

**PP-24****Comparison of Lipid Profiles of Patients with Pulmonary Tuberculosis (TB) with and without Human Immunodeficiency Virus (HIV)**

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**INTRODUCTION**

Pulmonary tuberculosis (TB) can co-occur with Human Immunodeficiency Virus (HIV). The inflammatory condition that accompany the infection causes the release of free radicals and Reactive Oxygen Species (ROS) which can affect the lipid profile through increase of lipid peroxidase. Previous studies showed that low serum triglycerides were found in TB and HIV-positive patients compared to the control group. Hypocholesterolemia encourages the development of TB while hypercholesterolemia leads to protection against TB with Mtb. This condition needs to be considered because it can affect the prognosis of HIV coinfecting TB. The purpose of this study is to compare lipid profiles in patients with TB with and without infection with HIV.

**METHODOLOGY**

This is a comparative analytic study. Data were taken from medical records. The population of this study were all patients with pulmonary TB with and without HIV infection in Haji Adam Malik Medan General Hospital on January 2014 to October 2018 with 72 samples for each group. Simple random sampling method was used. Data were analyzed using independent t test and Mann-Whitney U test.

**RESULTS**

We found that triglyceride levels were significantly higher in TB-HIV group ( $p < 0,05$ ) compared to TB group. The Zidovudin+Lamivudin+Efaviren regimen caused an increase in lipid profiles compared to other regimens. There were no significant differences in LDL, HDL and total cholesterol between the two groups.

**CONCLUSION**

The triglyceride levels in pulmonary TB-HIV patients are higher than pulmonary TB patients without HIV.

**PP-25****Radioiodine Therapy as an Effective Convenient Modality of Treatment for Thyrotoxicosis: An Attempt to Settle the Dust Once and For All!**

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**INTRODUCTION**

Thyrotoxicosis when treated inadequately with oral antithyroid drugs for longer than a decade may result in premature cardiovascular morbidity and mortality. In this respect, radioactive iodine therapy (RAI) has been used for more than seven decades in order to achieve lifelong remission. However not all patients become euthyroid or hypothyroid following a single dose of RAI. In view of a recent publication showing a dismal result of RAI therapy (a meager 50% success rate) in a local tertiary institution, we have embarked on a similar quest in order to address this issue once and for all.

**METHODOLOGY**

Participants were identified from the list of patients who underwent RAI therapy for thyrotoxicosis in our institution from January 2013 to April 2018. All of the patients were referred for RAI following failure of conventional antithyroid therapy to induce lifelong remission. Patients' characteristics, clinical outcomes and laboratory results were retrieved from the medical and laboratory records. Descriptive statistics were used to describe the data. Relationships were explored with appropriate statistics with significant findings established at  $p < 0.05$ .

**RESULTS**

A total of 168 patients were identified. The mean age was  $44.81 \pm 15.7$  years; 73% ( $n=123$ ) were females. 73% ( $n=119$ ) had Graves' disease and 23% had multinodular goitre ( $n=39$ ). Majority (88%) had a single course of RAI, whereas 12% needed a second course. The dose used was 15 mCi in 46% and 12 mCi in 48% of cases. Five (3%) patients were lost to follow-up following first RAI. Overall 85% ( $n=142$ ) of patients achieved remission as defined by hypothyroid or euthyroid state without any further intervention beyond 6 months of RAI.

**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.

**PP-26****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.

**PP-27****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 165 µmol/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.