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THE RELEVANCE OF IMMUNE RESPONSES TO PARTIAL BLADDER OUTLET OBSTRUCTION AND REVERSAL

Hypothesis / aims of study
Partial bladder outlet obstruction (PBOO) causes tissue inflammation, a significant increase in markers of systemic oxidative stress, and proliferation of circulating myeloid-derived suppressor cells. Here, we investigated the regulatory mechanisms underlying
inflammation and helper T cell involvement in PBOO.

Study design, materials and methods
Surgical PBOO was performed in four groups of rats: control (C), obstruction at 2 (O2) and 4 (O4) weeks, and 4 weeks after the relief of PBOO (R4) (n=6 each). The urinary levels of prostaglandin E metabolite (PGEM), expression of inflammatory cytokines (IL-6 and IL-17) in the bladder, numbers of peripheral blood regulatory T cells (Treg cells) and levels of TGF-
β1 were assessed via immunohistochemistry, flow cytometry, or ELISA.

Results
The levels of urinary PGEM, bladder IL-17, and TGF-β1 and the numbers of peripheral Treg cells (Foxp3) were all significantly increased at 2 and 4 weeks after PBOO. PGEM, IL-17, and Treg cells (Foxp3) were decreased after the relief of PBOO,while the levels of TGF-β1
continued to increase.

Interpretation of results
Dynamic changes in immune responses suggest that the levels of IL-6 in the bladder may serve as a marker for the acute stage of PBOO, while plasma TGF-β1 indicates the presence of PBOO. However, urinary PGEM and bladder IL-17 expression may result in an increase in circulating Treg cells and inhibitory cytokine production during obstruction. TGF-β1 may serve to restrict inflammation during the late or convalescent phase of PBOO.

Concluding message
Transient PBOO triggers an acute, reversible increase in inflammatory cytokines and Treg cells. The distinct dynamics of individual inflammatory markers support their potential
use as markers for monitoring bladder inflammation.

Disclosures
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