



SERUM CORTISOL LEVELS IN DEPRESSION PATIENTS

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Article Type:**Full Length Research****Keywords:**steroid hormone, cerebrospinal
fluid, Serum Cortisone.**Abstract**

Cortisone is a steroid hormone, responsible for the stress resp, released from the adrenal gland, altered activity of the hypothalamo-pituitary-adrenal (HPA) axis is one of the most consistent findings in depression. The present investigation aimed to study the serum cortisol level in patients with major depression. Serum cortisol level was higher in depression patients $14.5 \pm 3.01 \mu\text{g/dl}$ than in control group $11.56 \pm 1.33 \mu\text{g/dl}$. Conclusion: Hyper secretion of corticotrophin releasing hormone increased cortisol levels in plasma, urine and cerebrospinal fluid exaggerated cortisol responses to adrenocorticotrophic hormone and enlarged pituitary and adrenal glands occurs in individuals suffering from severe mood disorders.

Introduction

Cortisone (17-hydroxy-11-dehydrocorticosterone) is a steroid hormone. In chemical structure, it is a corticosteroid closely related to corticosterone. Cortisone suppresses the immune system, thus reducing inflammation and attendant pain and swelling at the site of the injury. Cortisone, a glucocorticoids is the main hormone released by the body as a reaction to stress. It prepare the body for a fight or flight response (Andersen, 2002).

Cortisone may also be used to deliberately suppress immune response in persons with autoimmune diseases or following an organ transplant to prevent transplant rejection. The suppression of the immune system may also be important in the treatment of inflammatory conditions such as severe IgE-mediated allergies (Bardi, et al.,2004).

Cortisone is one of several end-products of a process called steroidogenesis. This process starts with the synthesis of cholesterol, which then proceeds through a series of modifications in the adrenal gland (suprarenal) to become any one of many steroid hormones. One end-product of this pathway is cortisol. For cortisol to be released from the adrenal gland, a cascade of signaling occurs. Corticotropin-releasing hormone released from the hypothalamus stimulates corticotrophs in the anterior pituitary to release ACTH, which relays the signal to the adrenal cortex. Here, the zona fasciculata and zona reticularis, in response to ACTH, secrete glucocorticoids, in particular cortisol. In the peripheral tissues, cortisol is converted to cortisone by the enzyme 11-beta-steroid dehydrogenase. Cortisol has much greater glucocorticoid activity than cortisone, and, thus,

cortisone can be considered an inactive metabolite of cortisol. However, 11-beta-steroid dehydrogenase can catalyze the reverse reaction as well, and, thus, cortisone is also the inactive precursor molecule of the active hormone cortisol. Cortisone is activated through hydrogenation of the 11-keto-group, and cortisol is, thus, sometimes referred to as hydrocortisone (Beishuizen, et al., 2001).

Oral use of cortisone has a number of potential side-effects: hyperglycemia, insulin resistance, diabetes mellitus, osteoporosis, anxiety, depression, amenorrhoea, cataracts and glaucoma, among other problems (Castro, et al.,2000).

Cortisol is activated in a brain region called the locus coeruleus, which sends norepinephrine to communicate back to the amygdala, and so responsible for the stress response all over again and resulting in a destructive feedback cycle continuously. Depression is often associated with hypercortisolemia as the high levels of cortisol influence the distribution of various types of leukocytes in the blood stream. The hormone released during long-term stress are responsible for the depression but after years of circumstantial evidence, it is found that cortisol also causes mood disorders for some people. (Davidson, et al.,2006).

Major depression is a complex disorder characterized by disturbed mood and behavior as well as by neuroendocrine and immune abnormalities (Irwin and Miller, 2007). Altered activity of the hypothalamo-pituitary-adrenal (HPA) axis is one of the most consistent findings in depression (Pariante and Lightman, 2008 ,Zunszain et al., 2010). Mild to moderate hyperactivity of the HPA axis resulting in increased plasma cortisol is observed in 30–50% of depressed

Table 1: Cortisol serum level in depression patients and in control group

Cortisol serum level in µg/dl	In depression patients N =21	In control group N =20
Mean ± SD	14.5±3.01	11.56±1.33

P. Value for depression patients versus Control group was 0.017 significant

subjects ([Pariante and Lightman, 2008] and (Rubin et al., 2001). Moreover, non-suppression of cortisol secretion in the combined dexamethasone/corticotropin-releasing hormone (CRH) test has approximately 80% sensitivity in major depression, although this test can be positive in some other psychiatric disorders (Heuser et al., 1994). Hypercortisolemia, however, is not obligatory for major depression which can be accompanied by low plasma cortisol levels as well (Stewart et al., 2005)(Vythilingam et al., 2010).

In view of the above information, the present investigation is an attempt to study the serum cortisol level in patients with major depression.

