SHORT REPORTS

Assessment of severity of paraquat poisoning

Gramoxone (20% paraquat) causes death in 70% of patients who take it by mouth. In contrast, Weedol (3% paraquat) causes death in only 10%.[1] Though there is a simple screening test for the presence of paraquat in urine, which allows a history of ingestion to be confirmed, there is no method of assessing the severity of the episode and therefore of predicting the clinical outcome. We report here a correlation between the clinical outcome of paraquat poisoning and the excretion rate of paraquat in urine.

Patients, methods, and results

Over two years 16 patients who had poisoned themselves with paraquat were referred from hospitals throughout the West Midlands to the regional poisoning treatment centre. Fourteen of the patients were men and the mean age was 44·8 years (range 21-72 years; table). Gastric aspiration and lavage were performed in all cases. Fuller's Earth (250 ml 30% solution) and magnesium sulphate (20 ml 40% solution) were then given by mouth every six hours. Three patients (cases 11, 12, and 16) were treated by haemoperfusion through a charcoal column and four (cases 2, 3, 9, and 13) by forced diuresis.

All six of the patients who took Gramoxone died, but nine of the 10 who ingested Weedol survived. Four of those dying did so within 96 hours of ingestion; the remainder lived for up to 12 days. All patients who died showed evidence of liver and renal damage. Four survivors developed mild liver damage and two mild impairment of renal function. All survivors finally made a complete physical recovery.

Toxicology—Paraquat was assayed by colorimetry after extraction with an ion-exchange resin.[2] The procedure is easy to perform but takes two hours to complete. Total amounts excreted ranged from 0·6 mg to 386 mg. Hourly excretion rates were calculated for urine collected during the first few hours after admission (see table). Assays of sequential urine samples showed that the excretion rate of paraquat fell rapidly during the first 48 hours, although it diminished less rapidly in those who eventually died. The patient (case 12) who developed oliguria during the first 24 hours had a massive amount of paraquat in his urine.

All patients who excreted 1 mg/h or more of paraquat eight hours or more after ingestion died. One patient (case 3) excreted 1·1 mg paraquat per hour between four and a half and seven hours after ingestion, but the excretion rate then fell rapidly.

Comment

Paraquat, although taken by very few self-poisoned patients, is a significant cause of death in patients admitted to hospital. During the first 8-10 hours after ingestion, and despite the administration of Fuller's Earth, absorption is probably continuing. Excretion rates probably reflect plasma concentrations and can be interpreted only with reference to the length of time after ingestion. Though it has been claimed that urine assays are not helpful in the early assessment of poisoning,[3] we have found that a high mortality is associated with excretion rates of over 1 mg/h more than eight hours after ingestion. We do not have enough data to predict a "safe" excretion rate at 24 hours. Neither haemoperfusion nor forced diuresis appeared to affect the excretion rate appreciably. Until a rapid, easy, and reliable blood assay is available, urinary excretion rate is the only way of assessing severity of poisoning.

Details of self-poisoning with paraquat and outcome

Case No	Age and sex	Formulation and (estimated amount of paraquat ingested (g))	Outcome	1st urine collection			
				Time started after ingestion of paraquat (h)	Duration (h)	Volume (ml)	Paraquat excretion rate (mg/h)
1	42 M	Weedol (0·4)	Survived	6	4	460	0·2
2	42 M	,, (0·5)	,,	8	3	520	0·15
3	55 M	,, (1·8)	,,	4·5	2·5	210	1·1
4	50 F	,, (0·9)	,,	11	24	1300	0·05
5	24 M	,, (0·1)	,,	5·5	16·5	1800	0·03
6	54 M	,, (0·1)	,,	12	14	2600	0·12
7	33 M	,, (1·8)	,,	10	36	840	0·16
8	62 F	,, (1·1)	,,	0	23	560	0·11
9	42 M	,, (1·9)	,,	0	19	4200	0·34
10	57 M	,, (1·8)	Died	12	10	850	1·0
11	68 M	Gramoxone (3·0)	,,	6	4	1025	32
12	74 M	,, (10·0)	,,	0	4	545	24
13	21 M	,, (5·0)	,,	12	2	425	1·0
14	43 M	,, (10·0)	,,	10	2	315	4·3
15	53 M	,, (19·0)	,,	8	2	280	>1·5
16	34 M	,, (8·0)	,,	7	6	360	1·1

[1] Park, J, Proudfoot, A T, and Prescott, L F, *Clinical Aspects of Paraquat Poisoning*, ed K Fletcher, p 46. Macclesfield, Imperial Chemical Industries Ltd, 1977.
[2] Berry, D J, and Grove, J, *Clinica Chimica Acta*, 1971, **34**, 5.
[3] *Lancet*, 1976, **1**, 1057.

