Discussion

If patients with uncomplicated type II diabetes could be as satisfactorily looked after in general practice as in hospital clinics for diabetics, patients (by avoiding the time taken and finance incurred in attending the clinic), general practitioners (by maintaining the continuity of care of patients), and hospital clinics (by having more time available to see more complicated problems) would benefit. This study, however, shows that routine care by general practitioners is not as satisfactory as routine care by hospital clinics. One reason for this is that general practitioners often do not have at hand the facilities that are considered to be essential in any diabetic clinic. Dietetic advice, chiropody, rapid access to laboratory and radiographic services, though available, are not immediately to hand. Possibly most important is the absence in most general practices of an automatic recall system for patients who do not attend. This may well account for the low number of patients who were regularly reviewed by their general practitioner. In addition, most practices are not geared to giving people definite appointments for review four or six months later.

There have been several successful attempts at helping general practitioners to care for diabetic patients. These schemes fall into two categories: firstly, there is the miniclinic system, which has been described by Thorn[5] and in which general practitioners are encouraged to set up miniclinics within their practice, often with one partner taking a particular interest in diabetics; secondly, there are shared care schemes, such as that described by Hill,[6] in which the hospital clinic sees the patients rarely but provides a hospital based blood sugar assay service with recall of patients who do not attend. Some of these schemes now also measure glycosylated haemoglobin concentration and use ophthalmic opticians to screen for diabetic retinopathy.[7] Although these schemes have proved successful in certain centres they need considerable organisation.

Our study shows that the simple transfer of responsibility for continuing care from hospital clinics to general practice is unlikely to maintain an adequate standard of care. The need, however, to discharge patients back to their general practitioners remains. Evidently, careful planning will be needed before diabetics can be satisfactorily transferred back to the care of their general practitioner. Different patterns of shared care (including miniclinics where appropriate) may prove satisfactory in different areas. The clinics continue to play an important part as a focus for expertise to which general practitioners can rapidly turn; some centres may provide "review clinics," at intervals of one to two years, at which patients are thoroughly reassessed, and in between these visits patients have their diabetes monitored and treatment adjusted by their general practitioner.

Although any one system is unlikely to achieve nationwide success, a computerised system similar to that used for follow up of patients with thyroid abnormalities in many parts of the United Kingdom may be worthy of further study. This would recall the patients to see their general practitioner at regular intervals, warn him, and request him to collect both clinical information and blood for estimation of glycosylated haemoglobin concentration. General practitioners would be relieved of the necessity of having a complicated recall system and would be prompted to take appropriate clinical action by the computer print out. Such a system is under active consideration or development in a number of areas including our own.

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SHORT REPORTS

Kingella kingae septicaemia with a clinical presentation resembling disseminated gonococcal infection

We report the first case in Britain of an adult with pyrexia of undetermined origin and clinical features suggesting disseminated gonococcal infection[1] but in whom the cause was septicaemia due to *Kingella kingae*.

Case report

A previously healthy English woman, aged 21, was admitted on 7 February 1984 with a 10 day history of fever, malaise, and fleeting aches in the joints of the fingers, toes, ankles, and hips. Two weeks before her illness she had returned from a holiday visiting Florida and the Bahamas. She denied having had any sexual intercourse since October 1983. On admission she looked unwell, her temperature was 38·9°C, and there were sparse petechial lesions on her right thumb, left forefinger, and right big toe. During the next five days she developed rigors each night and new purple, 1-2 mm vasculitic skin lesions on the soles of her feet and other fingers, which were painful only at the time of their onset. On 10 February she had acute pleuritic pain in the left hypochondrium and tenderness in the left costal margin, which suggested either a splenic infarct or a splenic abscess.

