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BLADDER FUNCTION IN 17β-ESTRADIOL-INDUCED NONBACTERIAL PROSTATITIS MODEL OF THE RAT

Hypothesis / aims of study
Chronic pelvic pain syndrome/chronic prostatitis (CPPS/CP) is a devastating disease, symptoms of which include pelvic pain as well as irritative bladder symptoms such as frequent voiding and urgency. Funahashi et al reported that chemical prostatic inflammation model in the rat induced by formalin injection into bilateral ventral lobes of the prostate exhibited bladder overactivity (frequent micturition) [1]. On the other hand, Bernoulli et al reported that in the rat nonbacterial prostatic inflammation model induced by estrogen and androgen treatment, bladder capacity, micturition interval time and the relative bladder weight were significantly increased [2]. We investigated the bladder function of nonbacterial prostatitis (NBP) model that was induced by the injection of 17β-estradiol in castrated rats.

Study design, materials and methods
Ten-month-old male Wistar rats were divided into 2 groups (sham vs NBP; 8 rats in each). NBP model [3] was experimentally induced by castration followed by daily subcutaneous injection of 17β-estradiol for 30 days. On the 31st day after surgery, we investigated several parameters; (1) voiding behavior in metabolic cages overnight for 12 hours, (2) bladder blood flow (BBF), (3) measurements of the levels of proinflammatory cytokines (TNF-α and CXCL1) in the prostate and bladder, and histological examination, (4) Bladder contractile responses to electrical field stimulation (EFS: 2, 8, 32 Hz), carbachol (100 μM), and KCl (100 mM).

Results
Voiding behavior (average micturition volume, total urine volume and number of micturitions) and BBF were not significantly different between sham and NBP group. NBP led to a significant decrease in prostatic weight (Sham: 500.79 ± 15.99 vs NBP: 239.76 ± 20.39 mg; p<0.01) and increase in proinflammatory cytokine levels (TNF-α; Sham: 0.75 ± 0.18 vs NBP: 1.52 ± 0.18 pg/mg protein, CXCL1; Sham: 1.33 ± 0.26, NBP: 15.26 ± 1.50 pg/mg protein; p<0.01) in the prostate, while NBP did not cause a significant change in bladder weight or proinflammatory cytokine levels in the bladder. Bladder contractile forces in response to EFS, carbachol and KCl were not significantly different between sham and NBP group.

Interpretation of results
NBP did not cause a significant change in bladder weight or proinflammatory cytokine levels in the bladder. Bladder function was not significantly different between sham and NBP group.

Concluding message
The present study suggests that various rat prostatitis models differ in lower urinary tract dysfunction depending on the cause of prostatitis and severity of inflammation.

References

Disclosures
Funding: None Clinical Trial: No Subjects: ANIMAL Species: Rat Ethics Committee: All protocols were approved by Asahikawa Medical University Institutional Animal Care and Use Committee, and conducted in compliance with the Internal Regulations on Animal Experiments at Nippon Shinyaku Co., Ltd, which are based on the Law for the Humane Treatment and Management of Animals (Law No. 105, October 1, 1973, as revised on June 1, 2006).