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URINARY EXCRETION OF MONOCYTE CHEMOTACTIC PROTEIN-1 IN PATIENTS WITH INTERSTITIAL CYSTITIS

Aims of Study

Interstitial cystitis (IC) is a chronic inflammatory disease of unknown etiology characterized by mast cells recruitment and development of fibrosis in the bladder tissue. The involvement of chemokines in inflammation of the human urinary bladder is unknown. Monocyte chemotactic protein –1 (MCP-1) is excreted by activated cells incl. lymphocytes, macrophages, smooth muscle cells and by a variety of human tumour cells. MCP-1 is a potent chemokine which causes mast cell, eosinophil and macrophage recruitment, and provoke mast cell activation in vitro. Furthermore, MCP-1 has been shown to be involved in development of fibrosis and irreversibel tissue damage. However, nothing is known about the role of MCP-1 in patients with IC. In search for a urinary marker for IC we investigated whether MCP-1 is elevated in the urine of patients with IC compared to controls.

Methods

Urinary MCP-1 was determined with an enzyme linked immunoassay (ELISA) that has been validated for use in urine. Morning spot urine samples were collected from 20 patients with IC diagnosed according to NIDDK criteria and 20 healthy women. All IC patients were without bladder installations the last 2 months or oral treatment with Montelukast for 4 weeks. Aliquots of each specimen were immediately centrifugated and the supernatants were frozen at $-20^{o}C$ until assay. All determinations were performed in duplicate and normalized to urine creatinine.

Results

The mean mast cell count in detrusor biopsies from IC patients was 44.8 mast cell / mm^2 detrusor (range 11 to 79). All urine cultures were negative. The mean urinary MCP-1 excretion plus or minus standard deviation was 18.69 ± 8.05 and 24.78 ± 15.70 ng/mmol creatinine in healthy subjects and IC patients, respectively. The difference was not statistically significant ($p > 0.05$).

Conclusions

The results show that MCP-1 is not elevated in the urine of patients with IC compared to healthy subjects and thus indicate that MCP-1 may not be useful as a diagnostic urinary marker in IC. It is possible that MCP-1 may not reach the urine in sufficient quantities to be detected by ELISA. However, the results do not exclude a pathogenic role of MCP-1 in IC. MCP-1 may be secreted locally in the urinary bladder where it may be involved in inflammation and tissue damage. The role of chemokines in the inflammatory bladder disease Interstitial Cystitis needs further investigations.