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(Accepted 30 June 1982)

Endocrine Unit, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS

JOHN C STEVENSON, MB, MRCP, honorary lecturer and senior registrar
MICHAEL C WHITE, MB, MRCP, research fellow
GRAHAM F JOPLIN, PHD, FRCP, reader
IAIN MACINTYRE, DSC, FRCP, professor

Death due to overdose of indoramin

Indoramin (Baratol) is a recently introduced α_1 -adrenergic antagonist used to treat essential hypertension. I report a fatal case of self-poisoning with indoramin. This is the first such case to be described.

Case report

A 43-year-old housewife was admitted to hospital after being found unconscious at home. She had a seven-year history of essential hypertension and was being treated with indoramin and cyclopenthiadiazide. She was also taking temazepam for night sedation. In 1976 she had been a psychiatric inpatient after taking an overdose of Moduretic and ethanol and self-mutilation of her wrists. One week before admission she had become depressed and was drinking more alcohol than usual. On the day of admission she had prepared the evening meal and then gone to her bedroom; she was found one hour later unconscious. She had taken approximately 100 tablets of 25 mg indoramin.

On admission she was unconscious but responded to pain and was breathing spontaneously. Pulse was 90 beats/min and regular and blood pressure 120/60 mm Hg. The pupils were equal and reacted to light. She was areflexic. Shortly after arrival she had a series of generalised convulsions that were treated with intravenous diazepam. A cuffed endotracheal tube was inserted and gastric lavage carried out. Only a few indoramin tablets were identified in the lavage. Blood pressure fell rapidly to 60/0 mm Hg and she was given intravenous plasma and the foot of the bed was raised. Further inotropic support was achieved using intravenous dopamine by sage pump. Severe metabolic acidosis (pH 7.04, oxygen pressure 65 mm Hg (8.7 kPa), carbon dioxide pressure 48 mm Hg (6.4 kPa), base excess -18 mmol(mEq)/l) was corrected with sodium bicarbonate. Urea, electrolyte, and blood glucose concentrations were within normal limits. Blood was taken for poisons analysis. Over the next hour blood pressure rose to 130/80 mm Hg and electrocardiography showed a sinus tachycardia of 105 beats/min. Urinary output was satisfactory and her temperature stable at 36.9°C . Despite adequate haemodynamic control she remained comatose and continued to have further convulsions. Respiratory depression ensued and she required ventilation. Her condition deteriorated further with the onset of bradyarrhythmias unresponsive to atropine and isoprenaline. Cardiac arrest occurred and she died six hours after admission to hospital.

At necropsy the only finding of note was moderate pulmonary oedema. The results of the poisons analysis confirmed high concentrations of indoramin and ethanol (6.44 and 2250 mg/l respectively) but only therapeutic concentrations of temazepam and oxazepam (0.16 and 0.03 mg/l); paracetamol, barbiturate, and salicylate were not detected. Plasma indoramin concentration after an oral dose of 100 mg is 0.03-0.12 mg/l; thus the concentration in this patient was approximately 50-200 times greater than therapeutic values.

Comment

Indoramin is being increasingly used in the treatment of essential hypertension, yet little is known about its toxicology in man. It is well absorbed by mouth, and peak plasma concentrations occur one to two hours after ingestion.¹ It is thought to act by selective antagonism of postsynaptic α_1 -adrenergic receptors that normally mediate vasoconstriction.²

This is the first reported case of self-poisoning with indoramin. The main clinical features were deep sedation, respiratory depression, hypotension, and convulsions. Although the hypotension was satisfactorily controlled, the effects on the central nervous system were

resistant to treatment and proved fatal. There are no previous reports of indoramin-induced convulsions in man, but fits have been observed in animals given high doses (P K Marrott (Wyeth Laboratories), personal communication). The serum ethanol concentration, though raised, is unlikely to have accounted for the severity of the clinical state.

Treatment of indoramin poisoning should include prompt gastric lavage, circulatory support to control hypotension, and diazepam for convulsions. Although no specific antidote exists, infusion of α -adrenoceptor agonists, such as noradrenaline or metaraminol, may prove beneficial.