Material and Methods

This study was carried out on 21 patients as depression group, and 20 healthy subjects as control group matched the depression group in age and socio-economic status, serum samples were obtained from both patients with depression and healthy controls. Blood collection was done at 8 AM. All subjects gave written informed consent for study participation. Patients were recruited at the in-patients services of the Clinic of Psychiatry, El-Madina Hospital KSA. Patients with major depression, all males (age, mean ±S. D., 43.5±1.8 years) fulfilled strict criteria for major depressive disorder according to DSM-IV (DSM-IV; American psychiatric Association, 1994). the DSM-IV diagnosis of depression was determined via clinical interviews by two psychiatrists independently. Twenty male healthy participants (age, mean ± S.D., 42.8 ± 3.0 years) were also recruited and interviewed by a psychiatrist to exclude depression or other psychiatric disorders. Immediately before the blood sampling, all study subjects completed the Montgomery–Asberg Depression Rating Scale (MADRS) (Montgomery and Asberg, 1979, Muller et al., 2003). Exclusion criteria were significant psychiatric co-morbidity, organic mental disorder, mental retardation, bipolar disorder, anxiety disorders if primary and/or predominant, alcohol abuse or dependence during the last 12 months.

Results and Discussion

Quantitative determination of cortisol serum level was

performed using a Accu-Bind ELISA microwells product code 3625-300.

Data were expressed as Mean ± standard deviation, Comparisons were performed for normal distributed data using T. test for independent groups, P. value considered insignificant if it >0.05, significant if it ≤0.05, highly significant if it ≤0.001, the statistical analysis were done using SPSS V11.0 program. Cortisol serum level was higher in depression patients 14.5±3.01µg/dl than in control group 11.56±1.33 µg/dl . P. Value was 0.017 significant as shown in table 1 and figure 1

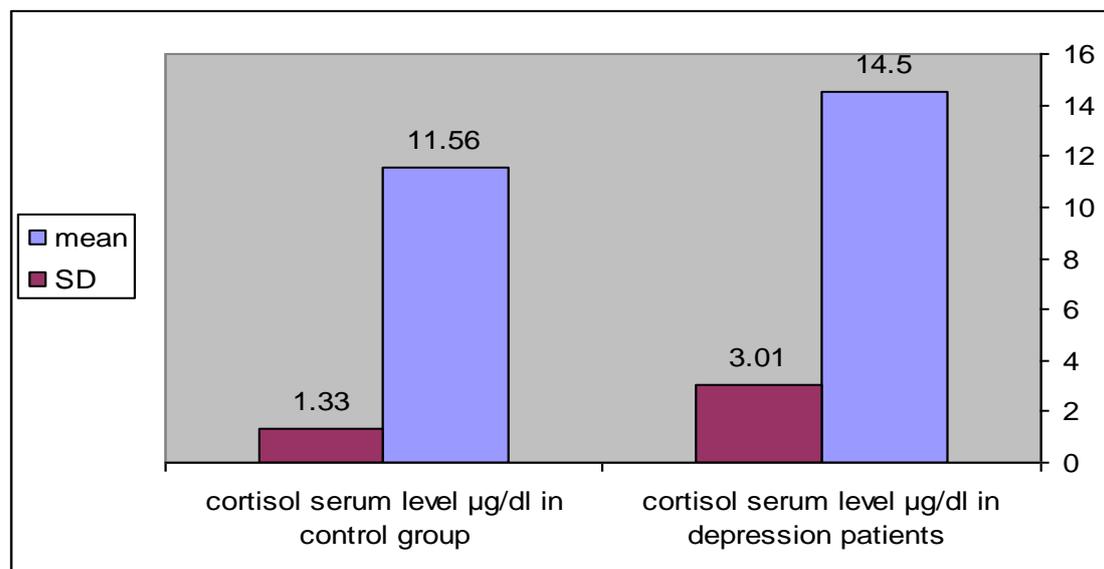
Psychopathologies such as anxiety- and depression-related disorders are often characterized by impaired social behaviors including excessive aggression and violence. Excessive aggression and violence likely develop as a consequence of generally disturbed emotional regulation.

Social behaviors in mammals are under control of steroid hormones and neuro-peptides. The alteration of the hypothalamic–pituitary–adrenal axis (HPA) and changes of the steroid metabolism associated with major depression (MD) was overviewed by Bradley (2000) and Young et al.(2002).HPA hyperactivity as manifested by hyper secretion of CRH increased cortisol levels in plasma, urine and CSF exaggerated cortisol responses to ACTH and enlarged pituitary and adrenal glands occurs in individuals suffering from severe mood disorders. Hyper-secretion of CRH causing hypercortisolemia may be a result of impaired feedback mechanism resulting from glucocorticoid receptor (GR) abnormalities such as decreased receptors number or altered function, this view is supported by the demonstration of GR abnormalities in postmortem studies of patients with severe mood disorders (Ingemar et al., 1994).

In this present study cortisol secretion was evaluated in 20 depression male patients, the study shown a significant increase in serum level of cortisol in depression patients 14.5±3.01µg/dl than in control group 11.56±1.33 µg/dl . These results are in accordance with other researches,

The neuro-endocrine studies are mainly focused on the elevation of cortisol in serum (Heuser et al., 1998; Takebayashi et al., 1998 and Weber et al., 2000) and in the saliva (Goodyer et al.,1998), as it is known to induce abnormal affective status (Lewis and Smith, 1983 and Michael et al., 2000).

Figure 1: Cortisol serum level in depression patients and in control group



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