CONCLUSION
Our study conclusively support the results obtained from other reputable centres in the West where RAI is effective in rendering hypo- or euthyroid state in more than 85% of patients with thyrotoxicosis following failed conventional antithyroid therapy.

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Diabetes Insipidus as a Clinical Signs for Adult Langerhans Cell Histiocytosis: A Case Series

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INTRODUCTION
Central diabetes insipidus (CDI) is characterized by polyuria, nocturia and polydipsia mainly by reduction of antidiuretic hormone secretion from the neurohypophyseal system. It is a rare condition. CDI more frequently caused by inflammatory processes and neoplastic infiltrations in adults as seen in germinoma, craniopharyngioma and Langerhans cell histiocytosis (LCH). Remote cases such as trauma resulting from surgery or by accident and genetic defects in the synthesis of vasopressin can also lead to CDI. Otherwise, about 30 to 50 per cent of cases is idiopathic.

METHODOLOGY
We reported three cases who are diagnosed to have Langerhans cell histiocytosis with CDI encountered by our endocrine department, Hospital Putrajaya.

RESULTS
Two women were diagnosed with CDI following the typical presentation of polyuria, polydipsia and extreme thirst. Biochemical evaluation consistent with hyperosmolar hypernatraemia followed by subsequent water deprivation test confirmed the diagnosis for both of them. Supplementation with MRI has shown both women also had a thickened pituitary stalk. Surprisingly, following that, cervical lymphadenopathy was seen in one woman and the other developed lytic lesion of the femur. Confirmatory diagnosis of LCH was made in both women following tissue biopsy of the respective area, thus they were co-managed by our haematology team. On the other hands, we have one man diagnosed with CDI following a year of LCH diagnosis and currently on hormone replacement therapy.

CONCLUSION
Our cases concluded that CDI can precede the diagnosis of LCH or can happen following the diagnosis of LCH. Hence, the diagnosis of LCH should be considered in an adult who presented with CDI symptoms.

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Thyrotoxicosis with Severe Hepatic Dysfunction: A Series of Four Cases

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INTRODUCTION
Hepatic dysfunction in thyrotoxicosis is common but rarely severe. It may be due to a multitude of reasons, namely uncontrolled thyrotoxicosis, antithyroid drugs, hepatic congestion from heart failure and associations such as autoimmune hepatitis.

We report four cases with thyrotoxicosis presenting with severe hepatic dysfunction, each with a unique etiology.

CASE 1:
A 33-year-old lady with type 1 diabetes mellitus and Graves' disease presented with severe mixed cholestatic-hepatocellular injury without fulminant hepatic failure 3 weeks after initiating propylthiouracil (PTU). Her liver function test (LFT) improved gradually 1 week after withholding PTU.

CASE 2:
A 36-year-old man with Graves' disease presented with severe cholestatic jaundice after taking carbimazole for 1 month. Total bilirubin was markedly elevated at 426 μmol/L with mild transaminitis and normal liver ultrasound. He made a full recovery after carbimazole was discontinued.

CASE 3:
A 33-year-old man presented with thyroid storm and thyrotoxic cardiomyopathy in failure. There was acute liver failure evidenced by INR of 4, total bilirubin of 173.8 μmol/L, alanine aminotransferase of 3227 U/L and aspartate aminotransferase of 3748 U/L. His LFT improved remarkably after adequate diuresis and normalization of thyroid function with Lugol's iodine, prednisolone and cholestyramine.

CASE 4:
A 53-year-old man presented with thyroid storm and jaundice with bilirubin level of 165μmol/L. Failure to improve his LFT following biochemical control with Lugol's iodine, prednisolone and cholestyramine led to the diagnosis of autoimmune hepatitis with positive anti smooth-muscle antibody and elevated immunoglobulin G level.

CONCLUSION
Severe hepatic dysfunction in patients with thyrotoxicosis carries a high mortality and limits the choice of thionamide therapy because of hepatotoxicity. It is imperative to distinguish the etiology early, attain rapid biochemical control followed by early definitive therapy with radioactive iodine or thyroidectomy.