independent of the diuretic and naturetic response. This vasodilator action is probably mediated by local prostaglandins, kinin and kallikrein activity. Pharmacologically, bumetanide is 40-fold more potent than frusemide on a weight for weight basis. The $K^+$ losses are less with bumetanide.

The role of bumetanide is acute pulmonary oedema and congestive heart failure is well established. Bailey has described the use of bumetanide in 10 patients 2 months after their renal transplants and no serious side effects or biochemical abnormalities occurred. As far as we are aware, this is the only documented report of the use of bumetanide in renal transplant patients. We recommend its use at an early stage in the cadaveric renal transplants to obtain diuresis.

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References


Pseudo-obstruction secondary to anticholinergic drugs in Parkinson's disease

Sir,

We would like to report a case of pseudo-obstruction in a patient with Parkinson’s disease who was taking lofepramine and oxybutinin. Neither drug has been reported to cause this complication, and thus this case illustrates the difficulty in treating patients with Parkinson’s disease with anti-cholinergic drugs.

A 60 year old man with a 12 year history of Parkinson’s disease, presented with urinary frequency, incomplete voiding, and increasing depression. The main features of his Parkinson’s disease were rigidity, marked dyskinesias, severe ‘on–off’ fluctuations and constipation. Drugs on admission were cocomalamp, co-beneldopa, subcutaneous apomorphine as required, domperidone, lactulose, frusemide, digoxin, nifedipine and aspirin.

Videocystometry showed an unstable bladder for which he was treated with oxybutinin 5 mg twice a day and 10 mg at night.

Lofepramine was started 3 days later, at a dose of 70 mg at night, for depression. Three days after this, the patient went into acute urinary retention and needed catheterization. The oxybutinin and lofepramine were stopped. Twelve hours later he was found to be hypotensive and oliguric. On examination the abdomen was distended and tender, bowel sounds were absent and the rectum was empty. Abdominal radiographs showed dilated large and small bowel, with air throughout the bowel. A single contrast barium enema showed free flow up to the caecum. Amylase, sodium and potassium were normal.

A diagnosis of pseudo-obstruction was made and treatment was conservative consisting of intravenous fluids and a nasogastric tube. After 5 days he had made a full recovery. All previous medication except the lofepramine and oxybutinin were reintroduced without recurrence of the pseudo-obstruction.

The temporal relationship between the administration of lofepramine and oxybutinin and the bowel obstruction strongly suggests that one or both drugs played a pathogenic role. It is of interest that lofepramine is widely thought of as having markedly less anti-cholinergic side effects than other tricyclic antidepressants. Nevertheless, all anti-cholinergic drugs produce smooth muscle relaxation which leads to bowel stasis and therefore may predispose to pseudo-obstruction. This is a relatively rare complication and is usually an idiosyncratic reaction.

Megacolon and dilatation of the small bowel have been described in patients with Parkinson’s disease not taking anticholinergic medication. Contributory factors in parkinsonian patients include disordered gut motility with generalized hypokinesia, delayed gastric emptying aggravated by levodopa, autonomic dysfunction and aggravation of constipation by anismus. Furthermore, Kupsky et al. have shown the presence of Lewy inclusion bodies in myenteric plexus ganglion cells in the oesophagus and colon in a patient with megacolon and Parkinson’s disease. This may reflect primary involvement of the enteric nervous system by degenerative processes in Parkinson’s disease. Thus, it can be seen that disturbances of alimentary function are a prominent feature in patients with Parkinson’s disease and may make these patients particularly vulnerable to the effects of anti-cholinergic drugs on the bowel.

This case therefore illustrates the difficulty in treating patients with Parkinson’s disease with anticholinergic drugs. Particular care is advised, when prescribing them to patients with Parkinson’s disease.

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