

M. SLAPAK *ET AL.*: TRANSPLANT LUNG

F. STARER: PARTIAL HYDRONEPHROSIS

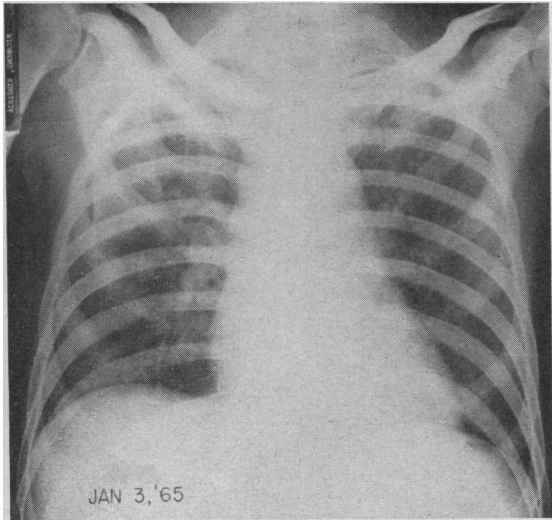


FIG. 5.—Case 3. 82nd-83rd post-transplant day. Arterial desaturation with pulmonary infiltrates.

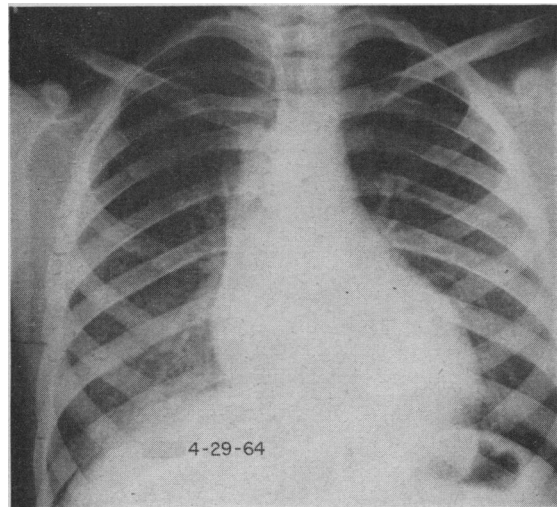


FIG. 6.—Case 4. 59th post-transplant day. Bilateral infiltrates with alveolar capillary block. This responded to corticosteroids.

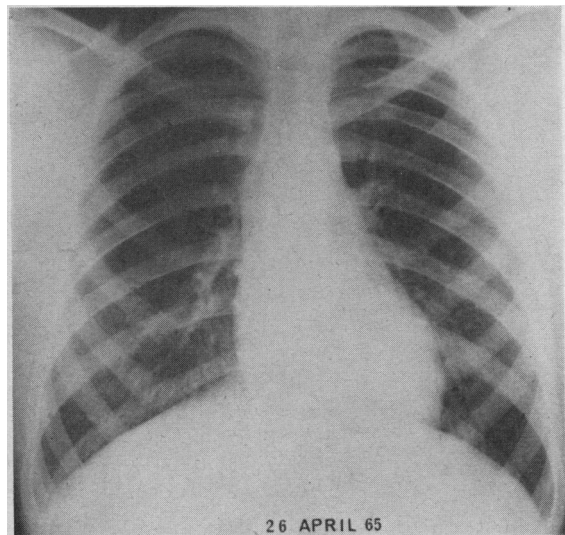


FIG. 7.—Case 4. 419th post-transplant day. One year later the chest x-ray picture was normal. Normal renal function.



FIG. 1.—Intravenous pyelogram showing dilated calices in upper part of left kidney.

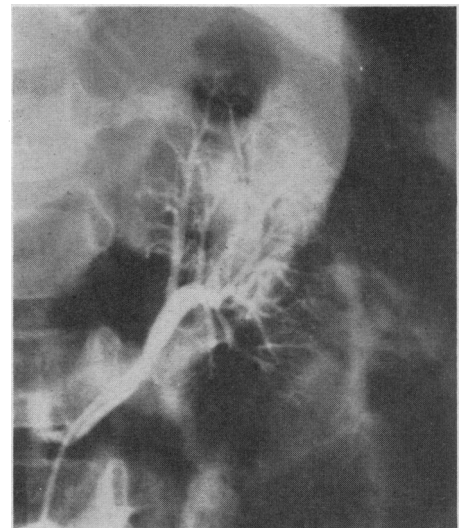


FIG. 2.—Selective renal arteriogram.

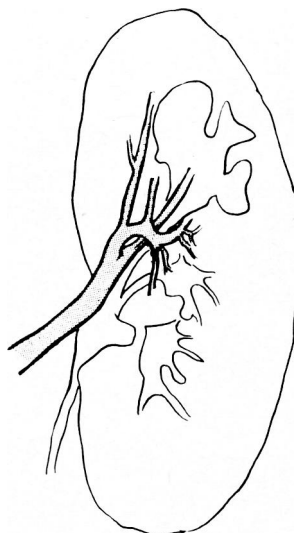


FIG. 3.—Artist's composite drawing to show relation of arteries and infundibulum.

