Renal Cortical Infarcts in Sickle-cell Trait

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The renal complications of sickle-cell trait are hyposthenuria (Zarafonetis *et al.*, 1954) and massive haematuria from renal papillary necrosis (Chapman *et al.*, 1955 ; Akinkugbe, 1967). These and several other reports have shaken the view that sickle-cell trait is a benign condition. Also, Raper (1952, 1953) claimed that cicatricial obliteration of glomeruli, which he observed from histological examination of necropsy kidneys, was often associated with sickle-cell trait.

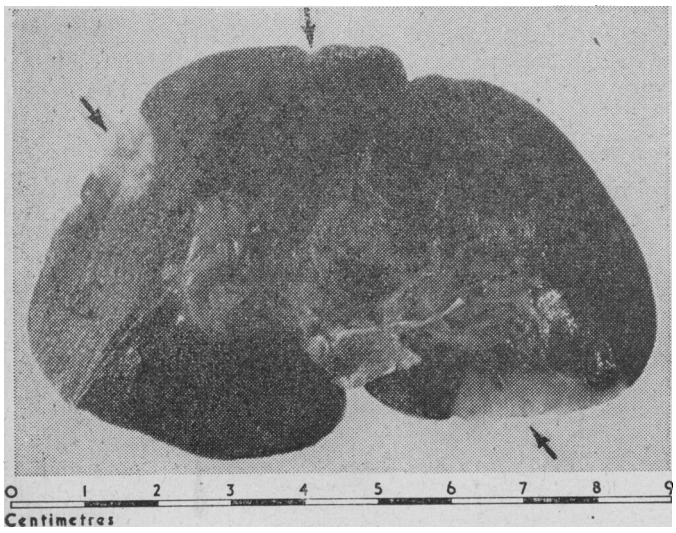
Though several cases of haematuria from renal papillary necrosis have been documented in patients with sickle-cell trait, nothing has been written about renal cortical infarcts in these patients. The purpose of this communication is to record an example of multiple renal cortical infarcts associated with severe hypertension in a young Nigerian with sickle-cell trait.

Case Report

A 16-year-old Nigerian boy was admitted to hospital on 23 September 1967. No history was available on admission. On clinical examination acute pulmonary oedema from severe hypertension was found. The pulse rate was 144, regular, with poor volume. Jugular venous pressure was 5 cm. above the sternal angle. The heart was enlarged, summation gallop was heard, and the blood pressure was 190/150. The lungs had basal crepitations. Fundoscopy showed nothing abnormal. Liver and spleen were not palpable. Urinalysis was normal, except for specific gravity of 1005. He was treated with bed rest, salt restriction, digoxin, bendroflumethiazide, and guanethidine. Control was finally achieved on 80 mg of guanethidine daily, his standing B.P. being 130/80.

After a few days in hospital he gave a history of having had good health until mid-August 1967, when he felt a dull pain of gradual onset in the low back and left loin. He attended another hospital for treatment. The pain disappeared within a week. Three days before the present admission he felt dizzy on standing upright and was breathless and had palpitations on exertion.

Investigations.—The following were found on his admission: Hb 9.8 g./100 ml. ; P.C.V. 33% ; sickling positive ; Hb genotype A.S. ; E.S.R. 79 mm. in the first hour ; blood urea 62 mg./100 ml. ; serum HCO_3^- 21 mEq/l. ; Cl^- 96 mEq/l. ; Na^+ 132 mEq/l., K^+ 4.2 mEq/l. Antistreptolysin O titre, 100 Todd units ; blood W.R. and Kahn test were negative ; serum cholesterol was 233 mg./100 ml. Chest *x*-ray examination showed an enlarged heart with pulmonary oedema. E.C.G. was of left ventricular hypertrophy pattern. Urine microscopy and culture were normal. With control of left ventricular failure the blood urea fell to 30 mg./100 ml. Intravenous pyelography (I.V.P.) showed a non-functioning contracted left kidney ; the right kidney was normal. Cystoscopy showed a very small left ureteric orifice. Retrograde pyelography was therefore not performed. Facilities for aortography were not available at that time. The 24-hour urinary catecholamines were normal.



The resected kidney. Arrows show infarcted areas.

Operative Findings (Professor A. O. Adesola).—On 26 October the left kidney was explored ; it appeared to be of normal size. Three branches of the renal artery were identified, but the superior branch, which appeared to be the largest, was not pulsating. Nephrectomy was performed.

Pathological Findings.—The resected kidney measured 8 by 4 by 2 cm. (see Fig.). The capsule stripped easily, revealing a smooth brownish-red surface with three wedge-shaped yellowish infarcted areas depressed for 2 to 3 mm. below the renal surface. One was at the upper pole and the others in the middle and lower zones. On sectioning, the renal vessels were seen to be filled with clotted blood. The cortex and medulla were clearly demarcated. Sections from various parts of the kidney showed large, small, and medium-sized vessels full of red cells with sickle appearances. Many glomeruli also showed plugging of glomerular capillaries with sickle cells. Some glomeruli were of normal size, while others were small and showed partial or total obliteration of the tufts. In some fields the glomeruli were little affected, but there was marked atrophy of surrounding tubules with an appearance of the so-called incomplete infarction. The appearance of the sections through the grossly infarcted areas was consistent with infarction. Sickle-cell plugging of renal vessels with chronic renal ischaemia and infarction was diagnosed.

The patient was controlled on guanethidine 20 mg. daily ; B.P. when recumbent was 140/105 and when standing 105/75 mm. Hg.

Comment

The multiple renal cortical infarcts in this youth could have been due to systemic embolism, thrombosis, or organic narrowing of the renal vessels. However, there were no clinical signs such as atrial fibrillation to incriminate embolism. Furthermore, careful examination did not show thrombosis or organic narrowing of the renal vessels.

The view is presented that the renal cortical infarcts were due to the sickling process. The distribution of the small wedge-shaped infarcts at the upper, middle, and lower zones of the kidney would tend to support a haematological abnormality such as sickling rather than thrombosis or organic narrowing of a major renal vessel. Infarcts of the renal papillae (Chapman *et al.*, 1955 ; Akinkugbe, 1967), lungs (Moser and Shea, 1957), and spleen during high altitude flying (Rotter *et al.*, 1956) are well-recognized complications of the sickle-cell trait.

Though aortography was not available, it is possible that some undisclosed organic narrowing near the aortic orifice triggered the sickling process. Whether sickling occurred de novo in the renal vessels or was triggered by some organic obstruction unrecognized by us, the sickling process seems a reasonable explanation for the multiple renal cortical infarcts.

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D. Femi-Pearse, M.B., M.R.C.P.ED., D.T.M.&H.,
Department of Medicine.

E. O. Odunjo, M.D., M.C.PATH.,
Department of Pathology.
University of Lagos, Lagos, Nigeria.

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