excretion is abnormally increased and significantly higher than that in comparable Europid patients. In Europid patients increased albumin excretion (microalbuminuria) indicates an increased risk of death, though the mechanisms are unknown.^{7,8} It also predicts the development of clinical proteinuria in patients with both insulin dependent^{4,6} and non-insulin-dependent diabetes.⁸

The high prevalence of microalbuminuria in Indian diabetics is especially important because of the strikingly high prevalence of known diabetes shown in the Southall diabetes survey¹ and the earlier age of diagnosis in Indians compared with Europids. The number and proportion of diabetic patients of all ethnic groups accepted for renal replacement in the United Kingdom increased appreciably from 72 (5%) in 1980 to 200 (11%) in 1984,¹² and 24% of these were patients with non-insulin-dependent diabetes mellitus. There are over 1.2 million Indians in Britain, and diabetic nephropathy in this ethnic group will probably become an increasingly important health problem.

In this study hypertension was positively associated with albumin:creatinine ratios in the Indian but not the Europid diabetics. Previous reports have given conflicting results on this point in Europids. In the Bedford study significant positive correlations were obtained between systolic blood pressure and the logarithm of albumin excretion rate in newly diagnosed diabetics.¹³ A low degree of positive correlation between albumin excretion rate and systolic blood pressure was also noted in subjects with fasting hyperglycaemia, known diabetics, and control subjects aged 60-74 in Denmark¹⁴ but not in Danish patients stratified by duration of diabetes and urinary albumin concentration.⁸ In another study albumin excretion rate was found to be increased before treatment in non-diabetic subjects with hypertension and to fall significantly after treatment.¹⁵ Such discrepancies may be real or due to differences in methodology or sample size or to sampling variation. Nevertheless, the higher proportion of patients with increased albumin:creatinine ratios among the Indian diabetics in this study cannot be ascribed to higher average blood pressure or to a greater prevalence of hypertension (table). Conceivably the kidneys of Indians may be more vulnerable to the effects of raised blood pressure or diabetes, or both.

Because of the high prevalence of diabetes in Indians and its early

onset increasing morbidity and perhaps mortality from clinical nephropathy may be expected. Research should aim at assessing the efficacy of potential preventive approaches—for example, the control of blood glucose concentrations and blood pressure and perhaps dietary protein restrictions.

We acknowledge financial support from the Wellcome Trust, Guy's Hospital trustees, the Department of Health and Social Security, and Ealing Hospital Research Fund. We thank Drs S W and F Rahman for their help in recruiting the non-diabetic control subjects, and the patients and control subjects for their cooperation.

References

- 1 Mather HM, Keen H. The Southall diabetes survey: prevalence of known diabetes in Asians and Europeans. *Br Med J* 1985;291:1081-4.
- 2 Nicholl CG, Levy JC, Mohan V, Rao V, Mather HM. Asian diabetes in Britain: a clinical profile. *Diabetic Medicine* 1986;3:257-60.
- 3 Samanta A, Burden AC, Feehally J, Walls J. Diabetic renal disease: differences between Asian and white patients. *Br Med J* 1986;293:366-7.
- 4 Viberti GC, Hill RD, Jarrett RJ, Argyropoulos A, Mahmud U, Keen H. Microalbuminuria as a predictor of clinical nephropathy in insulin-dependent diabetes mellitus. *Lancet* 1982;ii:1430-2.
- 5 Parving H-H, Oxenbøll B, Svendsen A, Christiansen JS, Andersen AR. Early detection of patients at risk of developing diabetic nephropathy. A longitudinal study of urinary albumin excretion. *Acta Endocrinol (Copenh)* 1982;100:550-5.
- 6 Mogensen CE, Christensen CK. Predicting diabetic nephropathy in insulin-dependent patients. *N Engl J Med* 1984;311:89-93.
- 7 Jarrett RJ, Viberti GC, Argyropoulos A, Hill RD, Mahmud U, Murrills TJ. Microalbuminuria predicts mortality in non-insulin-dependent diabetes. *Diabetic Medicine* 1984;1:17-9.
- 8 Mogensen CE. Microalbuminuria predicts clinical proteinuria and early mortality in maturity-onset diabetes. *N Engl J Med* 1984;310:356-60.
- 9 Freedman BJ. Caucasian. *Br Med J* 1984;288:696-8.
- 10 Gatling W, Knight C, Hill RD. Screening for early diabetic nephropathy: which sample to detect microalbuminuria. *Diabetic Medicine* 1985;2:451-5.
- 11 Keen H, Chlouverakis C. An immunoassay method for urinary albumin at low concentrations. *Lancet* 1963;ii:913-4.
- 12 Cameron JS, Challah S. Treatment of end-stage renal failure due to diabetes in the United Kingdom, 1975-84. *Lancet* 1986;ii:962-6.
- 13 Keen H, Chlouverakis C, Fuller J, Jarrett RJ. The concomitants of raised blood sugar: studies in newly-detected hyperglycaemics. *Guy's Hospital Reports* 1969;118:247-54.
- 14 Damsgaard EM, Mogensen CE. Microalbuminuria in elderly hyperglycaemic patients and controls. *Diabetic Medicine* 1986;3:430-5.
- 15 Pedersen EB, Mogensen CE. Effect of antihypertensive treatment on urinary albumin excretion, glomerular filtration rate, and renal plasma flow in patients with essential hypertension. *Scand J Clin Lab Invest* 1976;36:231-2.

