13th Annual Scientific Conference & Gathering

Theme:
Environmental Virology, Exposomics and Epigenetics

Venue:
Old Great Hall, College of Medicine, University of Lagos, Idi Araba, Lagos State

Date:
Wednesday 8th June 2016

Time:
8.00 am - 5.00pm

Programme & Book of Abstracts
THE ADRENAL GLAND AND THE PATIENT WITH PULMONARY TUBERCULOSIS INFECTED WITH HIV.

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Background and Objective: The adrenal gland, as studies have shown, is not spared from involvement by tuberculosis (TB). One of the recognized causes of adrenal insufficiency (AI) is TB. AI secondary to Human Immunodeficiency Virus (HIV) infection has been well studied. AI, mostly at the subclinical level, is common in persons with pulmonary TB (PTB) infection, occurring in about 23% of patients. Co-infection with PTB and HIV may compromise adrenocortical function and produce significant adrenocortical insufficiency. The objective of this study was to determine if co-infection with tuberculosis and HIV has a compound effect on adrenocortical function in persons with HIV and PTB co-infection.

Methods: Persons with sputum-positive PTB, treatment naive, who met other inclusion criteria, were selected. All recruited patients were screened for HIV (plus confirmatory test for all HIV positive). Baseline blood for cortisol, fasting plasma glucose (FPG), full blood count (FBC) and electrolytes were collected between 08.00 hour and 9.00 hours immediately before administration of adrenocorticotropic hormone (ACTH). The subjects received an intravenous bolus injection of 1 µg ACTH and blood sample was drawn for cortisol level at 30 minutes. Serum cortisol level was determined by an Enzyme Linked Immunosorbent Assay (ELISA) technique.

Results: 44 with PTB infection and 40 with PTB and HIV co-infection met the inclusion criteria for the study. The mean mass index in the participants with PTB and PTB and HIV co-infection was 18.9±2.9 and 21.8±2.6 kg/m² respectively (p<0.05). The adrenal response to 1 µg ACTH stimulation in participants with PTB and PTB and HIV co-infection showed the mean basal cortisol level in the 2 groups was not statistically significant. However, 30-minute post ACTH stimulation cortisol level was 630.8±372.2 and 980.4±344.8 nmol/L (p<0.001) and increment was 367.8±334.9 and 740±534.8 nmol/L (p<0.001) respectively. Fourteen persons (31.8%) out of 44 of those with PTB had subnormal cortisol response to ACTH stimulation while only 2 (5%) persons with PTB and HIV co-infection had subnormal response. There was no significant intergroup difference in the biochemical parameters.

Conclusions: Adrenal insufficiency, at subclinical level, was less frequent in those with PTB and HIV co-infection. This suggests that sufficient degree of infiltration of the adrenal gland to cause a clinically significant deficit does not occur.

Keywords: adrenal gland, adrenocortical insufficiency, pulmonary tuberculosis, human immunodeficiency virus.