studied 43 patients with chronic myeloid leukaemia for evidence of lens changes. Of these, eight patients undergoing treatment with busulphan had lenticular opacity and two (mean age 54 years) had definite posterior subcapsular changes consisting of central irregularity and polychromatic sheen.

Cataracts have not been reported in leukaemic patients though lesions of other parts of the eyes have been well documented (Allen and Straatsma, 1961). In southern India cataract is rare in young persons and when present is usually congenital, traumatic, or diabetic in origin. In the present patient no reason other than busulphan therapy could have accounted for the cataract. He had been taking busulphan

since 1966 with necessary adjustment of the dose, the average dose range being 1-2 mg daily before the development of cataract.

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Infectious Mononucleosis with Pulmonary Parenchymal Involvement

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Pulmonary parenchymal involvement is an uncommon finding in infectious mononucleosis. A case of infectious mononucleosis is reported in which bilateral pulmonary parenchymal infiltrates simulating septicaemia with metastatic pulmonary involvement occurred.

Case Report

A woman aged 31 was admitted to hospital in March 1969 with a pyrexial illness of 24 hours' duration. Her temperature rose to 41°C, her blood pressure fell to 80/50 mm Hg, and she became semi-conscious. During lucid periods she complained of headache and photophobia. The spleen was palpable and there was appreciable lymph node enlargement in both supraclavicular groups and in one axilla.

Four weeks before admission she had had an illegal abortion by dilatation and curettage for a first-trimester pregnancy. On admission to hospital she had a vaginal discharge and was thought to have developed a pelvic infection complicated by septicaemia. Vaginal swabs and repeated blood cultures by aerobic and anaerobic methods yielded no appreciable growth. These investigations had been undertaken before treatment was begun with intravenous fluids and a broad-spectrum antibiotic regimen consisting initially of penicillin, methicillin, and ampicillin. Kanamycin replaced ampicillin after two days. Pelvic examination including dilatation and curettage did not suggest a source of infection.

On admission the haemoglobin was 9.8 g/100 ml, the W.B.C. was 2,600/mm³ (normal differential), and the platelets were 50,000/mm³. Other blood measurements, results of urine analysis, and C.S.F. were normal. The electrocardiogram showed a sinus tachycardia of 130/min and a chest x-ray picture was suggestive of interstitial pulmonary oedema. After four days of antibiotic treatment there was no improvement in her condition. The results of bacteriological investigations were negative so treatment was stopped. She then became jaundiced, with serum bilirubin 3.9 mg/100 ml, serum alkaline phosphatase 68 King-Armstrong units, and serum alanine aminotransferase 230 units. The radiological changes in the chest were more pronounced (see Fig.). Some of the areas of opacity had become confluent and others showed appearances suggestive of early cavitation, with a small amount of pleural fluid present at

the left base. A Paul-Bunnell screening test for heterophil antibodies was strongly positive. The antibody titre was then measured in saline (1 in 224) after absorption with guinea-pig kidney (1 in 112) and after absorption with ox red cells (negative). The W.B.C. at this time was 6,000/mm³, of which atypical lymphocytes accounted for 62%.

Increased areas of consolidation with a small left pleural effusion.

During the next two days, without further treatment, the patient's level of consciousness improved, her blood pressure returned to normal, and she began to expectorate mucopurulent sputum. Over the following two weeks she continued to improve, her temperature fell, and her conscious state returned to normal. In the same period the liver function, platelet count, and peripheral W.B.C. also became normal and the abnormal chest x-ray appearances began to clear. She had a urinary tract infection due to a coliform bacillus which responded to treatment with nalidixic acid, and oral candidiasis was treated with nystatin suspension by mouth.

When discharged from hospital four weeks after admission she was weak but otherwise symptom-free. The chest x-ray picture, peripheral blood findings, and results of urine microscopy and culture were all normal. At follow-up seven months later she remained well.

Comment

Finch (1969) suggested that in infectious mononucleosis pulmonary parenchymal involvement with radiological appearances similar to those of "atypical viral pneumonia" is to be found in 3-5% of patients. The clinical and radiological studies he cited in support of this statement (McCort, 1949; Arendt, 1950) are unsatisfactory, especially as regards their

criteria for a diagnosis of infectious mononucleosis. In a more recent report of an outbreak of the disease Dunnet (1963) described 80 cases; the diagnostic criteria used were stringent. Though almost all of the 80 cases had evidence of hepatic involvement and 13 (16%) had electrocardiographic changes, only a few had the radiological appearances of enlarged hilar lymph nodes and none had pulmonary lesions.

