DISERTASI

MODULASI RESPONS IMUN DI ARTERIA PUDENDUS INTERNUS PADA TIKUS WISTAR DALAM KONDISI STRES

Suatu studi psikoneuroimmunologi menuju patogenesis Disfungsi Ereksi Psikogenik

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ABSTRACT

It has long been stated that psychogenic stressors like anxiety, depression, stress may cause Erectile Dysfunction (ED). Many clinical studies have shown the relationship between psychogenic conditions and the development of ED (psychogenic ED). However, until recently, the exact mechanism of ED in patients with psychogenic stressor has not been understood.

To clarify the pathogenesis of psychogenic ED, an animal experimental research, using psychoneuroimmunology (PNI) paradigm was conducted. The design chosen was a Randomized Control Group Posttest only Design.

Forty male rats, Rattus Nervigicus of Wistar strain were used and by random assignment divided into 4 groups of ten rats. Group I (control group for 5 days experiment), group II (experiment group 5 days), group III (control group for 10 days experiment), group IV (experiment group 10 days). The rats were 3-4 month old and had a body weight 150-200 gr.

An electric footshock (EF) were used as an psychogenic stressor (as independent variable), exposed to the rats. The dependent variables observed were Immune Response Modulations (IRM) of the neurohormonal changes expressed in cortisol, catecholamines (adrenalin, noradrenalin) blood level, amount of VCAM-1 expressing endothelial cells (ECs) and amount of IL-2, IFNγ, IL-4, IL-10 producing lymphocytes in the internal pudendal artery wall, which is the main artery for erection. A multivariate analysis was used to evaluated the data.

The results after 5 days EF exposure showed cortisol, adrenalin and noradrenalin blood levels increased; amount of VCAM-1 expressing ECs increased and amount of IL-2, IFNγ, IL-4, IL-10 producing lymphocytes decreased. After 10 days EF exposure cortisol, adrenalin and noradrenalin blood levels also increased; amount of VCAM-1 expressing ECs decreased and amount of IL-1, IFNγ, IL-4, IL-10 producing lymphocytes were more decreased.

To explain the mechanism, the functional contribution of each variables to the development of ED is needed. Based on discriminant analysis, only 5 variables (cortisol, adrenalin, VCAM-1, IL-4, IL-10) have contribution in IRM pattern. It showed that after 5 days EF exposure, IRM caused an increased adrenalin function, but decreased cortisol function. VCAM-1 expressing ECs and IL-4, IL-10 producing lymphocytes functions were increased.

After 10 days EF exposure there was a prominent increased cortisol function, but a decreased adrenalin function. IL-4 and IL-10 producing lymphocytes functions were more increased whereas VCAM-1 expressing ECs function was decreased.

In conclusion: the increased adrenalin function after 5 days EF exposure and increased VCAM-1 expressing ECs function may cause endothelial dysfunction and potentially may cause endothelial damage in the internal pudendal artery wall, and hence cause psychogenic ED.

After 10 days EF exposure cortisol function increased, which consequently cause increased IL-4 and IL-10 function. This condition where Th2 cells are dominant may cause endothelial damage of the internal pudendal artery wall through ADCC (antibody-dependent cell-cytotoxicity) process, and hence may cause psychogenic ED.

Keywords: Stress, Immune Response Modulation, Internal Pudendal Artery, Erectile Dysfunction, Psychoneuroimmunology