J. D. ACLAND *ET AL.*: CASE OF RENAL AMYLOIDOSIS

FIG. 1

FIG. 2

FIG. 1.—Kidney showing coarse surface granularity. ($\times 0.85$.)

FIG. 2.—Section of kidney treated with thioflavine-T and photographed in ultraviolet light to demonstrate amyloid deposits (pale areas) surrounding disorganized tubules. Arrowed are two arterioles with amyloid deposits in their walls. ($\times 450$.)

E. I. KOHORN *ET AL.*: ULTRASONIC COMPOUND B-SCANNING

FIG. 1

FIG. 2

FIGS. 1 and 2.—Hydatidiform mole. (a) Anterior abdominal wall at level symphysis. (LS) Longitudinal scan.

FIG. 3.—(a) Anterior abdominal wall at symphysis. (b) Bladder. (h) Foetal head. This patient presented with first trimester bleeding as a case of suspected hydatidiform mole.

FIG. 4

FIG. 5

FIG. 4.—Seven-week gestation. (a) Anterior abdominal wall at symphysis. (b) Bladder. (u) Uterus. (f) Foetus.

FIG. 5.—Ovarian cyst in early pregnancy. (b) Bladder. (c) Ovarian cyst in pouch of Douglas. (h) Foetal head.

the result of a calculus. In the presence of a double collecting system with double ureters the most likely cause in a child would be vesicoureteric reflux. Some milder cases of partial hydronephrosis may perhaps be brought out by a pyelogram performed with deliberate water loading and an increased dose of contrast medium. Angiography should certainly be more widely employed in the paediatric age group than it is now; with some experience it is technically not particularly difficult. On the whole, selective procedures should be reserved for specific problems. Conceivably a small aberrant branch could be missed unless a main aortic injection is made.

Renal Amyloidosis Associated with Gastric Carcinoma

[WITH SPECIAL PLATE BETWEEN PAGES 82 AND 83]

Brit. med. J., 1968, 1, 99-100

Amyloidosis is well recognized in association with certain forms of neoplasia (Azzopardi and Lehner, 1966). However, it has been described only six times in the literature in association with gastric carcinoma (Rosenblatt, 1933; Bannick *et al.*, 1933; Perla and Gross, 1935; Dahlin, 1949; Layani and Benhamou, 1954; Le Coulant *et al.*, 1960), and these reports probably refer to a total of four patients. We wish to report such a case with, in addition, an unusual distribution of amyloid material in the kidneys.

CASE REPORT

A 70-year-old man presented with a month's complaint of anorexia and loss of weight. Three years previously he had had a myocardial infarction and one year previously a prostatectomy. For six months he had been troubled by moderate dyspnoea of effort with angina. A tendency to develop oedema was controlled by small doses of a diuretic.

Examination showed a pale, wasted, ill-looking man. The blood pressure was 140/80 mm. Hg. A liver of normal consistency was enlarged two fingerbreadths below the costal margin. No other abnormality was noted.

Investigations.—The haemoglobin was 9.2 g./100 ml., the red cells showing some hypochromia and occasional burr cells. The white cell count was 6,500/cu. mm., with a normal differential count. The erythrocyte sedimentation rate was 71 mm. in one hour (Westergren). Chest x-ray examination showed calcified deposits in the apex of the right lung. A barium-meal examination showed a low lesser curve ulcer with associated deformity suggesting carcinoma.

Shortly after admission to hospital "acidotic" respirations were noted, the blood urea was 475 mg./100 ml., and the serum bicarbonate was 12 mEq/l. Of five specimens of urine tested for protein three were negative and two showed a slight trace of protein. There were a few red cells only in the deposit and culture was negative. Serum bilirubin was 0.1 mg./100 ml., acid phosphatase 1.5 i.u., alkaline phosphatase 180 i.u., alanine aminotransferase 2 i.u., thymol and zinc sulphate turbidity and flocculation tests were normal.

The patient died in terminal renal failure a fortnight after admission to hospital.

POST-MORTEM FINDINGS

Starting 4 cm. from the pylorus all coats of the stomach were infiltrated and thickened by a carcinoma encircling the whole of a 9-cm. length of stomach. Histological examination showed a poorly

My thanks are due to Mr. David Levi. The illustrations were prepared by the Photographic Department, Westminster Hospital.

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differentiated adenocarcinoma with involvement of local lymph nodes. No other metastatic deposits were found. The liver was macroscopically normal. Both kidneys were contracted, the right weighing 84 g., the left 70 g. There was severe coarse surface granularity with marked reduction in size of both cortex and medulla (Special Plate, Fig. 1). Both renal pelvis were normal with no evidence of recent or past pyelitis. Sections were stained with haematoxylin and eosin and with Congo red and were treated with thioflavine-T. Extensive regions of the interstitial tissues of the medulla were infiltrated with amyloid material deposited in numerous small irregular foci which stained with Congo red and fluoresced in ultraviolet light after treatment with thioflavine-T (Special Plate, Fig. 2). Many blood vessels in the medulla of the affected regions contained amyloid deposits in their walls. There were tubular atrophy and obliteration of glomeruli in the cortex overlying the affected regions, but the sclerotic areas and residual glomeruli contained no amyloid deposits. There was moderate trabeculation of the urinary bladder but no cystitis. The prostate showed residual mild benign hyperplasia. The heart weighed 420 g., with dilatation and hypertrophy of the left ventricle (2.4 cm. thick). There was a patch of fibrosis 2 cm. in diameter affecting the posteroseptal region of the left ventricle. No atheroma or narrowing of the main coronary arteries were demonstrated. The valves and chambers of the heart were otherwise normal.