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Dudley Road Hospital, Birmingham B18 7QH
N WRIGHT, MB, FRCPED, director, regional poisoning treatment centre
W B YEOMAN, FRIC, FRCPATH, director, regional toxicology laboratory
K A HALE, BPHARM, senior scientific officer, regional toxicology laboratory

Whole gut irrigation: a new treatment for constipation

Faecal stasis, constipation, and rectal impaction are grave problems for elderly patients and their attendants. Faecal impaction leads to faecal incontinence, which may necessitate admission to hospital. Despite the seriousness of the problem, doctors tend to regard it lightly and to consider it a matter for the nursing staff.

When first referred many elderly patients are severely constipated, often with a heavily loaded pelvic colon and rectum. The overloading sometimes extends as far back as the ascending colon and even the caecum. The clearing of these heavily laden bowels and the re-establishment of a more normal transit time and bowel activity pattern is difficult, unpleasant, and exhausting. In an effort to find a more successful and less humiliating process than the customary daily enema, we decided to try the technique of whole gut irrigation used by gastroenterologists for preparing the bowel for surgery and colonoscopy.[1 2] Some of the reported patients were elderly yet tolerated the procedure well. This procedure has also been used in severely constipated patients before operation.[1]

Methods and results

After diagnosing faecal stasis from the history, rectal examination, and plain radiographs of the abdomen, we gave a softening agent for five days before the irrigation. The technique was explained to the patient. A nasogastric tube was passed. Separate intravenous injections of frusemide 40 mg and metoclopramide 10 mg were given. Isotonic saline at roughly body

temperature was infused at a rate of 2·5-3 l/h. The patient was then seated on a cushioned rubber ring on a commode in a secluded, warm, and well-ventilated room. The infusion was continued until the effluent started to clear. Any vomiting was usually relieved by further metoclopramide. A check abdominal radiograph was taken the next morning.

Seventy patients (30 men, 40 women) with a mean age of 80 years (range 65-95 years) have been treated by this technique. Irrigation lasted a mean of 3·4 hours (range 1·4-5·2 hours), and a mean of 8 litres of irrigant was used in each case (range 3-20 litres). The mean weight change was an increase of 1 kg (range −3·0-+5·9 kg). In seven patients the technique failed: in one material was so impacted that no motion was passed, and six continued to vomit despite the metoclopramide. There were no significant changes in haemoglobin, urea, or electrolyte concentrations or in osmolality. No patients who were in cardiac failure were treated although several had been in cardiac failure previously and were receiving treatment. The one patient who was so impacted that the technique failed managed to cope with the fluid load by renal excretion.

Comment

This technique will need further evaluation in geriatric practice, but our elderly patients seemed to tolerate it well. This technique is especially useful for patients who have right-sided faecal stasis as shown by radiography. Caecal stasis seems to be much commoner in the elderly than suggested by Dresen and Kratzer.[3] The caecum normally contains only liquid material, but in the elderly it is quite common to find stippled solid faecal shadows in this area on the radiograph, and repeated enemas may fail to shift this material.

Prevention ought to be our main aim and constipation may be avoided by using adequate fibre-containing diets and encouraging physical activity, adequate fluid intake, and regular bowel habit, augmented when necessary by carefully chosen laxative regimens. But when severe constipation and faecal stasis are found whole gut irrigation is a useful adjunct to treatment in elderly patients, particularly those with right-sided stasis, and an alternative to daily enemas, which may in any case be ineffective.

We thank Professor James Williamson for his advice and Sister MacPherson and nursing staff for their co-operation.

[1] Crapp, A R, *et al*, *Lancet*, 1975, **2**, 1239.
[2] Hewitt, J, *et al*, *Lancet*, 1973, **2**, 337.
[3] Dresen, K A, and Kratzer, G L, *Journal of the American Medical Association*, 1959, **170**, 644.

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University Department of Geriatric Medicine, City Hospital, Edinburgh EH10 5SB
R G SMITH, MB, MRCP, senior lecturer
JANE E J CURRIE, RGN, research nurse

General Surgical Unit, Western General Hospital, Edinburgh
A D F WALLS, MB, FRCS, senior registrar

