Unilateral Renal Vein Thrombosis with Nephrotic Syndrome

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Bilateral renal vein thrombosis, though rare, has been described as a cause of the nephrotic syndrome. However, only a few case reports of unilateral renal vein thrombosis associated with the nephrotic syndrome have been published (Ashner, 1927 ; Pollak *et al.*, 1956 ; Morris *et al.*, 1963).

This report describes such a case. The diagnosis of renal vein thrombosis was suspected from the renal histopathology and radioisotope renography and was confirmed by inferior vena cavography.

Case Report

A 19-year-old youth who worked in a grain shop was referred to us for puffiness of the face and swelling of the feet of five months' duration. The onset was insidious and was not preceded by pain in the loins. There was no history of respiratory symptoms suggestive of pulmonary embolism.

On examination he had a moderate degree of anasarca, mainly in the face and the legs. There was no evidence of collateral circulation suggestive of inferior vena caval obstruction. Blood-pressure was 110/76 mm. Hg and the fundus oculi were normal. There were no other abnormal features.

Examination of the urine revealed a specific gravity of 1018, a quantitative proteinuria of 6 g./day, and, on microscopic examination, a moderate number of granular and hyaline casts and occasional pus cells. Urine cultures, repeated six times, were sterile. Routine blood counts were normal, and E.S.R. (Westergren) was 42 mm. at the end of one hour. Total proteins were 4.12 g./100 ml., and paper electrophoresis of the serum revealed that albumin was 1.082, α_1-globulin 0.249, α_2-globulin 0.874, β-globulin 0.998, and γ-globulin 0.915. Blood cholesterol was 286 mg./100 ml., blood urea nitrogen 11 mg./100 ml., and blood creatinine 1.2 mg./100 ml. The glucose-tolerance curve was within normal limits. The Congo-red test was negative. L.E. cells were not detected. Serum sodium was 132 mEq/l. and potassium 4.2 mEq/l. Bleeding, clotting, and prothrombin times were normal. Fluoroscopy of the chest did not reveal any abnormality. Intravenous pyelogram showed good excretion of the dye on both sides.

Renal biopsy, initially carried out on the left side, showed minimal thickening of the basement membrane of the glomerular tufts, severe degenerative and necrotic changes in the tubules, and oedema and infiltration with chronic inflammatory cells of the interstitium. Renal biopsy on the right side showed normal findings. The presence of interstitial oedema and marked tubular damage which was out of proportion to the glomerular involvement led us to explore the probability of renal vein thrombosis as the cause of the nephrotic syndrome.

Renogram studies were done after an injection of 25 μc of ^{131}I-labelled Hippuran (sodium iodohippurate). Two scintillation probes were placed over the kidneys, which were localized by means of the I.V.P. films, and simultaneous tracings of the pattern of excretion of Hippuran were obtained on the recorder. The renogram showed a lower peak of the vascular phase on the left side, thus supporting our belief that there was left renal vein thrombosis. The renogram curve of the right kidney appeared normal.

Inferior vena cavogram carried out through the retrograde saphenous vein confirmed the diagnosis of left renal vein thrombosis by revealing a block at the origin of the left renal vein.

The patient was given prednisolone in a dosage of 40 mg. daily for 10 days: the oedema subsided, but the proteinuria, though it lessened, persisted. Anticoagulants were not administered.

COMMENT

Renal vein thrombosis with or without thrombosis of the inferior vena cava is being increasingly recognized as a cause of the nephrotic syndrome. The mechanism which causes this association is not well understood. In experimental animals, partial obstruction of the inferior vena cava proximal to the renal veins or of one or both the renal veins themselves leads to persistent proteinuria (Rowntree *et al.*, 1913 ; Morris *et al.*, 1963). Similar proteinuria has been observed after thrombosis of the inferior vena cava or of one or both the renal veins in man (Shattock, 1913 ; Ashner, 1927 ; Derow *et al.*, 1939 ; Vilk, 1940 ; Hasson *et al.*, 1957 ; Bayley *et al.*, 1965). The considerable loss of albumin leads to hypoalbuminaemia and to a reduced colloid osmotic pressure (Squire, 1953). These factors, and other consequences of protein loss, lead to oedema (Pollak *et al.*, 1956).

Renal vein thrombosis may be found in association with hypercoagulable states, renal amyloidosis (Vilk, 1940 ; Harrison *et al.*, 1956 ; Hasson *et al.*, 1957), diabetic glomerulosclerosis (Hasson *et al.*, 1957), or malignancy (Harrison *et al.*, 1956). In many cases, however, the cause is not detected. Except during infancy, primary renal vein thrombosis is rare, probably because of rapidity of blood flow. In a review of 228 cases of renal vein thrombosis by Abeshouse (1945), 40% of the patients were less than 2 months old. In infancy it occurs especially as a complication of acute gastro-enteritis (Harrison *et al.*, 1956).

Clinically, the diagnosis of renal vein thrombosis in a case of the nephrotic syndrome may be suspected when (1) lumbar pains precede the onset (Abeshouse, 1945 ; Pollak *et al.*, 1956) ; (2) collateral circulation is present due to associated inferior vena caval obstruction (Pollak *et al.*, 1956 ; Morris *et al.*, 1963 ; Bayley *et al.*, 1965) ; or (3) there are pulmonary embolic manifestations (Blainey *et al.*, 1954 ; Pollak *et al.*, 1956).

In the case of the nephrotic syndrome described here there were no clinical findings suggestive of renal vein thrombosis. As we have already described the diagnosis was suspected from a disproportion between the gross renal tubular and interstitial changes compared with the relatively minor changes in the glomeruli, an observation already made by Kark in 1955. The radio-Hippuran renogram strengthened our doubts by demonstrating a functional impairment in the renal vascular flow. The diagnosis was established by inferior vena cavography.

The management of this type of case usually includes anticoagulants to prevent extension of the thrombus and pulmonary embolism (Pollak *et al.*, 1956 ; Morris *et al.*, 1963 ; Bayley *et al.*, 1965). In the present case anticoagulants were not thought necessary, as there was no evidence of those diseases already noted with which renal vein thrombosis may be associated.

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R. C. KHOKHANI,* M.D.,
Senior Medical Registrar and Research Associate.
P. RAGHAVAN,* M.D.,
Professor/Director of Medicine.
S. M. SANT,* M.D.,
Assistant Professor of Pathology.
R. D. GANATRA,† M.B., B.S., F.C.P.S., M.SC.,
Scientific Officer.
S. M. SHARMA,† D.M.R.T.,
Scientific Officer.
M. J. SHAH,* M.R.C.P., D.T.M.&H.,
Honorary Physician and Honorary Professor of Medicine.

* K.E.M. Hospital and Seth G.S. Medical College, Bombay 12.
† Radiation Medicine Centre, Medical Division, Atomic Energy Establishment, Trombay, Bombay.

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