Apart from basal bronchopneumonia and a "foetal" adenoma of the thyroid 2 cm. in diameter, no significant abnormality was noted in the other organs examined. Only portions of the stomach, representative gastric lymph nodes, left kidney, and thyroid gland were retained for histological examination.

COMMENT

The available evidence suggests that, among neoplasms, the lymphoma group, multiple myeloma, and renal carcinoma may have a definite causal relation to systemic amyloidosis, whereas associations between systemic amyloidosis and other neoplasms have not been reported often enough to exclude a chance relation (Azzopardi and Lehner, 1966). We present another case of the rare association between gastric carcinoma and amyloid disease, because further causal relations between neoplasms and amyloidosis may emerge as more data are accumulated.

Previous reports have not disclosed any unusual features in the distribution or staining properties of the amyloid material in patients suffering from gastric carcinoma. It is most unusual for amyloid to be deposited in the medulla only and to be absent from the glomeruli, as in our patient (Heptinstall, 1966). A few cases have been reported of renal amyloidosis confined to the pelvis (Chisholm *et al.*, 1967). Nephrosclerosis occurs in a proportion of cases of amyloidosis, but it is generally associated with deposits in the glomeruli (Rukavina *et al.*, 1956). In our patient nephrosclerosis appeared to have resulted from compression of the tubules in the medulla by the interstitial amyloid deposits, since the deposits in the blood vessels of the medulla were not sufficient to cause atrophy of the cortex through ischaemia. The absence of amyloid deposits from the

glomeruli would explain why significant proteinuria was not found.

We wish to thank Dr. G. F. M. Hall for his advice; the staff of the Pathology Department, the General Hospital, Nottingham, for performing the laboratory investigations; Mr. T. L. Platts, of the Department of Pathology, Sheffield University, for the photomicrograph; and Mr. E. S. McLeod for preparing the photographic reproductions.

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Lichenoid Eruption Due to Chlorpropamide

Brit. med. J., 1968, 1, 100

In this paper we describe a lichenoid eruption occurring inside the mouth of a patient receiving chlorpropamide (Diabinese) for diabetes mellitus. This drug is a widely used sulphonylurea derivative and may cause various rashes in a minority of patients, but its association with a lichenoid eruption does not seem to have been previously reported.

CASE REPORT

The patient, a temperature-taker in a steel works, aged 53, presented in December 1965 with nine months' thirst and polyuria. A random blood sugar of 480 mg./100 ml. confirmed the diagnosis of diabetes. He was treated initially by a restricted carbohydrate diet, but chlorpropamide 100 mg. daily was given in March 1966; the dose was increased in June to 250 mg. and in August to 500 mg. daily with subsequent good diabetic control.

On 1 November he attended the dental hospital with "white ulcerated sloughs" on the lips (see Fig.). In retrospect he believed these to have begun in June. By 4 November lesions had spread to the mucosa of his tongue and cheeks, and a biopsy at this time showed "appearances suggestive of lichen planus but with more cellular disturbances than average" (Professor J. J. Hodson). Careful examination failed to show any lesions elsewhere on the skin or other mucous membranes. On 22 November his mouth became worse and he could not wear his dentures, so the chlorpropamide was stopped. Five days after this he stated that his mouth symptoms

Left lower lip at first visit, showing raised nature of the lesions.

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had cleared, and when we saw him on 6 December there was no longer any evidence of the lichenoid eruption.

During January 1967 the patient had glycosuria again and restarted chlorpropamide on his own accord. After two days on 500 mg. daily, followed by two days on 250 mg. daily, the mouth lesions recurred, but subsided within four days after he ceased to take the tablets. The next week he again repeated chlorpropamide therapy on his own, and on the third day of treatment the lichenoid eruption recurred inside the mouth and was observed by us on 17 January; but once more it soon cleared when the tablets were withdrawn. Subsequently Dibotin (phenformin hydrochloride) slow-release capsules 50 mg. twice daily have controlled his diabetes and his mouth has remained clear.

COMMENT

Lichen planus is the commonest cause of lichenoid eruptions inside the mouth, but because of the prompt appearance and disappearance of this patient's lesions in relation to taking and stopping chlorpropamide on three different occasions, it seems certain that the lichenoid eruption was induced by this drug. The rapid clearing of the eruption is also quite unlike the slow resolution of the mucosal lesions of lichen planus. It is of interest that the eruption probably occurred only when the daily dose of chlorpropamide was raised to 250 mg.

Maculopapular eruptions, urticaria, exfoliative dermatitis, erythema multiforme, erythema nodosum, Stevens-Johnson syndrome, photosensitivity, purpura, and proctitis (Rothfeld *et al.*, 1960; Tullett, 1966) have been described as rare skin and epithelial complications of chlorpropamide therapy, but lichenoid eruptions due to this drug do not appear to have been previously reported. Lichenoid eruptions may also occur after treatment with gold, organic arsenic, bismuth, mepacrine, chloroquine, hydrochloroquine, and amiphenazole (Sneddon, 1965; Dinsdale and Walker, 1966), and represent yet another condition in which diagnosis and treatment are greatly helped by knowing what drugs the patient has been taking.

We thank Dr. E. W. Skipper for permission to report this case.

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