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LETTERS TO THE EDITORS

New insights into cortisol levels in PTSD

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The article ''Relationship of cortisol, norepinephrine, and epinephrine levels with war-induced posttraumatic stress disorder in fathers and their offspring'' by Yahyavi et al.[1] provided interesting data on the neuroendocrinology of post-traumatic stress disorder (PTSD). The mechanisms that underlie the associations of cortisol levels with traumatic exposures and PTSD are still not well understood and, as stated by the authors, findings are far from consistent.

Similarly to the findings of Yahyavi et al., in a sample of individuals exposed to trauma during the preceding 5 years, patients with PTSD had neither an increase nor a decrease in mean urinary cortisol levels.[2] Conversely, in a recently published study, Wingenfeld et al.[3] reported decreased cortisol values in outpatients with PTSD recruited from two Veterans Affairs medical centers. Furthermore, researchers have reported lower cortisol levels in the acute aftermath of trauma in patients who later developed PTSD.[4]

It seems that inadequate glucocorticoid release following stress not only delays recovery by disrupting biological homeostasis in the short run but can also interfere with the processing or interpretation of stressful information, resulting in long-term disruptions in memory integration.[5] Consistent with these findings is the fact that a single dose of hydrocortisone administered in the acute aftermath of trauma produced recovery while promoting enhanced synaptic plasticity and connectivity in the secondary prevention of PTSD.[5] In this sense, it remains unclear whether deregulation of the HPA axis, leading to low peritraumatic levels of cortisol, endures post-traumatically.

Another critical issue in this matter has to do with changes in cortisol levels secondary to pharmacological treatment of PTSD (e.g., sertraline), an important aspect not addressed by the authors.[6] Finally, it is worth mentioning that PTSD is characterized by the presence of four symptom clusters (intrusion, avoidance, negative alterations in cognitions and mood, and alterations in arousal and reactivity) instead of the three mentioned in the paper.

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Disclosure

The author reports no conflicts of interest.

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Clozapine-induced esophagitis at therapeutic dose: a case report

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The pharmacological profile of clozapine, which is often described as a ''broad spectrum'' antagonist, is different from that of other antipsychotics. It is thought that the side effects reported with clozapine may be attributed in part to its anti-serotonin action together with its anti-α-adrenergic, anticholinergic, and antihistaminic effects.[1]

A 58-year-old chronic schizophrenic patient had been treated with haloperidol for over 10 years. Haloperidol was eventually replaced with a combination of antipsychotics (olanzapine/zuclopenthixol and later aripiprazole/zuclopenthixol). Given the persistence of major psychotic symptoms, clozapine was gradually introduced in combination with the following treatments: aripiprazole (10 mg/d), zopiclone (7.5 mg/day, as needed), tropatepine (15 mg/day), and lorazepam (7.5 mg/d). During the first month of treatment, the dose of clozapine was gradually increased to 275 mg/d (at the end of 1 month). However, various side effects were observed, leading to the increase in tropatepine (30 mg/d) for extrapyramidal side effects and to the introduction of macrogol 4,000 (30 g/d) for constipation, heptaminol (563.4 mg/d) for orthostatic hypotension, and paracetamol (3 to 4 g/d) for headaches.

After a second month of treatment the patient continued to describe somatic complaints, including nausea. Aripiprazole was stopped, and tropatepine was gradually reduced to 10 mg/d. In the meantime, clozapine was gradually increased based on good hematologic