

CONCLUSION

Clinicians should have a high index of suspicion for PHPT in pregnancy and manage the condition with a multidisciplinary team (consisting of endocrinologist, endocrine surgeon, obstetrician, paediatrician and anaesthesiologist) due to its potential serious maternal and foetal adverse outcomes if left untreated.

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A CHALLENGING CASE OF PARATHYROID CARCINOMA WITH SEVERE HYPERCALCEMIA

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INTRODUCTION

Parathyroid carcinoma is a rare condition and it accounts for < 1% of cases of sporadic primary hyperparathyroidism (PHPT). Parathyroid carcinoma is commonly associated with severe and refractory hypercalcemia. We described a patient with parathyroid carcinoma who presented with multiple pathological fractures.

RESULTS

A young female presented with closed fractures of the right proximal humerus and bilateral femoral necks after a trivial fall. Her initial serum calcium was 3.18 mmol/L, serum phosphate was 0.56 mmol/L. The diagnosis of primary hyperparathyroidism was confirmed with high serum parathyroid hormone (iPTH) level of 1187.2 pg/mL (14.9-56.9). Neck ultrasound showed a right parathyroid lobulated lesion measuring 1.6 x 2.3 x 3.3 cm. The challenge in management was the refractory severe hypercalcemia despite standard treatment of hydration, bisphosphonate and calcitonin. Her serum calcium still ranged between 3.6-4.5 mmol/L despite the above therapy. She developed ECG changes typical for hypercalcemia (short QT interval). The surgery cannot proceed due to severe hypercalcemia. Hence, the decision for hemodialysis was made. She underwent 2 sessions of hemodialysis with low calcium dialysate and proceeded with emergency right inferior parathyroidectomy and right hemithyroidectomy. Postoperatively, she was put on calcium gluconate infusion for a few days on top of oral calcium supplement and oral vitamin D, in anticipation of hungry bone syndrome. Histopathological examination confirmed a right parathyroid carcinoma with infiltration to adjacent thyroid tissue. She was discharged well with oral calcium and oral vitamin D supplement. As for her multiple pathological fractures, they were treated conservatively.

CONCLUSION

Hypercalcemia in parathyroid carcinoma is very challenging to manage because it tends to be severe and refractory. Hemodialysis for treatment of severe hypercalcemia was shown to be effective for reduction of hypercalcemia while the patient prepare for parathyroidectomy.

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CURING HYPERTENSION: SUCCESSFUL ADRENALECTOMY FOR PRIMARY ALDOSTERONISM USING CONTRALATERAL SUPPRESSION INDEX

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INTRODUCTION

The diagnosis of Primary Aldosteronism can be challenging especially when it comes to localising an adenoma in patients who fail adrenal venous sampling. Even in expert hands, the right adrenal vein has been known to be difficult to cannulate during AVS. We present a case of a hypertensive woman with metabolic alkalosis and hypokalemia who was evaluated for primary hyperaldosteronism.

RESULTS

A 57-year-old Chinese female with a history of mini-gastric bypass with resultant resolved DM, attended clinic for poorly-controlled hypertension on 2 anti-hypertensives. She also had persistent hypokalemia (2.9-3.5 mmol/l) with metabolic alkalosis. She denied paroxysms or abnormal weight gain suggestive of pheochromocytoma or Cushing's syndrome. Physical examination was unremarkable. Her aldosterone-renin-ratio (ARR) was raised (>91) with a serum aldosterone of 747 pmol/L and renin of <1.8mU/L. A saline suppression test showed a non-suppressed aldosterone (767pmol/L). A CT-adrenal 3-phase confirmed a subcentimeter benign right adrenal gland nodule with an absolute washout of 92%, suggestive of Conn's adenoma. 24-hour urine catecholamines/metanephrines and 24-hour urinary cortisol were negative. She underwent adrenal-vein-sampling (AVS) with cosyntropin stimulation to localise the source of excess aldosterone secretion. Three series of cortisol samples demonstrated selectivity indices between 0.9-1 for the right and 6.4-24.4 for the left adrenal veins, reflecting failed right adrenal vein cannulation. Contralateral suppression index (CSI) was <1 for all 3 samples from the left adrenal vein. She was referred to the surgeons for right retroperitoneal adrenalectomy. Histopathology confirmed a 1 x 1 cm adrenal cortical adenoma. She was able to cease all anti-hypertensives and potassium supplementation immediately after the surgery.

CONCLUSION

Studies have shown that CSI has a positive predictive value up to 88.9%. Use of CSI in this patient was helpful in demonstrating absolute suppression of the contralateral zona glomerulosa of a normal adrenal gland, thus lateralising the culprit adenoma and curing a patient of hypertension.

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HYPOPHYSITIS COMPLICATED BY PANHYPOPITUITARISM AND CRANIAL DIABETES INSIPIDUS: A CASE SERIES

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INTRODUCTION

Hypophysitis is gaining greater clinical recognition over the years while continuing to be a diagnostic and therapeutic challenge. Hypophysitis is the collective term for conditions presenting with inflammation of the pituitary gland and infundibulum. It can occur as a primary entity or secondary to systemic conditions. Pituitary inflammation usually results in pituitary hormone deficiency and enlargement of the gland. Inflammatory expansion of the gland can result in compression of the optic apparatus with resulting neuro-ophthalmic consequences. We described two cases of hypophysitis complicated by panhypopituitarism and cranial diabetes insipidus (DI).

RESULTS

Case 1 is a 37-year-old man with severe intractable headache complicated by bilateral 3rd, 4th, 6th nerve palsies and left partial 5th nerve palsy. Pituitary MRI demonstrated diffusely thickened pituitary stalk with absence of posterior pituitary bright spot on T1WI. Hormonal profiles revealed panhypopituitarism with morning cortisol <11 nmol/L (185-624), ACTH <1.1 pmol/L, TSH 0.267 mIU/L (0.38-5.33), fT4 5.38 pmol/L (7.86-14.41), fasting testosterone <0.35 nmol/L (5.72-26.14), FSH 1.23 mIU/mL (1.27-19.26) and LH 0.32 mIU/mL (1.24-8.62). Connective tissue screening and tumour markers were unremarkable. Multiple analgesia failed to alleviate his headache and he was subsequently given IV methylprednisolone followed by tapering dose of prednisolone. Subsequently, his biochemical profiles demonstrated evidence of cranial DI. Case 2 is a 35-year-old woman with forgetfulness and profound lethargy. Pituitary MRI demonstrated empty sella with hypothalamic retrochiasmatic lesion with mammillary body involvement. Hormonal profiles revealed panhypopituitarism with morning cortisol 45 nmol/L, FSH 1.4 mIU/mL, LH 0.3 mIU/mL, oestradiol <18.4 nmol/L, TSH 1.03 mIU/L, fT4 below detection. Lumbar puncture cerebrospinal fluid analysis was normal. Serum angiotensin converting enzyme (ACE) was 44 U/L (16-85) and IgG-4 was 44.9 mg/dL (2.4-121). Connective tissue screening and tumour markers were unremarkable. She was replaced with hydrocortisone and thyroxine. Following glucocorticoid replacement, she demonstrated polyuria and biochemically confirmed cranial DI.

CONCLUSION

Evaluation of patients with suspected hypophysitis involves a thorough clinical and laboratory armamentaria to decide on the optimal management strategies.