INTRAVESICAL PROSTAGLANDINS INSTILLATION IN ACUTE URINARY RETENTION IN FEMALES

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SUMMARY

Females with underactive detrusor function often present with acute urinary retention after the administration of sedatives, anaesthetics, pelvic surgery and childbirth. Urological investigations are normal in such cases and bladder exercises are usually ineffective to restore normal micturition. The presence of prostaglandins in the bladder washout in experimental studies has triggered our interest to perform this study and to determine the clinical usefulness of prostaglandin in urinary retention in females without having an obstructive pathology.

INTRODUCTION

We are at times confronted with cases of acute urinary retention in females whereas no cause of obstruction is found on routine urological investigations. In women, the underlying cause of acute urinary retention is usually under active detrusor function.[1] A provocative factor is usually present in these women which may be the administration of sedatives or anaesthetics, pelvic surgery and childbirth. The treatment of acute urinary retention in such cases is urethral catheterization. However, removal of catheter is followed by recurrent retentions. In a number of these cases, the sensory threshold for exteroceptive stimuli in the lower urinary tract is abnormally high[2].

Bladder exercises to regain tone in the flaccid detrusor muscle have been disappointing.

Recent interest of the investigators in the role of prostaglandins in the process of micturition have given us a new thought. Experiments on dogs have shown that prostaglandins were released into the blood stream during and immediately after bladder distension[3] (1976)[4] reported the results of initial investigations on the effects of prostaglandin on the bladder in vitro and in vivo. It was established by these experiments that;

a. Prostaglandins were produced by the detrusor.

b. Prostaglandin E_2 resulted in dose related contraction of the detrusor muscle strip.

c. Prostaglandin synthesis inhibitors resulted in loss of tone and activity which was then restored by exogenous prostaglandin.

Clinical studies were then performed, based on these experimental findings on dogs. However, the patients selected were in chronic urinary retention with symptoms primarily of impaired detrusor function. 72% of these patients showed objective evidence of an immediate improvement in detrusor function and a prolonged, therapeutic benefit in 39% by bladder instillation of prostaglandin.

The aim of this presentation is to determine the clinical usefulness of pros-

taglandin E_2 in acute urinary retention in females without an obstructive pathology.

MATERIAL AND METHODS

12 female patients with acute urinary retention were treated by instillation of intravesical prostaglandin E_2. All patients presented with an indwelling catheter which they stated causes retention whenever removed on trial bases. 9 out of 12 patients were in retention after childbirth and 3 patients underwent some gynaecological surgery.

All patients were routinely investigated by ultrasound study of the urinary tract to exclude any organic pathology or out flow obstruction. Instillation of intravesical prostaglandin was done after bladder wash with normal saline.

1.5 mg of prostaglandin E_2 diluted with 20 ml saline was infused into the bladder through the catheter followed by a further 5 ml to flush the drug out of the catheter lumen. The catheter was then clamped and finally removed one hour after instillation of the drug. The patient was allowed to go after she had satisfactorily passed urine spontaneously. They were instructed to report if symptoms redevelop.

RESULTS

All the twelve patients responded favorably. Only one patient complained urgency and frequency of micturition 2 weeks later which was treated by urinary antibiotics.

Follow up at one and three months interval was uneventful in all and ultrasound study did not show "residual urine in all".

DISCUSSION

Micturition can normally be initiated at will, with the bladder emptying to completion. Patients with acute urinary retention, however, are unable to void even with full or overful bladder. In females, the acute urinary retention is usually associated with underactive detrusor function in contradistinction to males where the underactive detrusor function is not a major cause of acute urinary retention.

In nearly all patients of acute urinary retention, a provocative even is present which might be the factor responsible to exhaust the underactive detrusor. Experimental findings of Gilmore and Vane (1971)[6]; Bultitude et al (1976); and Ambacke and Zar (1970)[5], proved that increased prostaglandin concentration in blood was associated with bladder distension in dogs; bladder wash out fluid showed increased prostaglandin like material after pelvic nerve stimulation[3] the detrusor muscle exhibited excitatory changes to exogenously applied prostaglandins[5].

Intravesical administration of prostaglandin in females with chronic urinary retention apparently due to inactive detrusor, resulted in return of detrusor function. These clinical studies were based on urodynamic assessment not only in females with chronic retention but also in the management of any patient whose urinary symptoms were considered the result of impaired detrusor function.

Patients in acute urinary retention in our hands have also shown dramatic and favourable response to intravesical prostaglandin E_2.

From all these observations, we can conclude that prostaglandins may sensitise the stretch or tension receptors of the bladder wall so that reflex micturition is triggered at a lower threshold volume during bladder filling, prostaglandin may also exert an inhibitory action on adrenergic transmission in the bladder. The effect is long term and the patient rarely needs repetition of the procedure.

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