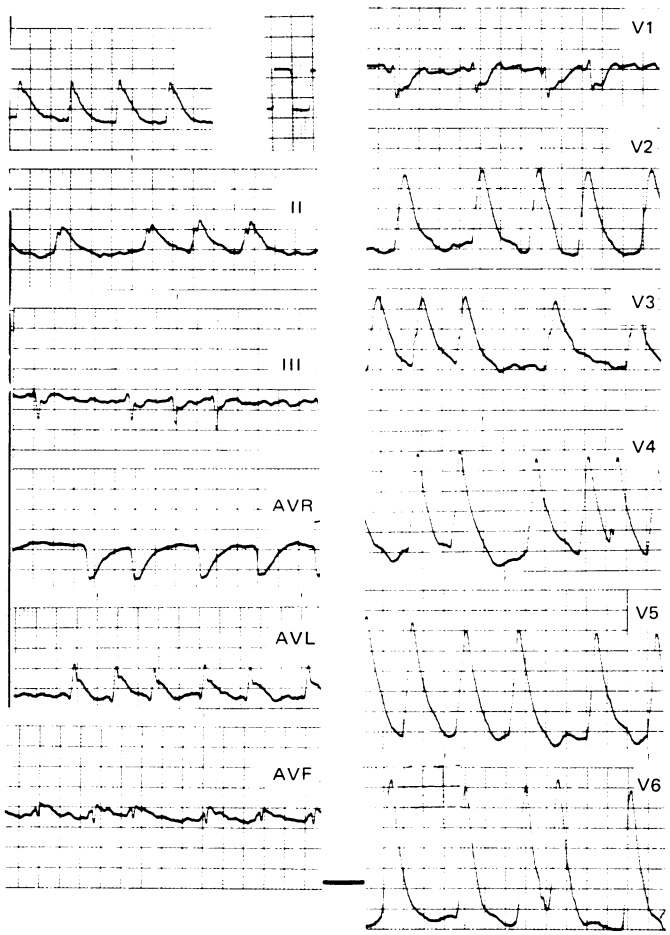

Electrocardiogram performed nine hours after admission. Plasma potassium concentration 3·3 mmol(mEq)/l. Serum calcium concentration 5·8 mmol/l (23·2 mg/100 ml).

glucose 6·4 mmol/l (115 mg/100 ml). Arterial blood pH was 7·42, P_{CO_2} 4·45 kPa (34 mm Hg), and P_{O_2} 5·76 kPa (43 mm Hg) breathing air. A supine portable *x* ray film of her chest showed pulmonary oedema. *X* ray films of her hand, pelvis, and chest showed no definite changes associated with hyperparathyroidism. The figure shows her electrocardiogram.

Other investigations included: serum alkaline phosphatase activity 411 (normal range 80-280) U/l, inorganic phosphorus concentration 1·49 (0·8-1·4) mmol/l (4·6(2·5-4·3) mg/100ml), and parathyroid hormone concentration, measured by radioimmunoassay for the C terminal, 9200(200-800) pg/ml. Concentrations of 25-hydroxyvitamin D_3 and 24, 25-dihydroxyvitamin D_3 measured by radioimmunoassay were normal (75 nmol/l (30 ng/ml) and 500 pmol/l (200 pg/ml respectively), but 1,25-dihydroxyvitamin D_3 was undetectable. Creatine phosphokinase activity was 1767 (normal range 25-195) U/l, and lactate dehydrogenase activity 1766 (230-460) U/l. Thyroid function and immunoglobulin concentrations were normal.

The hypokalaemia and hypoxaemia were corrected and intravenous dobutamine, cefotaxime, and thiamine given. Twelve hours after admission she suffered a respiratory arrest and was mechanically ventilated. At this time her serum calcium concentration was reported as having been 4·8 mmol/l (19·2 mg/100 ml) on admission. Acute peritoneal dialysis with conventional dialysate was started pending availability of calcium free solution. Intramuscular calcitonin and intravenous frusemide were administered. Her blood pressure, however, fell further and despite twelve hours of dialysis her serum calcium concentration increased to 5·7 mmol/l (22·8 mg/100 ml). She died of a cardiac arrest 28 hours after admission. At necropsy an adenoma of the left lower parathyroid gland measuring 4 cm in diameter was found. The heart weighed 500 g, and left ventricular hypertrophy and a yellow mottled appearance of the cut surface were noted. Valves and coronary arteries were normal. The lungs were oedematous and contained embolic antemortem thrombus, as did the leg veins. Macroscopic sections of the kidneys and vertebral bodies seemed normal. Histological examination showed extensive myocardial fibre calcification and degeneration and calcification of thyroid colloid. There was no pulmonary calcification and only slight nephrocalcinosis. Vertebral body sections showed patchy foci of slightly increased activity of osteoclasts, osteoblasts, and fibroblasts.