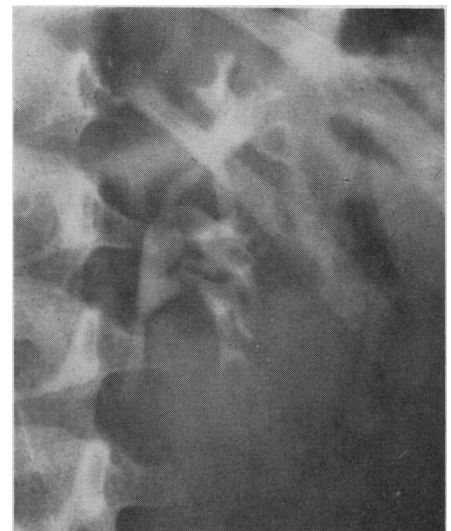


FIG. 4.—Intravenous pyelogram 10 months after operation.

DISCUSSION

The above work is believed to present direct evidence that the Ph¹ chromosome is present in the erythroid as well as in the myeloid cell series. Several workers (Tough *et al.*, 1963; Trujillo and Ohno, 1963; Whang *et al.*, 1963) have, on the basis of good circumstantial evidence, postulated the probable involvement of megakaryoblasts.

It therefore seems reasonable to suggest, on the basis of evidence to date, that the erythroid, myeloid, and possibly the megakaryocytic cell series are derived from a stem cell common to all three types.

Two further patients with chronic granulocytic leukaemia are at present being studied, and a more detailed report is in preparation.

SUMMARY

Chromosome preparations of the bone marrow of a patient with chronic granulocytic leukaemia were made after the cells

had been cultured with ⁵⁹Fe or ⁵⁵Fe. The autoradiograms of these preparations give direct evidence that the Ph¹ chromosome is present in the erythroid cell series.

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Medical Memoranda**Partial Hydronephrosis Due to Pressure
from Normal Renal Arteries**

[WITH SPECIAL PLATE BETWEEN PAGES 82 AND 83]

Brit. med. J., 1968, **1**, 98-99

The appearance of arterial impressions in a pyelogram is now well known (Kreel and Pyle, 1962). It is less widely recognized that normal renal arteries may, rarely, compress the infundibulum and cause a localized hydronephrosis.

CASE REPORT

A 3-year-old boy was admitted to hospital under the care of Mr. David Levi with a history of intermittent haematuria for two and a half months. There had been no other complaints and no abnormalities were discovered on physical examination. Numerous urine samples were normal microscopically and on culture, both for pyogens and tubercle bacilli. Examination of the blood was also normal.

An intravenous pyelogram on 8 October 1965 showed early but definite dilatation of the upper calicine group in the left kidney; no other abnormalities were demonstrated in the urinary tract (Special Plate, Fig. 1). A left selective renal arteriogram was then carried out. Under general anaesthesia a small nylon catheter with a small preformed curve was introduced through the right femoral artery, a modified Seldinger technique being used. The catheter passed quite readily into the left renal artery, and serial angiograms were obtained after the injection of 1.5 ml. of 45% Hypaque (Special Plate, Fig. 2). Comparison of the angiogram with the pyelogram showed that a filling defect in the infundibulum corresponded accurately with the point at which it was crossed by a curving artery, close to a second arterial branch. It was thought that the infundibulum was trapped between the two branches of the renal artery (Special Plate, Fig. 3).

On 25 January 1966 the left kidney was explored by Mr. Levi. The infundibulum in the upper pole was identified and was found to have been caught in a fork in the artery to the upper pole, one branch passing in front and the other behind the infundibulum. There was also a little fibrous tissue in the area. This and the posterior branch were divided. There was no evidence of ischaemia following division of the artery.

The patient made an uninterrupted recovery after the operation and was discharged on the twelfth day. He has remained well

since then, and there has been no recurrence of haematuria. When last seen, 10 months after the operation, there was no evidence of hypertension and an intravenous pyelogram was normal (Special Plate, Fig. 4).

COMMENT

There has been considerable controversy regarding the role of abnormal renal arteries in the pathogenesis of hydronephrosis. It is probable that such arteries with their accompanying fibrous tissue play a significant part in producing a hydronephrosis in some patients, but not in others. This would account for the still unresolved disagreement on this point. We had not encountered, at that time, a partial hydronephrosis caused by normal intrarenal arterial branches. Since then a similar series has been described by Fraley (1966), who records the case histories of four adult patients with right-sided loin pain and pyelographic appearances similar to those in our patient. All were cured by relief of the infundibular obstruction or partial nephrectomy. In two of these patients the obstruction was caused by pressure exerted by an artery and a vein. We have recently seen another boy with a partial hydronephrosis which corresponded accurately in its distribution with an aberrant renal artery arising from the aorta; at operation, however, obstruction was found to have been due to a vein accompanying the artery and not to the artery itself. Another case of a partial right hydronephrosis due to normal renal arteries is described by Frimann-Dahl (1966). Arterial impressions appear particularly common on the right upper group of calices, and this presumably correlates with the fact that five out of the six cases mentioned here have been on the right, the present case being the sole exception.

The evidence for an arterial cause of this patient's hydronephrosis would seem very strong, though the fibrous tissue encountered round the artery may also have played a part. Why the child developed haematuria as the presenting symptom is, however, far from clear. There was never any evidence of a urinary infection as a possible explanation. A further, as yet unresolved, problem is the possibility of the later development of ischaemic hypertension. It seems reasonable to assume that if the kidney had been made ischaemic to the right degree, hypertension would have developed immediately. The child is being followed with this in mind.

Partial hydronephrosis could be caused by stenosis of the infundibulum due to infection (particularly tuberculous) or as

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.

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

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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

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 pelves 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