

DOES PROLACTIN PLAY A ROLE IN VOIDING FUNCTION?- A STUDY ON THE HYPERPROLACTINEMIC RATS

Hypothesis / aims of study

Prolactin plays a role in fluid and electrolyte physiology and thus might affect voiding behavior. A study reported an increased OAB incidence in patients with hyperprolactinemia. However, the information with regard to the relationship between hyperprolactinemia and bladder function is limited.

Both the prolactin ligand and its receptors are normally expressed in human and rodent prostate. Previous study showed that increased levels of prolactin have significant stimulatory effects on prostate ductal development and lead to hyperplastic growth in the adult gland. Although hyperprolactinemia may induce prostate growth, the alteration of contractility of prostate under hyperprolactinemia is still unknown.

This paper investigated the effects of hyperprolactinemia on the in vivo voiding behavior and in vitro contractile function the bladder and prostate.

Study design, materials and methods

Male Sprague-Dawley rats (8 weeks old) were used in this experiment. Under light ether anesthesia, an incision was made in the left flank to expose the kidney. A small slit in the renal capsule was made to allow implantation of two anterior pituitary glands (AP) in the space beneath (AP group, n=8). Rats of the control group (cortex group, n=8) were implanted with a similar amount of brain cortex in a like manner. Seven weeks later, animals were put into the metabolic cage to record voiding frequency and voided volume. Then the animals were sacrificed and the bladder and urethra were obtained. Contractile responses of the bladder and prostate strips to electrostimulation, potassium chloride (KCL), bethanechol (bladder) and phenylephrine(prostate) were determined.

Results

Hyperprolactinemia did not have effect on the rat body weight and the weight of the prostate and the bladder (table 1). In vitro contractility, either contractile response to electrostimulation or pharmacological stimulation, of the bladder (table 2) or the prostate (table 3) was not significantly altered in the hyperprolactinemic rats. However, the urine amount of hyperprolactinemic rats during light cycle (sleep) was significantly lower than the control (table 4). Nevertheless, whole day voiding frequency and voided volume was not different between two groups (table 4).

Interpretation of results

The present study shows that contractile function of the bladder and prostate is not modified by high serum prolactin level. Since there is no documented evidence that prolactin receptor in the urinary bladder, it is reasonable to perceive that the contractile function of the bladder is not changed in hyperprolactinemic rats. Nevertheless, although prolactin receptor is present in the prostate, the function of prolactin receptor might relate to prostate growth or metabolism, rather than to contractile function of the prostate.

It has been demonstrated that kidney contains prolactin receptor and responds to prolactin with sodium and water retention and a reduced renal blood flow. In the present study, the reduced urine volume during light cycle might relate to persistent high serum prolactin level during light cycle.

Concluding message

These results indicate that prolactin does not affect bladder sensory and contractile function. However, owing to the anti-diuretic effect of prolactin, the urine volume of hyperprolactinemic rats is less during sleep. These results suggest that prolactin play a role in regulating nocturnal urine production.

Table 1. Animal Body weight, bladder weight and prostate weight (Mean ± SEM)

	Body wt. (g)	Bladder wt. (g)	Prostate wt. (g)
AP group	278.7 ± 3.2	0.1161 ± 0.0	0.5888 ± 0.1
Cortex group	280.2 ± 2.9	0.1392 ± 0.0	0.5461 ± 0.1

AP=anterior pituitary

Table 2. Bladder contractility gm(Mean ± SEM)

Group	ELC gm/g tissue	KCl gm/g tissue	Beth gm/ g tissue
AP group	318.1 ± 39.2	212.2 ± 24.4	312.1 ± 28.4
Cortex group	287.9 ± 36.9	196.9 ± 22.4	316.5 ± 36.2

AP=anterior pituitary; ELC= Electrostimulation; Beth=bethanechol

Table 3. Prostate contractility (Mean ± SEM)

Group	ELC gm/g tissue	KCl gm/g tissue	Phen gm/ g tissue
AP group	2.9 ± 1.0	1.4 ± 0.3	2.1 ± 0.5
Cortex group	2.0 ± 0.1	0.9 ± 0.2	1.4 ± 0.3

AP=anterior pituitary; ELC= Electrostimulation; Phen= Phenylephrine

Table 4. Urine volume and voiding frequency (Mean ± SEM)

Group	Urine volume(light cycle)	Urine volume(whole day)	Voiding frequency(whole day)
AP group	11.5 ± 1.4ml	27.4 ± 4.1ml	26.6 ± 1.8
Cortex group	22.6 ± 4.43ml*	43.2 ± 8.2ml	31.6 ± 4.0

AP=anterior pituitary

*= significantly different between two groups.

Disclosures

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