On investigation the haemoglobin concentration was 11·0 g/dl, the white cell count was $23{\cdot}6 \times 10^9$/l (predominantly polymorphs), and five sets of blood cultures yielded negative results. Numerous specific serological tests yielded negative results. Urethral and cervical cultures for gonococci were negative, and various scanning techniques failed to show any focus of infection. Increased complement concentrations (haemolytic complement 200% of normal and C3 136% of normal) and circulating immune complexes were shown in the serum by Professor Mowbray at St Mary's Hospital.

Her condition worsened, and a two week course of blind antibiotic treatment was started on 12 February with oxytetracycline 500 mg given by mouth once every six hours. Within 24 hours there was a noticeable improvement and she became afebrile. During the subsequent week her condition continued to improve but she had two further transient attacks of myalgia and arthritis affecting her toes. She was discharged home feeling well on 23 February. One week later she developed intermittent claudication in the left leg; the peripheral pulses were no longer palpable on the left side. These symptoms resolved during April, but the pulses remained absent.

After her discharge from hospital the last blood culture, collected on 12 February just before the start of tetracycline treatment, yielded growth of a fastidious Gram negative diplobacillus, which was β haemolytic, oxidase positive, catalase negative, and grew best on blood or chocolate agar incubated aerobically in a 10% carbon dioxide atmosphere. This organism was isolated only after 16 days' incubation of the carbon dioxide blood culture broth and was sensitive to tetracycline and penicillin. It was finally identified as *K (Moraxella) kingae*. A serum sample collected three weeks after the onset of her illness showed a fluorescence antibody titre of 1/1600 against her blood culture isolate containing kingellae, and countercurrent immunoelectrophoresis of the serum, using a suspension of this organism as the antigen, showed strong specific precipitin lines. Negative control serum samples showed no reaction with either of these tests.

Comment

This patient is the first adult to be reported in Britain with septicaemia due to *K kingae*. One adult has been reported on in the United States with clinical features due to *Moraxella osloensis* bacteraemia

resembling those of gonococcaemia.[2] There have also been reports from the United States of a child with leukaemia who had bacteraemia, arthritis, and skin lesions due to *K kingae*[3] and another child with moraxella endocarditis.[4] The organism may enter the bloodstream from the respiratory tract.[5]

Kingella, Moraxella, and *Neisseria* genera all belong to the *Neisseriaceae* family, and all can cause disseminated infection with similar clinical manifestations.

We thank Miss J Midgley of the microbiology department at this hospital and the National Collection Type Culture, Public Health Laboratory Service, Colindale, for help with the final identification of the blood culture isolate.

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Agranulocytosis caused by spironolactone

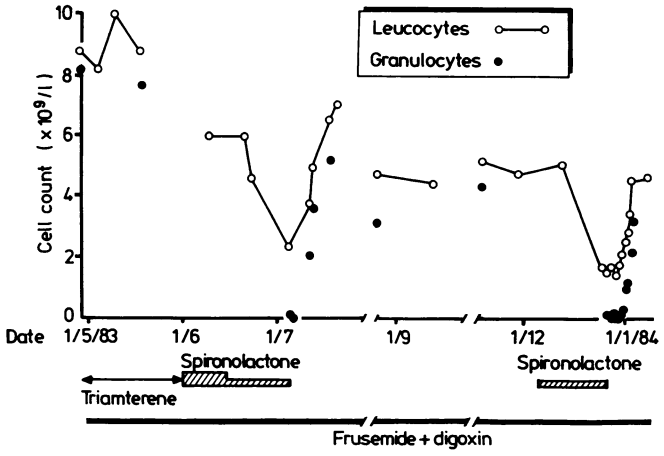
Spironolactone has been in widespread use for over 20 years, and so far as we know it has never been documented unequivocally as the cause of agranulocytosis. In only one report was the possible association mentioned briefly, and no details were given.[1] The manufacturer has received a few reports of agranulocytosis over the years but in none was a causal role of spironolactone convincing (H van der Hulst, Searle Pharmaceuticals, personal communication, 1984). We have observed a patient in whom complete agranulocytosis was induced by spironolactone on two occasions.

