



She was started on diazoxide for endogenous hyperinsulinemic hypoglycaemia with dose titrated up to 400 mg/day. One week after diazoxide initiation, she developed symptoms of diazoxide overdose, fever and thrombocytopenia (nadir level $10 \times 10^9/L$) with no source of infection. Diazoxide was stopped and she was switched to subcutaneous octreotide 100 mg TDS. Symptoms of overdosage, fever, and thrombocytopenia resolved after 4 days of stopping diazoxide. She then underwent distal pancreatectomy which was curative.

Thrombocytopenia is a rare complication of diazoxide and occurs due to platelet destruction from antibody formation. It is dose dependent and occurs 13-23 days after initiation of diazoxide and resolution will occur after discontinuing diazoxide.

CONCLUSIONS

Although thrombocytopenia is a rare complication of diazoxide, close monitoring of platelet count is needed to prevent complications.

PP-PN-08

DELAY IN GROWTH HORMONE THERAPY IS NOT DETRIMENTAL

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BACKGROUND

Pituitary hypothyroidism with growth hormone deficiency is often missed in primary care due to scarce health resources in developing countries. Well-meaning primary care with thyroxine for misdiagnosed primary hypothyroidism may be detrimental. However, the initial use of thyroxine in neonatal central hypothyroidism and growth hormone deficiency prevented long term intellectual decline even when it was inappropriately diagnosed as primary hypothyroidism.

CASE

Twin male siblings born to second degree consanguineous parents were managed by a pediatric specialist as primary hypothyroidism, requiring unusually high thyroxine doses. T4 was used to guide therapy. After consultation with an endocrinologist, the associated growth hormone deficiency was diagnosed. Pituitary structures on magnetic resonance imaging were normal.

The dose of thyroxine was beyond age-matched norms, with free T4 and free T3 values significantly high at 4 years of age. Confirmation of growth hormone deficiency was guided by hormone assay and auxology. Assistance from a growth hormone manufacturing company and government support helped manage both siblings from age 4 up to 18 years, notwithstanding limitations in periodic monitoring. The siblings are now in graduate technical college with respectable height gain.

CONCLUSION

Appropriate and timely diagnosis of pituitary hypothyroidism and associated growth hormone replacement, even in poor socioeconomic situations, can help nurture productive citizens.

PP-PN-09

AUTOIMMUNE POLYGLANDULAR SYNDROME (APS) TYPE 2 WITH CENTRAL DIABETES INSIPIDUS

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BACKGROUND

APS involves functional abnormalities in several endocrine and non-endocrine glands. Deficits may manifest over time. Screening for other manifestations is important. The major components of the syndrome are adrenal insufficiency, thyroid autoimmunity and type 1 diabetes. It is very rarely described with central diabetes insipidus (DI).

We present a very uncommon presentation of APS-2 with the onset of Addison's at age 7, developing other polyglandular associations over time and central DI in adulthood.

CASE

A 31-year-old woman with a history of Addison's disease diagnosed and treated with dexamethasone at age 7 presented with acute polyuria and polydipsia. She had negative water deprivation test according to the North Bristol protocol. Due to persistent symptoms, she was empirically commenced on desmopressin (Minirin) with good response. Testing finally diagnosed central DI: magnetic resonance imaging (MRI) demonstrated absence of the posterior pituitary bright spot and a 3 mm stalk thickening thought to be pathognomonic and possibly related to arginine vasopressin antibody status. Antibodies to thyroglobulin, thyroid receptor, ZnT8, GAD and IA2 were negative. TSH and HbA1c were normal.



CONCLUSION

APS-2 as an evolving polyglandular disease may be associated with other endocrine deficiencies, including central DI. Loss of pituitary bright spot and stalk thickening on MRI supports the diagnosis of central DI and should be considered in the work up of patients.

PP-PN-10

A CASE REPORT ON PITUITARY APOPLEXY FOLLOWING ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY AND LAPAROSCOPIC CHOLECYSTECTOMY

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BACKGROUND

Pituitary apoplexy is a rare neurosurgical emergency increasingly being precipitated by minor surgical procedures.

CASE

A 49-year-old male underwent ERCP and laparoscopic cholecystectomy for acute cholangitis. Two days postoperatively, he complained of dizziness with horizontal diplopia and slight left eye ptosis. Five days postoperatively, he became drowsy, with complete ptosis and blurred vision of the left eye and decreased motor strength on all extremities. Cranial CT Scan showed a sellar/suprasellar ovoid soft tissue focus. Pituitary MRI confirmed a sellar/suprasellar heterogeneous mass measuring 1.5 cm x 2.5 cm x 1.8 cm, hyperintense on T1 and hypointense on T2. Hormonal workup showed low IGF-1 [63.50 ng/mL, reference value (RV) 74-196], GH (0.60 ng/mL, RV 0-0.97), LH (0.16 mIU/mL, RV 1.5-9.3), total testosterone (<10 pg/mL, RV 164-753), ACTH (<5 pg/mL, RV 5-46), 0800H cortisol (<1.00 µg/dL, RV 3.7-19.4), PRL (1.11 ng/mL, RV 2.1-17.7), TSH (0.23 uIU/mL, 0.55-4.78) and FT3 (2.23 pg/mL, RV 2.3-4.2). He was managed as panhypopituitarism (secondary adrenal insufficiency, hypothyroidism, hypogonadism) secondary to pituitary macroadenoma with pituitary apoplexy. He was given hydrocortisone and underwent endoscopic transsphenoidal pituitary mass excision. He was discharged improved on prednisone 7.5 mg/day and levothyroxine 100 µg/day.

CONCLUSION

Pituitary apoplexy should be considered in patients with abrupt neuro-ophthalmological deterioration even after minor gastrointestinal surgeries. Early diagnosis allows immediate intervention to preserve vision and provide hormonal replacement.

PP-PN-11

A CONCURRENT FINDING OF A GROWTH HORMONE-PRODUCING PITUITARY ADENOMA AND A RADIOLOGICALLY-CONFIRMED SYMPTOMATIC RATHKE'S CLEFT CYST

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BACKGROUND

We present a case report describing a rare finding of concurrent growth hormone (GH)-producing pituitary adenoma and a radiologically confirmed symptomatic Rathke's cleft cyst (RCC) in a 65-year-old female patient.

CASE

Hormonal studies showed elevated insulin-like growth factor (IGF) [43.3 nmol/L, reference range (RV) 6.2-24 nmol/L and 51.3 nmol/L] taken two months apart. Other assays were normal (PRL 213 mIU/L, RV <700; 1000H cortisol 222 nmol/L, RV 14-690; TSH 1.3 mIU/L, RV 0.27-4.2; FT4 12 pmol/L, RV 12-22 pmol/L). OGTT revealed a failure to suppress serum GH to <1 µg/L, with nadir GH 2.3 µg/L. Pituitary meatus magnetic resonance imaging scan showed a 6.5 mm x 9 mm non-enhancing cyst in the pituitary sella which appeared to be displacing the normal pituitary tissue superiorly and slightly posteriorly. The optic chiasm was preserved, with no supra- or parasellar extension. After transphenoidal surgery, histopathologic studies revealed a strongly GH-positive adenoma, also positive for PIT1, SF1; and Ki67 1-2%. PRL, FSH and LH staining were negative.

CONCLUSION

Clinicians are reminded about increasing evidence of the concurrent occurrence of symptomatic RCC(s) and pituitary adenoma(ta). More explanations beyond case reports or case series evidence are needed to explain their seeming concurrence.