FACULTY OF CLINICAL SCIENCES

COLLEGE OF MEDICINE, UNIVERSITY OF LAGOS

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 •

FCS/FM/16/22

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 08.00hour and 9.00hour immediately before administration of adrenocorticotrophic hormone (ACTH). The subjects received an intravenous bolus injection of 1µg ACTH and blood sample was drawn for cortisol level at 30 minutes. Serum cortisol levels were 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 body mass index in the participants with PTB and PTB and HIV co infection was 18.9±2.9 and 21.8±2.6 kg/m^2 respectively ($p<0.05$). The adrenal response to 1µg ACTH stimulation in participants with PTB and PTB and HIV co infection was that the mean basal cortisol level in the 2 groups was not statistically significant. However, 30-minute post ACTH stimulated 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 740.8±[illegible] nmol/L ($p<0.001$) respectively. Fourteen persons (31.8%) out of 44 of those with PTB had subnormal adrenal 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