Case report

A 70 year old woman was admitted for cardiac failure and renal dysfunction. On admission she had dyspnoea, chest pain on exertion, peripheral oedema, and hepatomegaly. She had not been using any medicines. Abnormal haematological findings were: urea concentration 22·4 mmol/l (135 mg/100 ml) (normal < 7·5 mmol/l; < 45 mg/100 ml); creatinine concentration 162 μmol/l (1·8 mg/100 ml) (normal < 110 μmol/l; < 1·2 mg/100 ml); γ-glutamyltransferase activity 88 IU/l (normal < 40 IU/l); alanine aminotransferase activity 36 IU/l (normal < 30 IU/l); lactate dehydrogenase activity 550 IU/l (normal < 320 IU/l). Leucocyte counts were repeatedly normal with normal differentiation. Electrocardiography showed multiple ventricular extrasystoles. *X* ray film of the chest disclosed cardiomegaly and pulmonary congestion.

Treatment was instituted with digoxin 0·5 mg, frusemide 80 mg, and triamterene 50 mg daily and a sodium and protein restricted diet. She showed slow but definite improvement and digoxin and frusemide were decreased to 0·125 and 40 mg daily. Renal function remained impaired. Because of hyperkalaemia triamterene was discontinued. Once the serum potassium concentration had returned to normal spironolactone 100 mg daily was instituted (see figure). Five weeks later a routine blood count disclosed leucopenia ($2·6 \times 10^9$/l) with complete agranulocytosis, relative lymphocytosis, and eosinophilia (15%). Bone marrow biopsy showed normal red cell and platelet production but many immature myelocytes in the absence of mature granulocytes. Spironolactone was discontinued. Within one week the granulocyte count was normal ($4·9 \times 10^9$/l; 75% neutrophils) and two weeks later she was discharged.

One month after discharge the patient was readmitted because of dyspnoea and peripheral oedema. Frusemide was increased to 80 mg daily and digoxin to 0·25 mg daily and the importance of the diet re-emphasised. All signs and symptoms of congestive heart failure disappeared. Leucocyte counts were repeatedly normal. Renal function remained stable and the patient was discharged again. Two months later she was admitted for the third time. She had the same symptoms as before. In addition to her treatment regimen, which she had adhered to, she was again given spironolactone (50 mg/day). Three weeks later the leucocyte count (normal on admission and with normal differentiation) had fallen to $1·6 \times 10^9$/l. Differentiation showed complete agranulocytosis. The causal relation between spironolactone and the agranulocytosis was then recognised and the drug discontinued. Nine days later the leucocyte count was $4·5 \times 10^9$/l and showed normal differentiation.



Changes in white cell counts in relation to treatment with spironolactone.

Comment

In our opinion there is no doubt that spironolactone caused the agranulocytosis in this patient. All other drugs were continued without difficulty and no other cause was found. The temporal relation with the administration of spironolactone was clear on both occasions (see figure). In view of the apparent rarity of the reaction it may be idiosyncratic. Probably the mechanism is immunoallergic and acts by destroying granulocytes in the peripheral blood. The complete absence of granulocytes in the presence of many myelocytes in the bone marrow, the accelerated reaction to rechallenge, and the rapid and complete recovery after discontinuation of spironolactone on both occasions were compatible with a drug dependent antibody mediated reaction.

Even common drugs that have been used safely for years may unexpectedly cause life threatening adverse effects. Plainly drug monitoring should not be restricted to new drugs only.

We thank Dr A W F M van Leeuwen for the bone marrow biopsy report.

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Relation between use of tampons and urogenital carriage of group B streptococci

Although infection with group B streptococci in man was described in 1943, these bacteria were unfamiliar to clinicians until their association with neonatal infection was established in the 1960s.[1] The reason for this apparent increase in the incidence of infections due to group B streptococci remains obscure. In the study reported here we found a positive correlation between the use of tampons and the carriage of group B streptococci, which might have contributed to the increase.