overall there were fewer cases than expected (male, 19 cases *v* 31·2 expected; female, 71 cases *v* 85·0 expected). Within the parishes that formed the Copeland area around Sellafield nine cases were registered compared with 18·4 expected.

Comment

The decrease in radioactivity with distance from Sellafield is probably due to several factors, including the pattern of distribution of milk, which is generally assumed to be the principal source of ^{129}I. Several points emerged from the study. Firstly, the specific activities varied widely even within the same residential area. This could be due to natural variation and differing diet as well as to the varying amounts of time people spend away from their homes. Secondly, the activities were low, most being under 10 mBq/g dry weight and none above 22 mBq/g. This indicates that most of the sample population received less than an additional 0·67 μSv/year from this source,[5] and none are likely to have received more than an additional 1·5 μSv/year. This is small compared with the dose to the thyroid from normal background radiation, which in the Sellafield area is about 1000 μSv/year. Any cancers induced by this additional dose of ^{129}I are therefore unlikely to be detected above the natural incidence of the disease.

Even if higher levels of radioactivity occurred in the past these have not produced any detectable increase in the incidence of thyroid cancer. Two parishes with a significantly increased incidence were some distance from Sellafield; the increased incidence would therefore seem to be unrelated to any radioactive discharge from the nuclear fuel reprocessing works.

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Extension of selection criteria for extracorporeal shock wave lithotripsy for gall stones

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Extracorporeal shock wave lithotripsy and dissolution treatment for gall stones have yielded encouraging results.[1,2] Selection criteria, however, seem arbitrary and based on those already established for treatment with bile salts.[3] As such criteria may restrict the number of patients treated we therefore extended the criteria and report our experience.

Patients, methods, and results

We treated 62 patients as outpatients between August 1987 and June 1988 (12 male, 50 female; mean age 46 (range 11-81)). Patients were selected if they had (*a*) symptomatic gall stone disease; (*b*) radiolucent stones of any size or number or radio-opaque stones ≤3 cm; and (*c*) a functioning gall bladder on oral cholecystography. Thirteen patients were excluded: four failed to return after the first treatment, two emigrated, one became pregnant, and two could not tolerate bile salt treatment; lithotripsy was abandoned in four because of difficulty in visualising the gall bladder.

Clearance of gall stones with shock wave lithotripsy in 49 patients based on stone profile

No of stones	Size of stones (cm)	Clearance of stones: 100%	≥50%	<50%	0
	Radiolucent stones				
1	<1	1			
	2-3	6	1		2
	3-5			1*	1*
2, 3	<1	3	2		
	2-3	1	2		
≥4	<1	12*	4*	1*	2*
	2-3	1*	1*		1*
	Radio-opaque stones				
1	2-3				2*
2, 3	2-3	1*			
≥4	<1	1*			1*
	2-3	1*			1*
Total		27	10	2	10

*Patients who would not have been eligible for treatment with established selection criteria.

All patients received combination dissolution treatment comprising chenodeoxycholic and ursodeoxycholic acid 7 mg/kg/day each, administered as one tablet (Lithofalk), and one capsule of a terpene mixture (Rowachol) three times daily. Ultrasonography was used to focus the shock waves from a piezoelectric lithotripter on the stones within the gall bladder. Each patient received 6000 shock waves in each treatment session (frequency 2·5 shock waves/s, 100% power) up to a maximum of six treatments at intervals of two to three weeks. All patients were followed up every month clinically and by ultrasonography. Two consecutive ultrasound scans and an oral cholecystogram were required to establish that the stones had been cleared.

The 49 patients received 175 sessions of shock wave treatment, each patient requiring an average of three treatments (range one to six). The table shows the outcome related to number, size, and type of stones. We classified patients into four groups. One group comprised those in whom all stones were cleared (n=27; median follow up six months (95% confidence interval 4·8 to 7·5) to time of clearance). The second group comprised those with ≥50% clearance—that is, fragmentation with appreciable clearance of stones (n=10; median follow up nine months). The third group comprised those with <50% clearance—that is, fragmentation with little clearance of stones (n=2; median follow up seven months). The fourth group comprised patients who had insignificant fragmentation or clearance of fragments after six treatment sessions and follow up of six months (n=10).

Thirty one patients, including 16 of the 27 in whom all stones were cleared, would not have been deemed suitable for lithotripsy by previous criteria.[1,2] Lithotripsy was generally well tolerated. Eight patients suffered biliary colic during follow up, which usually occurred within 48 hours after lithotripsy. One patient in whom treatment failed had transient jaundice four days after treatment, which resolved spontaneously. None had skin ecchymosis or haematuria or any clinical or biochemical evidence of acute pancreatitis.

