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A CASE OF HYPERTENSION DUE TO RENAL ARTERY OCCLUSION

BY

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The cause of severe hypertension in man is not commonly ascertainable, still less commonly can it be demonstrated to be due to a renal mechanism of the type discovered by Goldblatt (1934). Unilateral renal disease causing hypertension is both interesting theoretically and important therapeutically. Since relatively few cases have been reported, it is thought worth while recording a case in which malignant hypertension was caused by a thrombosed aneurysm of the renal artery and was cured by nephrectomy.

Case Report

A man aged 48 was admitted to hospital on June 27, 1954, complaining of sudden pain in his left loin. He had a low fever and was tender in the left loin and iliac fossa; the blood pressure was 140/90 and the urine was free of albumin. He quickly improved and was discharged home.

On November 1, four months later, he was readmitted complaining of severe headache of two weeks' duration. The blood pressure was now 220/140 and the fundi showed early papilloedema with recent haemorrhages and exudates.

Investigations.—The urine contained albumin and a slight excess of red cells. The blood count was normal; W.R. negative; blood urea, 34 mg./100 ml.; urea clearance (maximum) 53% of normal. Intravenous pyelography showed a normal right pyelogram and poor excretion from a small left kidney. Retrograde pyelography confirmed the latter, and urine from the left ureter contained albumin and red cells, but was sterile on culture.

Hexamethonium tartrate by mouth had little effect on the blood pressure, and he was discharged on December 4. A month later he was admitted again as the blood pressure was still very high and the retinopathy unimproved; operation was decided on.

Left nephrectomy was performed on January 14, 1955. A small, grossly scarred kidney was removed along with an aneurysm of the renal artery occluded by thrombus.

Thirty-six hours after operation the blood pressure fell to 90/60, at which level it remained for 48 hours until a noradrenaline drip was begun; oliguria occurred, the urine output being 100 to 500 ml. per 24 hours from the third to the fourteenth post-operative day. The urine was of low specific gravity and the blood urea rose to 417 mg./100 ml.; it was thought that the sudden fall of blood pressure had caused a tubular lesion in the remaining kidney. A month later, at the time of discharge, the blood pressure was 150/100, there was no albuminuria, and the blood urea was normal. By October, 1955, the fundi had returned to normal, and in September, 1956, he remained symptom-free with a blood pressure of 120/80. By January, 1958, the blood pressure had risen to 160/100, but he was well and the fundi were normal.

Pathology.—The kidney was coarsely scarred and showed fibrosis and loss of pattern. Microscopically there were numerous old and recent infarcts with a few small areas of normal renal tissue between. The renal artery showed fibrosis of the media and well-marked lymphocytic infiltration of the vasa vasorum. Where the artery was dilated to form the aneurysm, the histological picture resembled that of syphilitic arteritis, although there was no other evidence of this disease and no positive serology. Obstruction of the blood supply appeared to have taken place in stages, but the remnants of functioning renal tissue were probably supplied by capsular vessels.

Discussion

It seems probable that the episode of pain in June, 1954, was due to a major infarction of the left kidney. The blood pressure was certainly normal then, but had risen to malignant levels four months later. Nephrectomy produced a rapid and sustained fall of blood pressure with a marked hypotensive overswing. Similar hypotension often occurs after removal of a pheochromocytoma and, after removal of an ischaemic kidney, has been commented on by Hunter and McElmoyle (1956), but does not occur in all such cases. There was no evidence of bleeding in the present case, and the hypotensive overswing might have been due to long-standing inhibition of a normal pressor mechanism or possibly to a circulating depressor substance.

Although pyelonephritis is the most frequent unilateral kidney disease to produce hypertension, obstruction of the main renal artery is more closely comparable with the Goldblatt method of producing high blood pressure in experimental animals. It is in such cases that one might most expect to isolate a pressor substance from the peripheral or renal vein blood, although the evidence that such exists is not very convincing in spite of the claim of Margolin *et al.* (1957). Poutasse (1956) lists 21 cases of renal arterial occlusion with hypertension which came to necropsy, and 19 whose hypertension was relieved by nephrectomy; other cases are reported by Perera and Haelig (1952), Hunter and McElmoyle (1956), Stone *et al.* (1957), Haller *et al.* (1957), and Connor *et al.* (1957). Hypertension due to renal embolism is described by Bourne (1954) and by Prinzmetal *et al.* (1942), and due to an aneurysm of the renal artery by Howard *et al.* (1940).

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