Case Reports

HYPERTENSION RELIEVED AFTER NEPHRECTOMY FOR RENAL TUBERCULOSIS

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MANOR (1962) reviewed the literature on the subject of unilateral renal tuberculosis causing hypertension. The total number of cases successfully treated by nephrectomy was twenty-two. The present communication reports the occurrence of hypertension in a patient with unilateral renal tuberculous pyonephrosis.

Case Report

A man, aged 22 years, was admitted to Western District Hospital, Glasgow, on April 28th, 1962, for investigation of hypertension. This had been found during a routine medical examination carried out in connection with emigration procedure. The patient had no symptoms; he denied having suffered from headache, dyspnoea, palpitation or excessive fatigue; and there had been no disturbances pointing to a diagnosis of phaeochromocytoma or pyelonephritis. Apart from measles in childhood, he had had no previous illness, and he was unaware of having been exposed to patients suffering from tuberculosis. He had been employed as a butcher for six years. There was no relevant information in the family history.

On Examination. The patient was an alert, well-built man, with warm, moist hands and a fine tremor of the outstretched fingers. There was no goitre, blemish of the skin, clubbing of the fingers or cyanosis, and no oedema or lymphadenopathy. Pulse 100/min., regular, radial arterial wall in palpable; pulses in legs and arms of normal volume. B.P. 200/130 mm. Hg. (R. arm), 190/130 (L. arm), 215/135 (R. leg), 220/135 (L. leg). There were no signs of cardiac enlargement. The second heart sound at the clinical aortic area was accentuated. The right kidney was just palpable. There was no murmur to suggest renal artery stenosis. There was no papilloedema and blood vessels of the ocular fundi were normal. Examination of the other systems showed no abnormality.

Investigations. Hb. 14.0 g./100 ml.; PCV 47%; WBC 8,300/c. mm. (differential normal); ESR (Westergren) 5 mm./hr. Serum urea and electrolytes normal. A mid-stream specimen of urine showed no albuminuria but contained occasional pus cells; culture showed *E. coli* and *proteus*. After deprivation of oral fluid for 16 hours, urinary S.G. 1030.

The injection of 5 mg. of phentolamine intravenously did not produce a significant fall of blood

pressure. The urinary excretion of catecholamines was within normal limits. Radio-iodine studies of thyroid function using ¹³¹I gave normal results. X-ray examination of the thorax showed minimal fibrosis at the left pulmonary apex with a calcified focus in the right lower zone. There was no active disease. Screening of the heart showed the size and pulsation to be within normal limits. ECG showed indirect signs of left ventricular hypertrophy; high voltage of S in V₁ and of R in V₄, V₅ and V₆. X-ray of the pituitary fossa was normal. Intravenous pyelography showed the left kidney function to be within normal limits. There was no dye excretion on the right side, but there was a suggestion of a large right kidney shadow. Attempts at retrograde pyelography on two occasions were unsuccessful; the catheter could not be advanced beyond 2 cm. along the right ureter. The right ureteric orifice appeared to be retracted. The patient was transferred to Stobhill General Hospital, Glasgow, for further investigation. On May 29th, 1962, lumbar aortography was reported (Dr. S. Haase): "There is enlargement of the left kidney with probable hypertrophy of its cortex; it is supplied by two main arteries arising from the aorta about one vertebral body apart at their origin. Opposite the upper left renal artery, on the right side a small vestigial renal artery is noted but there is no evidence of any functioning renal tissue on the right side."

Operation. On June 4th, 1962, transabdominal exposure of right kidney was carried out by one of us (W.G.) and a large pyonephrotic kidney was discovered. There was considerable fibrosis of the peri-renal fat which made stripping difficult and definition of the renal vessels impossible. The fibrosis extended along the ureter as far as could be reached; the ureter was divided above the pelvic brim and the stump ligated. The grossly enlarged kidney—about twice the normal size—was then removed. There were bosses on the surface of the kidney, and incision of one of these bosses released turbid fluid. The ureter was surrounded by densely adherent fibro-fatty tissue.