Sedation³ and depression⁴ are recognised central side effects of indoramin. This case shows that indoramin taken in overdose is fatal. It would therefore seem wise to exercise caution before prescribing indoramin to patients with a history of depressive illness.

I thank Dr P K Marrott of Wyeth Laboratories and Dr G N Volans of the Poisons Unit, New Cross Hospital, London for help with the poisons analysis.

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(Accepted 15 July 1982)

Southern General Hospital, Glasgow G51 4TF

ROBERT HUNTER, BSC, MB, medical senior house officer

Cheap and effective method of weighing bed-bound patients

Recording a patient's weight is easy in ambulant or semi-ambulant patients, but seriously ill patients pose a greater problem. Most of the existing devices capable of lifting and weighing a normal hospital bed are bulky, difficult to manoeuvre, and expensive. We have developed a simple, inexpensive, and easily transportable device for weighing bed-bound patients. Most of the patients were receiving nutritional support, and consequently we wished to chart their weight throughout their treatment.

Method

The technique requires use of four sets of bathroom scales and one stretcher. One set of scales is placed under each leg of the bed on which the patient is lying, the scales having been previously "zeroed." (To maintain the bed leg in a constant position on the scales a furniture leg "load spreader" is glued to the centre of the scales.) A reading is then taken from each of the scales and the four values summed. This gives a value for bed plus patient. A variable-height trolley with detachable canvas portion (Institutional Industries, Cincinnati, Ohio) is then used to raise the patient clear of the bed: the canvas portion is placed under the patient, the tubular frame manoeuvred into position so that the canvas can be reattached to the frame, and the trolley then wound up so that the patient is lifted clear of the bed. Drainage bags, etc, may be left in situ as the patient need be lifted only a few inches. Consequently only the patient, and not the apparatus and drained fluid, is weighed (figure). Once the patient has been raised clear of the bed the four scales are read again and the readings summed. This total represents the weight of the bed alone. By subtracting the bed alone value from the bed plus patient value the patient's weight may be calculated.

To test the accuracy of our method we conducted a trial with 20 ambulant volunteers. Five different hospital beds were used. For each volunteer we derived two readings, one for bed alone and one for bed plus patient, and then calculated the volunteer's weight using our simple equation. The scales were removed and zeroed if required. The volunteer was then weighed on each of the four scales. The readings were taken by a second observer, who was unaware of the volunteer's calculated weight. The calculated average reading represented the volunteer's actual weight.

The calculated weight was less than the actual weight in 15 of the 20 subjects and greater in four; in one case the two weights were identical. The range of error was -1.6 to $+0.9$ kg, with a mean of 0.36 kg; this error was not significant (Student's *t* test).

Scales and variable-height trolley being used to weigh patient.

Comment

By using this method the weight of a bed-bound patient may be measured with an average error of 0.36 kg. We believe that this is an adequate method of assessing the trend of a patient's weight even during a period of immobilisation. This is of particular value to any clinician who must provide nutritional support for the patient.

In the present atmosphere of restricted budgets the cost of weigh-beds or even "sitting scales" may be prohibitive, especially for smaller hospitals. All hospitals, however, would have the equipment needed for the method described here.

We thank John N Livingstone of Vicarey Davidson & Co, surgical instrument makers, for supplying the scales.

(Accepted 23 July 1982)

Department of Surgery, Victoria Infirmary, Glasgow G42 9TY

DOUGLAS T HANSELL, FRCS, registrar in surgery
GAVIN D SMELLIE, FRCS, consultant surgeon

Maintenance of remission in ulcerative colitis with oral preparation of 5-aminosalicylic acid

Sulphasalazine (salazopyrin) is the most effective agent for maintaining remission in ulcerative colitis,¹ but its use is limited by side effects. It consists of sulphapyridine and 5-aminosalicylic acid joined by a diazo bond, which is split by bacteria in the colon releasing the constituents.² 5-Aminosalicylic acid is the active compound that probably has a topical effect,³ while sulphapyridine serves as a carrier molecule and is probably responsible for the side effects.

