



Acute urinary retention associated with flecainide

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■ A case of acute urinary retention, presumably secondary to the use of the antiarrhythmic drug flecainide, is presented. A mechanism for this effect is proposed based on the possibility of a local anesthetic effect on bladder mucosa.

□ INDEX TERMS: ANTI-ARRHYTHMIA AGENTS; URINATION DISORDERS □ CLEVE CLIN J MED 1990; 57:86-87

WE PRESENT a case of acute urinary retention associated with flecainide. This antiarrhythmic agent has not, to our knowledge, been previously described as a cause of acute urinary retention. A local anesthetic effect seems possible.

CASE REPORT

A 74-year-old man with severe diffuse atherosclerotic heart disease was admitted to the hospital for control of ventricular tachycardia. His medications included isosorbide, digoxin, captopril, and furosemide, which he had taken chronically without difficulty. His previous antiarrhythmic therapy had consisted of successive courses of quinidine, procainamide, tocainide, and mexiletine. Each antiarrhythmic medication had been associated with significant gastrointestinal side effects; this led to poor compliance with the drug regimen so that he had been taking no antiarrhythmic agents for

four months prior to admission.

Prior to admission he had a syncopal episode. Monitoring in the hospital revealed episodes of ventricular tachycardia. Urologic history was significant for a remote transurethral resection of the prostate, though he had been voiding without difficulty for several years up to the time of admission. Specifically, he described his urinary flow as being excellent.

The patient was started on flecainide, 50 mg twice daily. A diminished urinary stream was noted 48 hours later and complete urinary retention developed within four days. Physical examination at that time revealed a distended bladder though the patient was not uncomfortable. Rectal examination revealed a small 15-g benign prostate.

Further studies were performed, including a cystometric and cystoscopic evaluation. This revealed a normal capacity of 360 mL. There was no evidence of a neurogenic lesion, however, no detrusor reflex was elicited. At cystoscopy, there was no urethral stricture. The prostatic fossa was well resected and the bladder neck was open. The patient was treated with intermittent catheterization. The flecainide was discontinued, and within 48 hours the patient began voiding spontaneously. Post-void residuals were checked and were consistently less than 25 mL. He has remained free of voiding difficulties since that time.

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DISCUSSION

Flecainide is a Class Ic antiarrhythmic drug.^{1,2} Like all Class I antiarrhythmics, the action is based on sodium-channel blockade. All Class I antiarrhythmics ultimately result in the depression of myocardial conductivity and excitability.³

Class I antiarrhythmics include lidocaine (Ia), quinidine, procainamide, and disopyramide (Ib), and flecainide (Ic).² All have similar side effects of gastrointestinal upset, visual disturbances, and vertigo. All Class I antiarrhythmics have local anesthetic properties, but only disopyramide has been reported to cause urinary retention.⁴ Urinary retention is thought to be secondary to the anticholinergic properties of disopyramide, as disopyramide-induced retention can be reversed with pyridostigmine, an acetylcholinesterase inhibitor.⁴ While a review of the literature reveals no cases of urinary retention associated with flecainide, there clearly appears to be a relationship between flecainide use and urinary retention in the patient reported here.

Detrusor activity is mediated primarily via the parasympathetic nerves S-1 through S-3. It is the net response to the integration between the sensory input

from the bladder and the modulating effect of the suprasacral centers. Thus, any derangement in sensory or motor function to the bladder, or integrative center thereof, may result in detrusor dysfunction. This results in either failure to store urine or failure to empty the bladder.

The pathophysiology of flecainide-induced urinary retention is not clear. However, 86% of the drug is excreted in the urine as flecainide or one of its metabolites.¹ It is possible, then, that a direct, local anesthetic action on bladder mucosa may block normal sensation and the detrusor reflex. Such a mechanism would be consistent with the painless retention noted in our patient. Alternatively, an anticholinergic-like effect of the drug on a metabolite may be a possible mechanism leading to urinary retention.

CONCLUSION

We have identified a case of flecainide-induced acute urinary retention. While flecainide has proven to be a useful antiarrhythmic agent, it is not without side effects. Flecainide needs to be added to the list of drugs implicated in urinary retention.

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