Histology. Sections from the kidney showed a florid tuberculous pyonephrosis. The renal parenchyma was reduced to a thin rind containing many obliterated glomeruli and it enclosed tuberculomata and caseous pus. A section from the distal ureter showed active tuberculous tissue which had produced gross thickening of the wall.

Progress. His post-operative course was satisfactory and he was discharged on June 15th, 1962—seven weeks after admission. On the second post-operative day the blood pressure was 170/120, on the fourth it was 160/105, and by the eleventh day (on discharge) the reading was 135/90. On June 8th, 1962, intensive antituberculous therapy was started: streptomycin sulphate 0.5 g. b.d. for six weeks, para-aminosalicylic acid 10 g. each day and iso-nicotinic acid hydrazide 200 mg. each day for eighteen months.

Follow-up. When examined on February 19th, 1963, ten months after the operation, he had no complaint. He had no symptoms referable to the urinary tract. His appetite was good and his weight had increased by 10 lb. (4.4 Kg.). B.P. 120/80, X-ray examination of the chest showed no active lung disease and the cardiac shadow was normal. ECG normal. Urological examination on May 15th, 1963, showed a normal bladder, and on retrograde pyelography a normal left kidney.

In August, 1964, more than two years after operation, the patient continued to be symptom-free. The blood pressure was 150/90 and the electrocardiogram was normal.

Comment

Hypertension is not a common occurrence in renal tuberculosis. In more than a thousand cases of renal tuberculosis treated by one of us (W.B.)

this is the first occasion on which co-existing hypertension has been relieved by nephrectomy.

The patient has been under observation for two years since the operation was performed. The blood pressure remains normal. There is no clinical evidence that the remaining kidney suffered damage during the period preceding surgical intervention. It is, of course, impossible to say how long the hypertensive state had lasted. From the nature of the tuberculous lesions it can be surmised that the kidney had probably been a non-functioning organ for many months—and perhaps for a year or two; and during most of this time there was probably some degree of hypertension.

We are grateful to Professor S. Alstead for permission to publish this case, to Dr. S. Haase for the X-ray examinations and to Dr. G. Slavin for his interpretation of the morbid anatomy.

REFERENCES

- MANOR, E. (1962): Amelioration of Asthma and Hypertension after Nephrectomy for Renal Tuberculosis, *J. Urol (Baltimore)*, **87**, 767.

RENAL PAPILLARY NECROSIS AND PHENACETIN: TWO FURTHER CASES

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THE CONNECTION between phenacetin abuse and renal disease was postulated by Spuhler and Zollinger (1953) but reports of this association were confined to the Swiss and Scandinavian literature until 1960. The first case to be described in the United States was that of Moolten and Smith (1960) and the number of American cases reached 18 by 1964. An enquiry by Friend (1963) suggested that many cases, as yet unpublished, were known to American urologists. The findings of the European workers received substantial support from Australia in 1962 when Jacobs and Morris reported a study of 47 cases of renal papillary necrosis occurring in phenacetin takers; simultaneously, another six cases were published by McCutcheon (1962). Some authorities have questioned the importance of the role of phenacetin in the causation of renal disease but recently it has been shown that this compound can cause papillary necrosis in the rat (Abrahams, Rubenstein, Levin and Wunderlich, 1964).

No case of renal papillary necrosis due to phenacetin had been described in the United

Kingdom until recently (Sanerkin and Weaver, 1964). The ensuing correspondence, in the *British Medical Journal*, revealed two more cases (Sanerkin, 1964; Jacobs, 1964) and a further case has been described by Scott (1964). The present paper described two cases seen in Liverpool during the last year. This brings the total of British cases to six and indicates that this condition may be more common in this country than the literature suggests.

Case Reports

Case No. 1

This woman was admitted to hospital for the last time in November 1963 when she was aged 66 years. She had suffered from arthritis for 37 years and had received a course of gold injections when she was aged 40 years. At the age of 53 hysterectomy was performed for carcinoma of the body of the uterus. In 1952, when she was aged 56, it was noted that she was taking compound codeine tablets for the relief of her joint pains. In 1955 she had a course of treatment with phenylbutazone and in the following year a short course of steroid therapy was tried but