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A paper published in the current issue of psychotherapy and psychosomatics provides a new hypothesis concerned with the role of aldosterone in combat PTSD.

6 Caucasian male outpatients, aged 34-64 years (mean 54.1 years) discovered to have hyperreninemic hypoaldosteronism (HH) during an endocrine workup of medical comorbidities.HH was defined as a plasma renin activity greater than 2 ng/ml/h and a plasma aldosterone concentration to plasma renin activity ratio of less than 2.

The patients were all combat veterans of the Persian Gulf (n = 2) or Vietnam conflict (n = 4). They were all diagnosed with PTSD by DSM-IV-TR criteria . Two had comorbid major depressive disorder and alcohol dependence, in remission. All met the clinimetric definition of allostatic overload (AO) and were considered disabled due to the severity of their psychiatric symptoms.

The cohort had the following medical comorbidities: endocrine/nutritional/metabolic disease [obesity (n = 4), hyperlipidemia (n = 5), diabetes mellitus (n = 3), and hypogonadism (n = 4)], nervous system disorders [chronic pain (n = 6)], circulatory disease [myocardial infarction (n = 2)], hypertension (n = 5), respiratory disease [asthma, chronic obstructive pulmonary disease (n=2)], digestive disease [irritable bowel disorder, gastric ulcer, gastroesophageal reflux (n = 2)], and musculoskeletal disease (n = 6).

Abnormal a.m. cortisol was observed in patient 1. All had adrenocorticotropic hormone levels within the reference range. Three patients showed evidence of proinflammatory profiles by elevated interleukin-6 and erythrocyte sedimentation rate. The etiology of HH in this population is currently unclear.

The allostatic model of maintaining stability through change is an intriguing possibility. This model posits that vulnerable individuals develop AO as a result of attempted allostatic accommodation to chronic uncontrollable stress. The brain, as the primary regulator of allostatic accommodation, potentially develops maladaptive alterations in the

hypothalamic-pituitary-adrenocortical axis and sympathetic-adrenal-medullary stress axis to maintain homeostasis in the presence of AO.

This multisystem dysfunction has been hypothesized to lead to a cascade of events resulting in a predisposition to medical comorbidities as a result of chronic stress. The above cases, though observational in nature, suggest psychoneuroendoimmunologic disruption as a possible predisposing factor for the development of comorbidities in vulnerable populations.

The allostatic theory would not suggest that PTSD leads to medical comorbidities. PTSD, however, may be a relatively early manifestation of morbidity in the presence of AO. Such a possibility needs further exploration.

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