathy. Investigations showed preserved renal function with markedly elevated 24-hour urinary metanephrines (normetanephrine 90.75 µmol/L). Computed tomography revealed a lobulated, heterogeneously enhancing mass measuring 5.0 × 6.1 × 5.4 cm along the left margin of the abdominal aorta at the infrarenal level, suggestive of an extra-adrenal lesion. Gallium-68 PET scan demonstrated a somatostatin receptor–avid left peritoneal mass. The patient underwent exploratory laparotomy and tumor excision, complicated intraoperatively by blood pressure lability requiring nitroprusside and inotropic support. Postoperatively, he improved significantly and was able to wean off all antihypertensive medications. Histopathology confirmed left retroperitoneal paraganglioma.

CONCLUSION

Primary peritoneal paraganglioma is a rare but important cause of secondary hypertension, especially in young patients presenting with hypertensive emergency. High index of suspicion is essential for early diagnosis. Management requires a multidisciplinary approach with careful preoperative optimization to minimize perioperative complications. Surgical resection remains the definitive treatment and, as demonstrated in this case, can result in marked clinical improvement with resolution of hypertension.

EP_A087

WARBURG EFFECT–ASSOCIATED NON–INSULIN-MEDIATED HYPOGLYCEMIA IN CHRONIC INFECTION

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INTRODUCTION

The Warburg effect refers to a metabolic shift in which cells preferentially utilize aerobic glycolysis rather than oxidative phosphorylation despite adequate oxygen availability. This phenomenon is increasingly recognized as the mechanism for tumor-associated hypoglycemia, or paraneoplastic syndromes such as insulin-like growth factor-2 (IGF-2)–mediated non-islet cell tumor hypoglycemia. We present a case of Warburg effect with management challenges.

CASE

A 47-year-old male with advanced, treatment-naive retroviral disease presented with chronic cough, progressive right thigh swelling, constitutional symptoms over several months, followed by reduced responsiveness for 2 days prior to admission. Clinically, he was delirious, cachectic with generalized lung crepitations and right thigh mass. He was diagnosed with smear-negative disseminated tuberculosis, and thigh mass was considered either a tuberculous granuloma or sarcoma.

During hospitalization, he developed recurrent hypoglycemia with lactatemia despite intravenous dextrose, optimized enteral feeding and a 1-day course of intravenous octreotide. His blood investigations showed full blood count, renal function test, liver function test, and serum ketone within normal range. His arterial blood gas analysis showed type B lactic acidosis with persistent lactatemia.

At the time of hypoglycemia (RBS: 3.1 mmol/L), his serum insulin was suppressed, C-peptide was low–normal, morning cortisol was elevated, and IGF-1 was markedly reduced suggestive of non-insulin-mediated hypoglycemia secondary to Warburg effect.

CONCLUSION

In patients with retroviral disease and tuberculosis, Warburg effect may lead to poorer clinical outcomes and management may be challenging. Thus, definitive antimicrobial therapy with second generation somatostatin analogues (Pasireotide) may be considered in selected cases to modulate dysregulated hormonal and metabolic pathways. Early identification and timely targeted intervention are crucial to improving outcomes in this high-risk population.

EP_A088

WHEN HYPOGLYCEMIA SPEAKS LOUDER THAN THE CHEST: A DECADE-LATE RECURRENCE OF IGF-2-MEDIATED NON-ISLET CELL TUMOR HYPOGLYCEMIA

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INTRODUCTION

Non-islet cell tumor hypoglycemia (NICTH) is a rare paraneoplastic syndrome caused by tumor secretion of insulin-like growth factor-2 (IGF-2), leading to recurrent hypoinsulinemic hypoglycemia. It is most commonly associated with large mesenchymal tumors, such as solitary

fibrous tumor, particularly those arising from the pleura or lungs. This phenomenon, also known as Doege–Potter syndrome, may precede tumor detection or signal tumor recurrence. We report a striking case of late malignant recurrence presenting solely with hypoglycemia after a decade of remission.

CASE

A 68-year-old female was initially presented in 2016 with respiratory symptoms and recurrent symptomatic fasting and post-prandial hypoglycemia, and was found to have a large left upper lobe mass. Tumor resection in 2017 resulted in complete resolution of hypoglycemia. She remained asymptomatic for several years. In 2023, she developed recurrent hypoglycemia without respiratory or constitutional symptoms. Biochemical evaluation demonstrated hypoinsulinemic hypoglycemia with suppressed insulin and C-peptide, low IGF-1, and markedly elevated IGF-2, resulting in an IGF-2:IGF-1 ratio of 25, consistent with IGF-2-mediated NICTH. Computed tomography imaging revealed a large left thoracic mass with invasion into the intercostal muscles and diaphragm with extension toward the stomach, associated with contralateral lung nodules and possible liver metastases, suggesting recurrent malignant disease. Hypoglycemia improved with glucocorticoid therapy, and the patient was referred for oncological assessment. Despite initiation of systemic chemotherapy, her disease progressed and she succumbed during treatment.

CONCLUSION

This case highlights several important lessons: Firstly, recurrent hypoinsulinemic hypoglycemia warrants evaluation for NICTH even in the absence of tumor-related symptoms. Secondly, solitary fibrous tumors may recur or undergo malignant transformation after prolonged disease-free intervals; and glucocorticoids provide effective metabolic control but do not alter oncologic prognosis. Long-term surveillance should be considered in patients with prior solitary fibrous tumors due to the risk of delayed recurrence and paraneoplastic complications.

EP_A089

MATERNAL HYPOGLYCEMIA WITH LARGE UTERINE FIBROID AND PARADOXICAL FETAL OVERGROWTH: PLAUSIBLE MECHANISMS

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INTRODUCTION

Uterine fibroids are common in reproductive-age women and are associated with obstetric complications. Their potential contribution to maternal metabolic disturbances is poorly described. We report a case of maternal hypoglycemia in the setting of a large uterine fibroid and fetal overgrowth, suggesting a possible endocrine–metabolic interaction.

CASE

A 39-year-old multiparous female with no history of diabetes or endocrine disease had a large lower-segment uterine fibroid measuring 8 × 10 cm. Pregnancy was otherwise uncomplicated until 39 weeks, when she underwent classical Caesarean section for transverse lie and delivered a large-for-gestational-age infant.

Postpartum, she developed recurrent symptomatic hypoglycemia despite normal hemoglobin A1c (5.0%) and preserved thyroid and adrenal function. There was no evidence of sepsis, liver disease, medication exposure, or insulinoma. Hypoglycemia resolved spontaneously following delivery. Postnatal imaging showed persistence of the fibroid (6 × 9 cm). She remains under follow-up, considering interval laparoscopic myomectomy with bilateral tubal ligation.

The coexistence of maternal hypoglycemia and fetal overgrowth raises the possibility of altered maternal–fetal glucose dynamics. Large mesenchymal tumors may produce insulin-like growth factor 2 (IGF-2), causing non–islet cell tumor hypoglycemia. Fibroid-related uteroplacental hemodynamic changes or increased fetal glucose demand may also contribute. While causality cannot be established, the temporal resolution post-delivery suggests a contributory role of the fibroid in metabolic disturbance.

CONCLUSION

This case highlights a rare but clinically relevant association between a large uterine fibroid, maternal hypoglycemia, and fetal overgrowth. In pregnant patients with unexplained hypoglycemia, uterine fibroids may represent an overlooked factor. Multidisciplinary follow-up and further research into IGF signaling and placental–tumor interactions are warranted.