(Accepted 26 October 1987)

SHORT REPORTS

Corticosteroids in treatment of disseminated tuberculosis in patient with HIV infection

Tuberculosis in association with human immunodeficiency virus (HIV) has been well described.^{1,2} Usually it responds satisfactorily to conventional chemotherapy.¹ Patients with tuberculosis who are desperately ill are often given corticosteroids as well as antituberculous chemotherapy to try to reduce the toxicity of the disease and to prevent death early in treatment.³ Corticosteroids may achieve this effect by suppressing or at least modifying cell mediated immunity.

A patient with HIV infection and disseminated tuberculosis continued to have a hectic fever and to lose weight despite treatment with quadruple chemotherapy. He showed a dramatic response to the addition of corticosteroids.

Case report

A 30 year old bisexual man was admitted with a two month history of productive cough, lethargy, and weight loss (15 kg). He was wasted and feverish with a temperature of 39°C and he had mild cervical lymphadenopathy. Blood count showed pancytopenia (haemoglobin 110 g/l, white cell count $3.0 \times 10^9/l$, and platelets $129 \times 10^9/l$). A bone marrow biopsy specimen on the same day showed numerous acid fast bacilli but no epithelioid cell granulomas. Non-reactive (non-granulomatous) disseminated tuberculosis was diagnosed and he was

given quadruple chemotherapy (rifampicin, isoniazid, pyrazinamide, and ethambutol).

A chest radiograph showed some indefinite mottling in the right lower zone and an enlarged paratracheal lymph node. Sputum examination showed acid fast bacilli and later cultures were positive for *Mycobacterium tuberculosis*; the organisms were fully sensitive to the standard drugs. A Mantoux test with 10 tuberculin units was negative. Antibodies to HIV were detected. Serological testing for hepatitis B and syphilis gave negative results. Erythrocyte sedimentation rate was 28 mm in the first hour.

For six weeks the patient's condition continued to deteriorate: he remained feverish, lost weight, and grew steadily weaker (figure). At this point prednisolone 30 mg/day was added to the regimen. His clinical condition improved almost immediately. Within three days he began to gain weight and was afebrile in 12 days. In six weeks his weight increased from 45 kg to 62 kg.

At the same time the minor radiological pulmonary changes cleared completely and the haematological abnormalities reverted to normal (haemoglobin 137 g/l, white cell count $8.3 \times 10^9/l$, platelets $279 \times 10^9/l$, and erythrocyte sedimentation rate 5 mm in first hour). He was finally discharged from the hospital fit and well after five months' treatment. Steroids were gradually withdrawn before discharge, but quadruple chemotherapy was continued.

Comment

Tuberculosis, including disseminated tuberculosis, is a common feature of the acquired immune deficiency syndrome (AIDS).¹ It usually responds satisfactorily to treatment even though granuloma formation is almost always impaired. A case has, however, been described of a woman with HIV disease and non-granulomatous tuberculosis who failed to respond to antituberculous chemotherapy and died.⁴ In our patient with non-reactive

Temperature and weight chart before and after the introduction of corticosteroids.

tuberculosis quadruple therapy alone proved ineffective, even though the organisms were fully sensitive to the drugs. Added corticosteroids, however, achieved a dramatic improvement leading to cure. If the patient had not had HIV antibodies we would have used steroids much earlier in the illness. We were inhibited firstly by the possibility that we were dealing with an atypical mycobacterium with multiple drug resistance and secondly by the apparent illogicality of using steroids in a patient already immunocompromised. (The negative tuberculin reaction and the absence of granuloma formation certainly supported an assumption of impaired cell mediated responses.)

Evidence has recently been presented that corticosteroids may dramatically improve the outcome in patients with AIDS and severe *Pneumocystis carinii* pneumonia.⁵ The response to steroids in our patient suggests that corticosteroids may have a part to play in some other infections in patients with AIDS or AIDS related complex.

