

URINARY INCONTINENCE CAUSED BY PRAZOSIN

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ABSTRACT

Urinary incontinence is a common problem in elderly women. However, urinary incontinence secondary to the commonly used antihypertensive, prazosin, is a rare phenomenon. The possible mechanism of this drug in causing incontinence is described in this case report. The possibility of an exaggerated response to the drug by women suffering from borderline stress incontinence is highlighted.

Keywords: Urinary incontinence, prazosin, adrenergic receptor blockade.

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INTRODUCTION

Prazosin is a widely used drug in the treatment of hypertension. Its hypotensive effect is the result of peripheral vasodilation produced by alpha-adrenergic receptor blockade⁽¹⁾. The first dose phenomenon and postural hypotension is a well known side effect of prazosin. Sexual dysfunction and failure of ejaculation have also been noted in patients with prazosin, but less frequently. However urinary incontinence is a very rare side effect of this drug. We report a case of urinary incontinence in a woman taking prazosin. The possible mechanism of this drug in causing incontinence is also discussed.

CASE HISTORY

Mrs S K, a 62-year-old Para 4, presented at the gynaecological clinic of the University Hospital, Kuala Lumpur, with a 5-year history, suggestive of stress incontinence. The problem had become worse over the years. She was a known hypertensive and was on prazosin (minipress) 2 mg twice daily for the last 3 years. She was post-menopausal for 7 years.

There was nothing of significance in her previous obstetric history. She had 4 full term uncomplicated vaginal deliveries and her last child birth was 30 years ago.

On examination she was well. Her blood pressure was 160/90 mmHg and the pulse was 88 per minute. There was no pallor and she appeared well. There was no abnormality noted in the cardiovascular or respiratory system. Abdominal examination revealed no masses. Pelvic examination revealed a moderate cystocele. Stress incontinence was demonstrated clinically. There was no enterocele or rectocele. The cervix and uterus were atrophic. First degree uterine descent was noted. There were no adnexal masses. A diagnosis of first degree utero-vaginal prolapse with moderate cystocele and stress incontinence was made. She was referred to the physiotherapist

for pelvic floor exercise. However there was no improvement of the condition. Thus she was scheduled for a vaginal hysterectomy, anterior colporrhaphy with insertion of Kelly's stitch for repair of the stress incontinence. Pre-operative urine analysis showed no evidence of urinary tract infection and renal function tests were normal. The surgery was uneventful. Her post-operative recovery was good and she was discharged well on the 6th post-operative day.

She however presented a month later with history of recurrent stress incontinence. On examination stress incontinence was demonstrated, the severity of which was much less than when she presented to us initially. There was no evidence of any urinary tract infection. As there were no facilities available for urodynamic studies, she was treated for possible detrusor instability without much success. However the symptoms persisted. Local oestrogen cream to the vagina was applied in the hope of increasing urethral tone and even this was unsuccessful.

On reviewing the literature for persistent stress incontinence and the rare possibility of prazosin being the offending cause, it was discontinued and the hypertension was meanwhile treated with alpha methyl dopa 250 mg three times a day. She became continent soon after cessation of prazosin therapy. To ascertain whether prazosin was the actual cause of the stress incontinence, she was, after being informed of the possible problem, restarted on prazosin. The incontinence recurred. The prazosin was discontinued and she was recommenced on alpha methyl dopa. She has remained symptom free for about a year now.

DISCUSSION

Although first documented in 1978⁽²⁾, cases of prazosin induced urinary incontinence have been sparsely reported in the literature. The bladder and the urethra are innervated by both parasympathetic and sympathetic nerve fibres. Parasympathetic stimulation of the detrusor causes the emptying phase of bladder activity while sympathetically innervated smooth muscle of the urethra plays a significant role in the maintenance of urethral closure⁽³⁾. The sympathetic activity is mediated via the alpha-adrenoreceptors located in the proximal urethra and contributes significantly to the urethral pressure profile (see Fig 1). Thus alpha adrenoreceptor blocking agents, like prazosin, can precipitate incontinence in women.

Thien et al⁽²⁾ had reported a possible 10% incidence of urinary incontinence in patients on prazosin. This incidence however is high and may reflect inclusion of a susceptible group of patients, mainly older women, in whom mild degrees of urinary stress incontinence commonly occur. This could have been possible in our patient who did have partial relief from surgery. Withdrawal of prazosin led to immediate disappearance of symptoms on two occasions.

