Methods

Urinary MCP-1 levels were measured in 36 patients with OAB and 13 controls. Patients were treated after the first visit by different OAB treatments (anticholinergic, Beta-3 agonist, onabotulinum toxin A, neuromodulations). Urinary MCP-1 levels were measured using enzyme-linked immunosorbent assay (ELISA), normalized by urinary creatinine levels, and expressed as picograms (pg) per milligram (mg) of creatinine.

The urinary MCP-1 levels were compared at baseline (pre-treatment) and 3 months (post-treatment). Severity of OAB symptoms were compared at baseline, 1 month and 3 months. Different validated OAB questionnaires were used.

Conclusions

OAB-responders had significantly reduced urinary MCP-1 levels in association with a decreased severity of OAB symptoms after treatment. Understanding the pathophysiology of OAB, neurophysiological signaling in the bladder function, identification of a potential marker, and/or lead to the development of new drug targets for the treatment of patients suffering from OAB.