CASE
A 31-year-old female with underlying diabetes mellitus, hypertension and a history of previous treatment for Cushing's disease presented with symptoms of weight gain, hirsutism and purplish striae for 2 years. MRI showed a pituitary adenoma measuring 2.8x4.4x3.3 mm. A 24-hour urinary cortisol and overnight dexamethasone suppression tests were not suppressed. She underwent endoscopic TSS. Post-surgery, her cortisol levels reduced from 419 to 57.3 nmol. Subsequently, she was found to have a saddle pulmonary embolism and extensive right lower limb deep vein thrombosis requiring pulmonary thrombectomy. Post-procedure, she was started on anticoagulants.

Hypercoagulation in Cushing's disease is due to the increase in clotting factors II, V, IX, and VIII, fast-acting plasminogen activator inhibitors and the decrease of tissue-type plasminogen. The stress post-surgery causes an abnormal Von Willebrand Factor pattern production leading to platelet aggregation and the drop in cortisol levels will trigger an inflammatory response that initiates the coagulation cascade. The elevated thrombotic risk will decrease after 3 months to a year later as the glucocorticoid effect takes time to wean off, hence requiring anticoagulation.

CONCLUSION
Recognition of thrombosis post-surgery for Cushing's disease is vital to prevent mortality and morbidity. An individualized strategy based on the degree of thrombosis is therefore essential in the management.

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HYPONATRAEMIA SECONDARY TO SIADH: COULD IT BE METHAMPHETAMINE USE?

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INTRODUCTION/BACKGROUND
Hyponatraemia can be a result of SIADH. Patients with methamphetamine use frequently present with hyponatraemia, possibly secondary to SIADH, among other complications.

We describe a patient with a history of methamphetamine use presenting with persistent hyponatraemia secondary to SIADH.

CASE
A previously well, 50-year-old male with a history of methamphetamine usage (last intake 30 years ago), presented with generalized body aches and weakness, vomiting, reduced oral intake and constipation for 1 week. On examination, his GCS was full, blood pressure was 120/72 mm Hg, heart rate was 72 and afebrile. Other systemic examinations were unremarkable. Blood parameters showed hyponatraemia with hypokalaemia (sodium 121 mmol/L, potassium 3 mmol/L). Despite 4 days of intravenous drip hydration and oral sodium chloride, his clinical condition and sodium levels did not show any improvement. His lowest sodium was 110 mmol/L, and hyponatraemic workup was consistent with SIADH (serum osmolality: 238 mOsm/kg, urine osmolality: 819 mOsm/kg and urine sodium: 187 mmol/L). Morning cortisol, thyroid function test, renal profile, Synacthen test, ACTH and tumour markers were normal. Patient was diagnosed with symptomatic hyponatraemia secondary to SIADH due to methamphetamine. Subsequently, he was started on intravenous hypertonic saline for 2 days coupled with fluid restriction of 500 mL/day. In view of imperceptible improvement of clinical symptoms and sodium level (115 mmol/L), fludrocortisone 0.1 mg tablet bid was then added. After more than 1 week of treatment, the peak serum sodium level achieved was 120 mmol/L.

Literature showed that amphetamines can be associated with serotonin-mediated hyponatraemia. This can happen as a result of SIADH or excessive water intake from hyperpyrexia following drug ingestion.

CONCLUSION
This case illustrates possibility of hyponatraemia secondary to SIADH which could be a result of methamphetamine use. Absence of urine toxicology test upon patient's presentation causes difficulty to confirm this diagnosis, and the possible duration of effect of methamphetamine-induced SIADH is yet unknown. Nevertheless, a history of recreational drug consumption should be included in the clinical evaluation of unexplained hyponatraemia.