Elderly women with borderline stress incontinence (probably related to child-bearing, obesity and post menopausal

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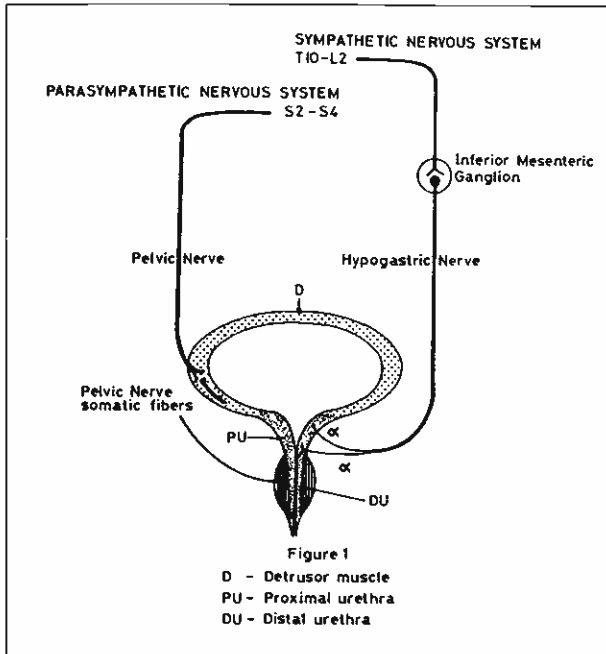
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Fig 1 - Bladder Innervation



D - Detrusor muscle
 PU - Proximal urethra
 DU - Distal urethra

change in pelvic musculature) may exhibit an exaggerated response to prazosin⁽⁴⁾. Thus it should be borne in mind that urinary incontinence secondary to prazosin may occur more frequently than is generally recognised. Should incontinence occur, reducing the dosage or withdrawing the drug will usually result in a return to continence.

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ANSWER TO ELECTROCARDIOGRAPHIC CASE

Diagnosis: Acute pericarditis.

DISCUSSION

The history is suggestive of acute pericarditis. The distinct clinical abnormality detected was a pericardial friction rub with systolic and diastolic components. The 2D echocardiogram demonstrated a small circumferential pericardial effusion. The ECG illustrates diffuse ST segment elevations. The differential diagnosis of ST elevations includes: artefactual of spurious causes (for example, baseline wander and artificially pacing), normal variants (the early repolarization pattern), transmural myocardial ischaemia or acute myocardial infarction, ventricular aneurysm, acute pericarditis and left bundle branch block. The ECG findings in this patient are however typical of the initial phase of the 4 stage sequences of an acute pericarditis as described by Spodick⁽¹⁻⁴⁾. There is diffuse ST segment elevations in leads I, II, aVF and V2-6 without reciprocal ST segment depression as those sometimes seen in acute myocardial infarction. Typically the ST elevation is concave upwards and is higher in lead II than in III as is seen in this case. There is also associated ST segment depression in aVR and VI. These findings may sometimes be mistaken as an acute inferior and anterior myocardial infarction. This is unlikely from the clinical feature as a patient with such extensive infarctions will usually be in cardiogenic shock. There are also no Q waves abnormalities which are usually seen in acute myocardial infarctions. The ST segment elevations of acute myocardial infarction are usually also more marked and is associated with T wave inversion usually⁽⁵⁾. The presence of PR

segment depression which are best seen I, II, aVF and V3-6 with PR segment elevation in aVR are also very characteristic of acute pericarditis⁽⁶⁾. The absence of symptoms and failure to evolve through the various stages of ECG changes as described by Spodick for acute pericarditis helps to differentiate it from the early repolarization variants in normal individuals⁽⁷⁻⁹⁾. Ginzton⁽⁷⁾ additionally suggests that an ST/T ratio ≥ 0.25 in V6 discriminated patients with acute pericarditis from normal variants. The repeat ECG in this patient performed 3 months later showed complete resolution of the ST and PR segments changes with no development of Q waves. Note however that atypical variants may sometimes occur⁽¹⁰⁾, simulating acute myocardial infarction or Prinzmetal's angina.

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