Abstract

**Background:** Interactions between the neuroendocrine and immune system play an important role in maintaining homeostasis. This communication is mediated by cytokines, neurotransmitters and hormones through endocrine, paracrine and autocrine signaling. Prolactin (PRL), hormone of anterior pituitary, is produced by a number of other tissues and cells of immune system. On periphery, PRL is cytokine. Sepsis is an inflammatory response of the organism to severe infection, Th1 immune response is activated and PRL could participate in it. Toll-like receptors (TLR) play a key role in a recognition of bacterial components and mediate a systemic response (with PRL secretion) during infection. It is supposed that activated immune system leads to increasing of PRL, TLR2 and TLR4 gene expression. We detected PRL, TLR2 and TLR4 mRNA levels in monocytes from patients with systemic inflammation. We studied influence of single nucleotide polymorphism (SNP -1149 G/T) in PRL gene promoter, it supposed that G allele increases PRL expression.

**Materials and Methods:** For the pilot study 30 patients diagnose with severe infectious event. Collectoin of patients blood samples was performed consequently three times. Control group comprised 40 healthy individuals. One blood sample was taken from each healthy subject. For testing of PRL, TLR2 and TLR4 gene expression in monocytes (immunomagnetic separation) was used the Real Time PCR method with phosphoglycerate-1 as an internal control. For detection SNP -1149 G/T in PRL gene promoter was used the restriction fragment length polymorphism method (RFLP) with XapI enzyme.

**Results:** Levels of PRL, TLR2 and TLR4 mRNA in monocytes from peripheral blood of patients in consequent samples revealed no statistically significant change. But we detected statistically significant increase TLR2 and TLR4 mRNA levels in patients as compared with controls. We didn’t detect statistically significant difference between PRL mRNA levels in controls and patients. We detected statistically significant increasing of PRL mRNA levels in GG genotype in healthy controls.

**Conclusion:** The obtained results suggest that prolactin production in monocytes is activated during severe infection. It seems that SNP -1149 G/T may influence the physiological levels of PRL peripheral expression in monocytes. Increased TLR levels suggest connection with increased PRL gene expression.

**Keywords:** prolactin, cytokine, immunity, sepsis, toll-like receptor