OVERACTIVE BLADDER IS ASSOCIATED WITH UPREGULATION OF MONOCYTE CHEMOATTRACTANT PROTEINS: A PROSPECTIVE SINGLE BLINDED CONTROLLED STUDY

Hypothesis / aims of study

The contemporary etiology of OAB is believed to be through the activation of the local network made up of urothelium and nerve endings in the suburothelium, regulated by interstitial cells (IC). Neurogenic and myogenic sources have been assumed for OAB pathogenesis (1). Chemokines, a large family of small proteins, were found to have a critical role in immune response and inflammatory reactions. Monocyte Chemoattractant Proteins (MCPs) represent chemokines that are strongly implicated in inflammatory and allergic conditions. Bouchelouche and associates have recently reported the secretion of MCP1 from detrusor smooth muscle cells when cultured in inflammatory environment in vitro (2). In this study; we measured the MCPs in the urine of patients with OAB and compared with normal subjects.

Study design, materials and methods

This is a prospective, single blinded study which included twenty healthy pre-menopausal women (group I) and 20 women suffering from OAB (group II). Urine samples were collected, centrifuged, and mixed with protease inhibitor solution, pH 7.4 to prevent protein degradation, frozen, and stored at −80°C. Urinary total proteins were quantified using BCA protein micro assay kit in accordance with manufacturer’s instructions. Differential expression profile analysis of MCPs in urine samples of the two groups were performed using a human cytokine protein chip. The levels of each chemokine were expressed and compared as mean +/− SE in normal subjects and OAB patients.

Results

MCPs were found significantly increased in OAB patients compared with normal subjects. MCP-1, MCP-2 and MCP-3 were found to have 2 fold or more positive expression in OAB patients versus normal subjects (p< 0.007).

Interpretation of results

These results suggest a possible inflammatory mechanism in OAB. The inflammatory environment is proposed to induce MCP secretion by the detrusor muscle cells.

Concluding message

The over expression of MCPs in the urine of OAB patients compared with control subjects suggests an inflammatory role in the pathogenesis and development of OAB. Correlation between MCPs levels and severity of symptoms is a potential target for further research.

References