Comment

This woman showed typical clinical and pathological features of acute hyperparathyroidism.[3] The bizarre changes observed on her electrocardiogram have to our knowledge not been previously reported. They represent the combined effects of accelerated membrane repolarisation due to hypercalcaemia, myocardial injury, pulmonary embolism, and left ventricular hypertrophy. The low concentration of 1,25-dihydroxyvitamin D_3 is surprising because a high concentration of parathyroid hormone normally stimulates 1-hydroxylation of vitamin D in the renal cortex. This finding may have been due to the acute renal failure or a direct effect of the severe hypercalcaemia.

Hyperparathyroidism should be suspected as the cause of severe hypercalcaemia in patients presenting in good general condition without obvious clinical or radiological signs of multiple myelomas or other malignancy. They may have a history of renal calculi or peptic ulceration and may have a palpable mass in the neck, due to the tumour.[4] *X* ray films of the hand show subperiosteal erosions in three quarters of these patients, but, as this case emphasises, extensive metastatic calcification can occur without radiologically evident bone disease.[2]

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Can patients benefit from reading copies of their doctors' letters about them?

The question of patients' access to their medical records is raised by the Data Protection Act. Studies in general practice, obstetrics, and hospital patients show that patient access can be safe and improve patient-doctor communication.[1-3] Physicians often hold negative views about patients routinely seeing general hospital notes, but it might have advantages and may be inevitable. As a first step towards examining its effect we studied the responses of 50 new outpatients who received a copy of our letter to their general practitioner. We wanted to see if there were advantages for communication. We compared reading the letter with further discussion with paramedical staff and we obtained patients' and general practitioners' opinions on these approaches.

Patients, methods, and results

Fifty consecutive new patients referred to a rheumatology clinic received an unedited copy of the letter sent to their general practitioner after their first consultation. A second group of 50 consecutive patients had an explanatory discussion with a paramedical member of the rheumatology unit after their medical consultation. Using a questionnaire they graded each of these approaches on a five point scale (very good to very poor) for comprehension, information, help, or whether it was a good or bad idea. Eight patients sent copies of the letters did not respond to the questionnaire; 10 patients offered an explanatory talk did not want it.

Subsequently another 50 patients and 50 local general practitioners were given a second questionnaire seeking their preferences for the two methods outlined above and two additional suggestions for improved communication—namely, (*a*) sending patients a standard letter about their condition in "everyday" language, and (*b*) asking patients to visit their general practitioner for discussion after the clinic letter arrived. They graded these on a four point scale from best to least satisfactory. All 50 patients and 38 general practitioners responded to this second questionnaire.

Both reading the hospital letter and talking with paramedical staff were acceptable and had advantages for doctor-patient communication. The table summarises the results. The letter compared favourably with a further discussion. Over half of the patients thought seeing the letter both helpful and clear; many found it informative. It was not always as useful as talking with paramedical staff. Only one patient found the letter confusing; her problems were solved promptly on return to the clinic.

Responses to the second questionnaire showed that patients were equally divided in their opinions of the alternatives; 19 (38%) thought further discussion with their general practitioner the worst alternative. General practitioners were more polarised; 21 (55%) preferred the idea of patients having further discussion with paramedical staff, but only 5 (13%) thought patients reading the clinic

Patients' assessments of reading the letter to their general practitioner and further discussion with paramedical staff. Figures are numbers (percentages) of patients.

General category	Grade of response*	Letter to general practitioner (n=42)	Discussion with paramedical staff (n=40)
Comprehensive (How clear?)	1-2	31 (74)	40 (100)
	3	8 (19)	0
	4-5	3 (7)	0
Information (Were you more informed?)	1-2	20 (48)	25 (63)
	3	20 (48)	15 (37)
	4-5	2 (54)	0
Help (Did you find it helpful?)	1-2	35 (83)	39 (98)
	3	6 (14)	1 (2)
	4-5	1 (2)	0
Good idea (Is this a good idea?)	1-2	41 (98)	37 (93)
	3	0	1 (2)
	4-5	1 (2)	2 (5)

*1-2 = Very good to good. 3 = Average. 4-5 = Poor to very poor.

letter was the best option; half thought that letting patients read their copy letter was least likely to improve communication. By contrast, only one fifth of patients shared these latter sentiments.

Comment

Our results show that patients are pleased to read copies of our letters to their general practitioners. They derived considerable benefit from doing so. A further discussion with paramedical staff was helpful but staff and facilities are not always available. Recent opinion on open access remains divided. We found considerable opposition from some general practitioners to even limited release of information. Fears that the letters may be confusing were not confirmed.[4] We did not censor letters but none made reference to malignant disease or psychiatric conditions—two sectors where problems may arise.[1,5] Open access to notes need not be the spectre that physicians fear but may have the positive advantages of improved communication and understanding. The debate whether patients should have open access to their medical notes should concentrate more on the potential advantages which may result and give less attention to the possible drawbacks.

Allowing patients to see copies of single letters is the first place to start in what is clearly a difficult ethical issue. We think that there is a case for a rational prospective evaluation of the methods and value of giving patients more access to their medical notes.

We thank Kay Roberts for help in collating our results.

