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PLASMA CORTISOL IN TUBERCULOSIS

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Cherednikova (1973) reported that corticoids medication, as a part of complex antituberculous therapy, was beneficial in the treatment of tuberculosis in children.

Ionescu (1973) reported that in 70 cases of Addison's disease, there was a past history of tuberculosis in 85% of these addisonian patients, one to five years before the incidence of Addison's disease. These findings directed our attention to study the suprarenal cortical function in tuberculosis.

MATERIALS AND METHODS

47 human males and females of an age ranging 15~62 years comprise the experimental subjects. These subjects were classified into 3 groups:

(I) 16 normals.

(II) 15 patients with active tuberculosis.

(III) 16 patients after treatment from tuberculosis.

From each subject 4 plasma samples were taken to estimate the plasma cortisol, by Mattingly method (1962).

(a) The first sample of plasma was taken at 5 P.M.

(b) The second sample of plasma was taken at 8. A. M. fasting, after which 1/2 m*l*. of I. M. "synacthen Depot Ciba" was given.

(c) The third sample of plasma was taken at 5 P.M. of the same day i.e. 8 hours after synacthen stimulation.

(d) The fourth sample of plasma was taken at 8 A.M. of the next day i.e. 24 hours after synacthen stimulation.

Results: are illustrated in the following table.

Average of Plasma Cortisol in T.B.

Group	5 P.M. μg %	8 A.M. μg %	D.V μg %	D.V %	8 hours stimulation μg %	Response in μg %	% Response increase	24 hours stimulation μg %
I-Normals	11.8	19.4	7.6	64.0	35.0	15.6	80.4	11.7
II-Active T.B.	25.6	35.8	10.2	39.8	51.6	15.8	44.1	24.2
p	<.01	<.01	<.01		<.01	<.2		< .01
III-After treatment compared with normals	14.6	24.8	10.2	96.8	38.0	13.2	53.4	13.0
p	<.01	<.01	<.01		<.01	<2.4		<1.3
After treatment compared with active								
p	<.01	<.01			<.01	<.01		< .01

DISCUSSION

Plasma cortisol level increased significantly in active tuberculosis, the increase is apparent in all the four samples taken. This might be due to the continuous stress induced by the tuberculous toxins and the catabolic process of the Mycobacterium tuberculosis on the adrenal cortex itself. The high levels of plasma cortisol at 5 P.M. and 8 A.M. would lead to a decrease in the diurnal variation compared to normals.

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The increased response recorded in normals (80.4%) after 8 hours stimulation by synacthen depot was found to decrease to 44.1% in active tuberculosis indicating a significant decrease in the adrenocortical reserve capacity, which might be due to derangement of the adrenal cortical function due to infection with tuberculosis.

Abdel Kader *et al.*, (1975) noticed a persisting hypoglycaemia after glucose tolerance curves in tuberculous patients which was attributed to hyperinsulinism. This hyperinsulinism might cause an increased level of plasma cortisol as a compensatory mechanism to increase the plasma glucose level in active tuberculosis.

After treatment of tuberculosis there was a significant decrease in all levels of plasma cortisol compared to the active tuberculous state. The diurnal variation returned to normal after treatment, the reserve capacity (8 hours after stimulation) is still significantly decreased indicating a permanent damage in the adrenal cortical function.

The levels of plasma cortisol after treatment were still high compared to normals, indicating a disorder in the hormonal balance accompaning tuberculosis and persisting after its treatment.

These high levels of plasma cortisol after treatment might be due to hyperplasia and hypertrophy of the adrenal cortex by the long stress of the disease. Follow up of these patients for a longer time is required to explore the adrenal cortical function after several years of treatment.

SUMMARY

Plasma cortisol estimated in active tuberculosis has been shown to be high compared to normals, but there is a significant decrease in the reserve capacity of the adrenal cortex recorded after 8 hours stimulation with a synacthen-depot-"Ciba". The diurnal variation is significantly decreased in the active disease. After treatment, plasma cortisol level significantly decreased compared to the active disease, the diurnal variation returns to normal, but the reserve capacity of the adrenal cortex is still decreased indicating a permanent damage in the adrenal cortex.

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