Comment

We estimate that up to 60% of the patients we treated after extending the selection criteria for treatment would otherwise have been treated by cholecystectomy. Treatment was most successful in patients with many small stones (≤1 cm diameter). The median time to clearance of stones in this group was seven months. This was shorter than would be expected with oral dissolution treatment alone, suggesting that lithotripsy played an important part.[3,4]

The relative importance of extracorporeal fragmentation and oral dissolution treatment in this treatment is unclear. Sackman *et al* showed that bile salt treatment had a major influence on their results: stones were completely cleared in only 30% of their patients two months after lithotripsy compared with 93% at 18 months.[1] We added a terpene compound to the bile salts, which may have enhanced dissolution and had an important antispasmodic effect, thus reducing biliary colic.[5] Interestingly, only 16% of our patients had biliary colic compared with 35% reported by Sackman *et al*.[1]

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Hypersensitivity vasculitis due to ofloxacin

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Since its introduction in 1985 the new fluoroquinolone antibiotic ofloxacin has gained widespread use and much information has accumulated about its possible adverse effects.[1] Skin reactions have been uncommon[1,2]; in particular, no established case of hypersensitivity vasculitis directly related to ofloxacin has been reported.[1] We report such a case.

Case report

A 67 year old man was admitted to hospital because of a rash that had developed two days earlier. For the past 15 years he had had diabetes mellitus and congestive heart failure, for which he was taking isophane insulin 40 units in the morning and 12 units in the evening and frusemide 40 mg daily. Two weeks before admission his right foot had been accidentally burnt and became infected. As povidone iodine baths were ineffective ofloxacin 200 mg twice daily had been started three days before admission. One day later a pruritic purpuric rash had developed on his feet and legs. Ofloxacin was stopped and two days later the rash cleared. On the evening before admission he had taken another tablet of ofloxacin 200 mg, and a similar rash had developed.

On admission the rectal temperature was 36·9°C. Physical examination showed an extensive papular purpuric rash on the feet and legs, swelling of the scrotum, pitting oedema of the legs, and an infected wound on the right foot. Blood cultures and cultures from the wound were sterile. The erythrocyte sedimentation rate (Westergren) was 42 mm in the first hour. A blood count yielded $11{\cdot}4\times10^9$ leucocytes/l with 6% band forms and 2% eosinophils. Blood glucose concentration was 24·5 mmol/l. Plasma urea concentration was 7·6 mmol/l, creatinine 203 μmol/l, alanine transaminase 114 U/l, lactate dehydrogenase 407 U/l, and alkaline phosphatase 288 U/l. Blood tests for antinuclear antibody, rheumatoid factor, hepatitis B surface antigen, and cryoglobulins and the Venereal Disease Research Laboratory test yielded negative results. Serum C3 concentration was 0·56 g/l (normal 0·70-1·76 g/l) and C4 concentration normal. Urine analysis showed a protein concentration of 3 g/l, a glucose concentration of 3 g/l, and 10 red cells per high power field. A mast cell degranulation test for ofloxacin gave a strongly positive result. A skin biopsy taken from the purpura shortly after admission showed leucocytoclastic angiitis.

Ofloxacin was stopped on admission and intravenous cefoxitin sodium and oral prednisone 40 mg daily started. Insulin and frusemide were continued. The rash and the pedal and scrotal oedema resolved, and the liver and kidney functions improved. The dose of prednisone was rapidly reduced. On the 17th day in hospital the patient was discharged.

Comment

This patient had clear evidence of a hypersensitivity vasculitis characterised clinically by a diffuse papular purpuric rash and histologically by a leucocytoclastic angiitis. Allergic vasculitis is a well known adverse effect of many drugs, including antibiotics and thiazide diuretics,[3] and may occur with frusemide.[4] Our patient, however, had taken frusemide for many years without adverse effects and was still taking it when the rash resolved. Thus frusemide is unlikely to have caused the hypersensitivity vasculitis in this patient. Infection can cause allergic vasculitis, but this patient's vasculitis resolved despite a worsening infection. The temporal relation between the use of ofloxacin and the development of purpura and the resolution of the purpura after the drug was stopped and its reappearance after rechallenge suggest a causal relation. The strongly positive result of the mast cell degranulation test for ofloxacin confirms hypersensitivity to the drug.

Although four cases of allergic vasculitis during ofloxacin treatment have been reported in postmarketing surveillance studies, three cases seem to have been unrelated to the drug and not enough is known to evaluate the fourth.[1] Hypersensitivity vasculitis induced by ciprofloxacin, another fluoroquinolone antibiotic, has been reported.[5] We suggest that hypersensitivity vasculitis be added to the growing list of adverse effects of the fluoroquinolones.

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