- 1 Pitchenik AE, Cole C, Russell BW, Fischl MA, Spira TJ, Snider DE Jr. Tuberculosis, atypical mycobacteriosis, and the acquired immunodeficiency syndrome among Haitian and non-Haitian patients in south Florida. *Ann Intern Med* 1984;101:641-5.
- 2 Doble N, Hykin P, Shaw R, Keal EE. Pulmonary Mycobacterium tuberculosis in acquired immunodeficiency syndrome. *Br Med J* 1985;291:849-50.
- 3 Crompton GK. Steroids in respiratory disease. *Br J Hosp Med* 1982;28:340-8.
- 4 Scully RE, Mark EJ, McNeely BU. Case 2—1986. Case records of the Massachusetts General Hospital. *N Engl J Med* 1986;314:167-74.
- 5 MacFadden DK, Edelson JD, Hyland RH, Rodriguez CH, Inouye T, Rebeck AS. Corticosteroids as adjunctive therapy in treatment of *Pneumocystis carinii* in patients with acquired immunodeficiency syndrome. *Lancet* 1987;i:1477-9.

(Accepted 15 October 1987)

Department of Chest Medicine, St Mary's Wing, Whittington Hospital, London N19

T MASUD, MA, MB, senior house officer
E KEMP, MB, BS, senior house officer

Correspondence to: Dr T Masud, Department of Dermatology, St Bartholomew's Hospital, London EC1A 7BE.

Transvaginal ultrasound in suspected ectopic pregnancy

Transabdominal ultrasound is performed in most suspected ectopic pregnancies. Its main value lies in ruling out intrauterine pregnancy. The appearance of the adnexa with ectopics is rarely diagnostic, as an extrauterine gestational sac and fetus are seen in fewer than 5% of cases.¹

After experience of transvaginal scanning in infertility, with its superior quality and greater acceptability to patients, we have broadened its application to investigate gynaecological emergencies. We report its use in assessing suspected ectopic pregnancies.

Case reports

Case 1—A 30 year old primigravida presented with pain in the lower abdomen and vaginal bleeding at seven weeks' amenorrhoea. The bladder was emptied for

bedside testing for human chorionic gonadotrophin with a urine monoclonal antibody enzyme immunoassay (Abbott Test Pack, Abbott Laboratories, North Chicago IL 60064), which detects urinary β human chorionic gonadotrophin with a sensitivity of 50 mIU/ml.² The result was positive. Transvaginal ultrasound was performed with a Dasonics DRF200 7.5 MHz mechanical sector transducer (95° field of view, 8 cm depth of field). The probe, inserted in a condom with coupling gel on either side, was introduced with the patient in the lithotomy position. Thick echogenic midline uterine echoes suggested a decidual reaction, and a 3.5 cm gestational sac containing a fetal pole was seen in the left tubal ampulla. Both ovaries were normal. Free fluid in the cul de sac contained echogenic debris, which "floated" on pressing in with the probe, suggesting a dependent clot. A leaking tubal pregnancy was confirmed at laparotomy.

Top: Blood and clot in the cul de sac (case 1). Bottom: Gestational sac with fetal pole in the left tube (case 2).

Case 2—A 29 year old was admitted at six weeks' amenorrhoea with left sided pain. There was no bleeding, and urine testing showed the presence of human chorionic gonadotrophin. Transvaginal ultrasound with an empty bladder elicited tenderness over the left adnexa, where a 1.2 cm gestational sac was seen. The uterus was empty. The diagnosis of left sided unruptured tubal ectopic pregnancy was substantiated at laparotomy within 1½ hours of admission.

Comment

The standard investigation of suspected ectopic pregnancy comprises, in order, vaginal examination, urinary pregnancy testing, and eventually transabdominal ultrasound. Urinary testing for human chorionic gonadotrophin and transvaginal ultrasound offer several advantages over the conventional approach.

Firstly, unlike transabdominal scanning, the transvaginal approach does not require a full bladder, which is usually emptied on admission for analysing urinary human chorionic gonadotrophin. Time is not wasted while the patient fills her bladder, either by drinking, which may further postpone any surgical procedure while gastric emptying is awaited, or by intravenous infusions and diuretics, undesirable in the many women who are suspected of having an ectopic pregnancy but are later shown to have intrauterine pregnancies.

Secondly, the better quality of image than is seen with the transabdominal technique is partly due to higher frequencies and thus superior resolution.³ In addition, the transducer is not distanced from the pelvic structures to be