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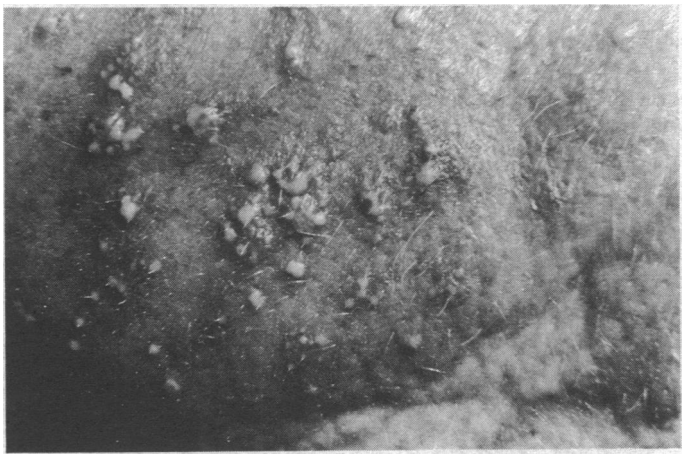
Pustular dermatosis induced by co-trimoxazole

Four patients developed a generalised pustular dermatosis during treatment with co-trimoxazole, which resolved spontaneously when the drug was stopped.

Case reports

Case 1—A woman aged 70 was treated with co-trimoxazole (two tablets twice daily) for cystitis. After five days a pustular eruption developed on her face and hands, which became generalised the next day. The pustules seemed to be superficial and non-follicular and occurred on an erythematous base (figure). Her white cell count was 11 × 10^9/l (neutrophil leucocytosis) and she was febrile (37·5°C), but skin culture did not show pathogenic organisms. After withdrawal of the drug her fever settled and the rash resolved over three weeks.



Superficial non-follicular pustular eruption.

Case 2—A woman aged 77 was treated with co-trimoxazole (one tablet twice daily) for dysuria. After five days a pustular rash developed on her face and then extended symmetrically to other areas, favouring acral sites. The pustules were superficial and non-follicular, occurring on a moderately inflamed base. Skin culture showed only commensal flora. Her white cell count was 13 × 10^9/l (neutrophil leucocytosis). The rash desquamated and resolved over two weeks after withdrawal of the drug. Intercurrent diabetes was controlled throughout with metformin (500 mg thrice daily).

Case 3—A woman aged 95 sustained a laceration to her forehead and was given prophylactic treatment with co-trimoxazole (one tablet twice daily). Six days later a superficial pustular and erythematous eruption developed at the margins of the wound. Over 24 hours this developed at other facial and body sites. Culture of the wound showed commensal flora, and the pustules were sterile. Her white cell count was normal (7·1 × 10^9/l) and the urea concentration increased (11·3 mmol/l (68 mg/100 ml)). The rash diminished slowly after withdrawal of the drug, with full resolution after a month. Intercurrent drugs (which were continued unchanged) included carbamazepine (100 mg twice daily), spironolactone (5 mg daily), frusemide (5 mg twice daily), and phenytoin (25 mg daily).

Case 4—A man aged 60 was treated with co-trimoxazole (two tablets twice daily) for a chest infection. Three days later a pustular eruption developed on exposed skin sites. Within a day his palms, soles, arms and legs, and, to a less extent, other body sites were affected. The pustules tended to coalesce and to occur with erythema. A history of scaly elbows suggested psoriasis, but this was not confirmed. Bacteriological examination showed only commensal skin flora. His white cell count was 11·8 × 10^9/l (76% neutrophils, 10% eosinophils), and his urea concentration was raised at 15 mmol/l (90 mg/100 ml). The rash desquamated and resolved in response to withdrawal of the drug.

Comment

Drug reactions characterised by extensive formation of sterile pustules are rarely reported and have not been recognised in association with co-trimoxazole. No cases have been reported to the manufacturer (Wellcome), but the Committee on Safety of Medicines has received one report (personal communication). The presentation and morphology of the rash were similar in all our patients and necessitated admission to hospital. None of the patients was known to have taken the drug previously.

Reports of generalised pustular drug rash have implicated different drugs,[1,2] including corticosteroids (but not sulphonamides). The morphology and clinical course of the condition in these reports varied. Recently, toxic pustuloderma that may have been induced by carbamazepine was described,[3] but in one of our patients (case 3) we were able to continue treatment with carbamazepine while the rash resolved after withdrawal of co-trimoxazole.

Histological examination of the skin (three cases) showed subcorneal pustules, but in only one case was inflammatory infiltration noted, and there was no evidence of vasculitis (as described for acute generalised pustular bacterid[4]). Exanthematous pustular psoriasis was considered (case 4) but could not be confirmed. The possibility of subcorneal pustular dermatosis (Sneddon-Wilkinson disease) was discounted on clinical grounds.

Co-trimoxazole is known to cause several different cutaneous eruptions.[5] Our cases suggest that it may also cause a distinctive exanthematous pustular dermatosis.

We are grateful to Dr E J Raffle and Dr V K Dave for allowing us to study their patients.