The present case satisfied the accepted diagnostic criteria for infectious mononucleosis. There was no direct evidence of infection other than of infectious mononucleosis, and the pulmonary changes coincided with the development and remission of the primary disease. It seems likely that the pulmonary opacities were due to foci of infiltration by mononuclear cells or atypical lymphocytes, as has been described in a minority of the few cases coming to necropsy (Ziegler, 1944; Allen and Kellner, 1947; Natvig, 1962). If these pulmonary opacities were indeed due to lymphocytic proliferation, we are reminded that the mechanism of proliferation and above all the limitation of lymphocytic prolifera-

tion in infectious mononucleosis is inadequately understood (Dameshek and Gunz, 1964).

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Hyperaldosteronism from Adrenal Carcinoma

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A case of primary aldosteronism caused by an adrenal carcinoma is reported. It reinforces the view of Spark and Melby (1968) that patients with aldosteronism due to an adrenal adenoma should be treated by removal of the primary lesion (whether adenoma or hyperplasia) rather than medically with spironolactone.

Case History

A man aged 35 was admitted to hospital with a history of pain, tenderness, and weakness in the muscles which had begun one month previously and gradually progressed until the day of admission, when he could not walk. Biceps, deltoids, quadriceps, and adductors and abductors of the hip were weak. He was unable to sit up from a lying position but was just able to lift both legs from the bed. The left knee jerk was obtainable only with reinforcement and the right ankle jerk was absent. Haemoglobin was 13 g/100 ml, W.B.C. 4,800/mm³, and E.S.R. 9 mm/hr (Westergren). Plasma electrolytes were sodium 145 mEq, potassium 1.7 mEq, and bicarbonate 40 mEq/l, and the blood urea was 22 mg/100 ml. Potassium supplements were given by mouth and intravenously on the first day to a total of 600 mEq without appreciable improvement. Continuing potassium supplements of from 250 to 400 mEq a day led to a gradual increase in serum potassium concentration and a return of muscle strength. X-ray examination of the chest and an intravenous pyelogram showed nothing abnormal.

Three months later aortography showed the left kidney to be displaced downwards and flattened. This depression of the kidney over a short period of time suggested a non-benign lesion of the left adrenal, and tomography showed a calcified opacity surrounded

by a soft tissue mass 12 cm in diameter displacing the kidney downwards and the spleen laterally. Further investigation showed aldosterone excretion rates of 48, 30, 43, and 25 µg a day (normal daily range 0-16 µg), and they were unaffected by changing dietary sodium and potassium. Excretion of 17-oxosteroids was normal but that of the 17-oxogenic steroids was 26-47 mg a day (normal daily range 5-20 mg). The normal rate of cortisol secretion suggested that some precursor of 17-oxogenic steroids other than cortisol was being produced. This explanation was supported by the abnormally high 11-oxygenation index of 1.1 (normal mean 0.32 ± 0.19 S.D.) and by the findings of high values for the excretion of pregnanetriol and tetrahydro-11-deoxycortisol. The excretion of 6-β-hydroxycortisol was found to be three times normal. Plasma renin activity was measured by radioimmunoassay for angiotensin 1. Normal range for our laboratory is 0.68 (± 0.25 S.D.) ng/ml/hr in the supine position on a diet containing 215 mEq of sodium and 50 mEq of potassium. The patient's level was 0.84 ng/ml/hr and rose normally on exercise to 1.25 ng/ml/hr.

At operation a large firm tumour with some cysts was removed. Postoperatively the patient made an excellent recovery. The serum potassium rose above 3.5 mEq/l. and stayed there without potassium supplements or spironolactone. The production of 17-oxogenic steroids and aldosterone returned to normal. The tumour measured 15 cm by 14 cm by 10 cm and weighed 1,000 g. It was partly solid, partly cystic, and contained yellow necrotic areas. Histologically it was an adrenal cortical carcinoma distinguished by pleomorphic cells with many mitoses and areas of necrosis.

Hypokalaemia did not recur after the operation but the patient had pulmonary metastases, which were controlled with cyclophosphamide. Presumably they were not secreting mineralocorticoid material.

Comment

There is little doubt that the adrenal neoplasm produced an excess of aldosterone leading to hypokalaemic weakness and virtual paralysis. That the neoplasm was a carcinoma is suggested by the infiltrative nature of the primary tumour, the histological picture of pleomorphic cells with many mitoses, and the appearance of pulmonary cannon-ball metastases. Clinical features of Cushing's syndrome which characterize most adrenal carcinomas were not pronounced. The increased excretion of 17-oxogenic steroids was due to metabolites of non-glucocorticoid precursors of cortisol, and the raised excretion of tetrahydro-11-deoxycortisol is a common feature of adrenal cortical carcinoma (Lipsett and Wilson, 1962). This, together with the increased aldosterone excretion rate, suggests that many biochemical pathways in the tumour were overactive. The only similar case was that reported by Foye and Feichtmeir (1955), who also described an adrenal carcinoma with sodium retention and potassium loss.

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