5-Aminosalicylic acid cannot be given by mouth since it is unstable in acid and is absorbed by the small intestine. We recently described a colonic delivery system whereby drugs coated with an acrylic-based resin were carried intact to the colon and then released.⁴ This study compares 5-aminosalicylic acid coated in this way with sulphasalazine for maintenance of remission in colitis.

Methods and results

We studied 72 patients (36 men, 36 women). All were in remission with ulcerative colitis or proctitis and had passed three or less stools daily without blood or slime during the previous month; were taking at least 2 g salazopyrin daily; and gave their informed consent. Admission to the trial depended on normal sigmoidoscopic findings. Rectal biopsy specimens were taken, coded,

and later reviewed by a pathologist, who graded them from 1 (normal) to 2 to 4 for mild, moderate, or severe inflammation.

Patients were seen after four, 12, and 16 weeks and completed diary cards throughout. Sigmoidoscopic examination was repeated at the end of the trial and in all patients with recurrent symptoms. Relapse was defined as a recurrence of symptoms with sigmoidoscopic changes, including contact or spontaneous mucosal haemorrhages, pus, or ulceration.

The study was randomised and double blind, using identical placebo tablets for both sulphasalazine and 5-aminosalicylic acid. All patients were given two sets of tablets, either active 5-aminosalicylic acid and placebo sulphasalazine, or placebo 5-aminosalicylic acid and active sulphasalazine. Trial tablets were based on the patient's usual dose of sulphasalazine. Tablets of 5-aminosalicylic acid contained 400 mg, equivalent to 1 g sulphasalazine; at least three tablets of 5-aminosalicylic acid were taken daily with an increase of one tablet for each gram of sulphasalazine above the entry dose of 2 g for patients taking high doses of sulphasalazine. Compliance was checked at each hospital visit. 5-Aminosalicylic acid powder was obtained from Aldrich Chemicals and the tablets coated with an acrylic-based resin (Eudragit S) between 100 and 130 μ thick.⁵

Five of the 72 patients (four women, one man) were withdrawn because of pregnancy in two, constipation in one, and failure to comply in two. Of the remaining 67 patients, 15 relapsed and 52 completed the trial (table). Nine

Details of patients treated with 5-aminosalicylic acid or sulphasalazine during 16-week trial

	5-Aminosalicylic acid	Sulphasalazine
No of patients	34	33
Sex (M:F)	14:20	21:12
Mean ($\pm$ SD) age (years)	44.9 $\pm$ 15.3	50.2 $\pm$ 15.6
Mean ($\pm$ SD) duration of disease (years)	7.2 $\pm$ 5.5	9.3 $\pm$ 6.4
Mean ($\pm$ SD) time since last attack (years)	1.6 $\pm$ 1.4	2.1 $\pm$ 2.3
No of patients with:		
Proctitis	17	19
Left-sided disease	13	5
Extensive disease	4	9
No of patients relapsing	9	6

patients taking 5-aminosalicylic acid and six taking sulphasalazine relapsed; the difference was not significant (χ^2 test).

Headache, nausea, or indigestion were reported by 16 patients, with no difference between groups. Satisfactory biopsy specimens from 61 patients confirmed the absence of inflammation in most (51). Four showed grade 2 and six grade 3 changes.

Comment

This study showed that 5-aminosalicylic acid coated with an acrylic-based resin (Eudragit-S) was as effective as sulphasalazine in maintaining remission in colitis. In our patients relapses occurred over 16 weeks, an adequate period since previous studies have shown that most relapses occur within 12 weeks.¹

This preparation is an important advance in the management of colitis since it may be given to patients unable to take sulphasalazine and greater doses may be tolerated.

We thank Regent Laboratories, who produced both active and placebo tablets of 5-aminosalicylic acid, and Boots Pure Drug Company, Nottingham, for placebo sulphasalazine tablets.

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(Accepted 20 July 1982)

Department of Gastroenterology, University Hospital of Wales, Cardiff CF4 4XW

M J DEW, MD, MRCP, senior medical registrar
P HUGHES, MB, CHB, research registrar
A D HARRIES, MA, MRCP, research registrar
G WILLIAMS, MRCPATH, senior lecturer in pathology
B K EVANS, PHD, principal pharmacist
J RHODES, MD, FRCP, consultant physician