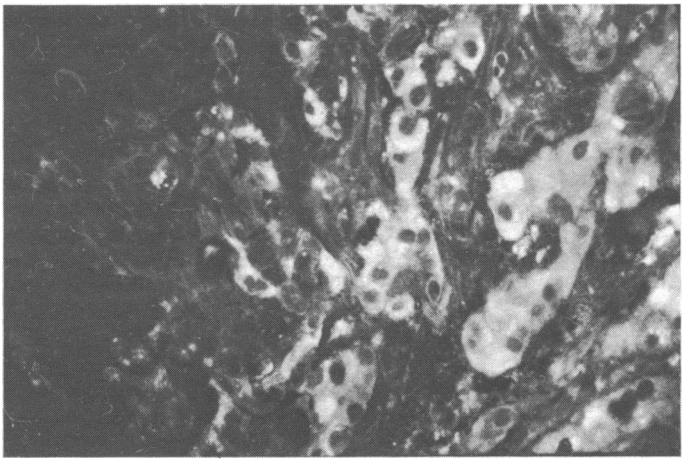
Ileal loop carcinoma after cystectomy for bladder exstrophy

Exstrophy of the urinary bladder is an uncommon congenital anomaly for which cystectomy is recommended because of a high risk of developing carcinoma.[1] We report a patient treated by cystectomy and ileal loop diversion, who, 20 years later, developed carcinoma of the ileal loop. The tumour was associated with hypercalcaemia and production of human chorionic gonadotropin (HCG), α-fetoprotein (α-FP), and carcinoembryonic antigen (CEA).

Case report

A 28-year-old man was admitted to hospital with cholestatic jaundice, polyuria, anorexia, constipation, and weight loss. At birth he had severe bladder exstrophy and epispadias. Various repair operations had been performed, including cystectomy and transplantation of ureters into the rectum at the age of 4 years. Four years later the anus had been sutured because of rectal prolapse and incontinence and the ureters retransplanted into an isolated ileal loop fashioned into an ileostomy.

He was confused, wasted, and severely jaundiced. His liver was hard, irregular, and enlarged 3 cm. Blood concentrations of urea were 14 mmol/l (84 mg/100 ml), potassium 5·6 mmol(mEq)/l, alkaline phosphatase 78 KA units/100 ml (normal 3-13 KA units/100 ml), calcium 3·4 mmol/l (13·6 mg/100 ml) (normal 2·1-2·6 mmol/l (8·4-10·4 mg/100 ml)) and phosphorous 1·6 mmol/l (5·0 mg/100 ml) (normal 0·8-1·4 mmol/l (2·5-4·3 mg/100 ml)). On radioimmunoassay α-FP was 210 μg/l (normal <40 μg/l) and CEA 7·8 μg/l (normal <2·5 μg/l). The plasma β subunit of HCG was raised at 1080 IU/l. Immunoreactive parathyroid hormone was low at 0·56 μg/l (normal 1·0-9·0 μg/l). A radiotechnetium liver scan showed multiple filling defects. A liver biopsy specimen showed invasion of liver tissue by metastatic pleomorphic anaplastic tumour with transitional, squamous, and mucin secreting areas. In some areas tumour cells reacted specifically with antibodies to β-HCG (see figure) and showed moderate argyrophilia with Grimelius silver impregnation technique. Anuria supervened and the patient died from renal failure 10 days after admission.



Immunofluorescent tumour cells reacting specifically with antibodies to HCG.

Necropsy showed an ulcerated tumour in the proximal end of the ileal loop close to the insertion of the ureters. The tumour was infiltrating part of the sigmoid colon adherent to the loop. Metastases were seen in the para-aortic and mediastinal lymph nodes, peritoneum, liver, pancreas, and lungs. The microscopical appearances of the tumour and metastases were similar to those seen in the liver biopsy. Multiple sections of the tumour and adjacent structures confirmed the tumour's origin in the ileal loop with secondary invasion of the colon. No urinary bladder tissue was found and the ureters, parathyroid glands, and both testes were normal.

Comment

This is apparently the first report of a carcinoma developing in an ileal conduit constructed after cystectomy for a benign bladder lesion. A few similar tumours have been reported after cystectomy for malignant tumours,[2 3] but these are usually thought to represent tumour recurrence. Smith and Hardy[4] reported a carcinoma developing in the ileal part of an ileocystoplasty; we cannot accurately compare our case with theirs, however, because the function performed by the ileal tissue was different in each case. On the other hand, 42 cases of large bowel neoplasms were reported after ureterosigmoidostomy;[5] 34 ureterosigmoidostomies were performed after cystectomy for benign bladder lesions. Probably, therefore, the mechanisms implicated in the development of such colonic tumours may also operate in ileal conduits. The difference in incidence may be due to the difference between colonic and ileal mucosae in their susceptibility to develop neoplasms or it may be an apparent difference due to the relative late start of using ileal conduits in treating such patients.

The multiple secretory potentials of the tumour may be related to the unusual circumstances in which it developed. Future immunological studies on patients with similar lesions, or at risk, might help in explaining the pathogenesis.

Early detection of these tumours can be achieved by regular cytological screening of urine.[2]

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