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Radiological assessment of renovascular hypertension in Ibadan, Nigeria

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Summary

Eight cases of renovascular hypertension (RVH) encountered in the University College Hospital (UCH), Ibadan, Nigeria between 1977 and 1982 are presented and reviewed along with four additional cases, previously reported from this hospital. Seven of these cases were due to fibromuscular disease (FMD); four were due to involvement of the renal arteries by aortic aneurysms and one was due to Takayasu's arteriopathy. No case due to atherosclerosis was seen, which is in sharp contrast to the situation in the western world where atherosclerosis is the commonest cause of renal artery stenosis (RAS). Detection and management of RVH are highlighted and the current concepts and features of FMD are briefly discussed.

Résumé

Huit cas de l'hypertension réno-vasculaire (RVH) qui se sont présentés à l'University College Hospital (UCH), Ibadan, Nigeria entre 1977 et 1982 et 4 anciens cas sont revus. Sept sont dûs a la maladie fibromusculaire (FMD); quatre à la complication des artères rénales par l'aneurysme aortique et un autre à l'arteriopathie Takayasu. Au contraire aux pays occidentaux où l'arthérosclerose est la cause la plus fréquente de la sténose de l'artère rénale (RAS) aucun cas de l'arthérosclerose ne s'est présenté. La détection et le traitement de RVH sont mis en lumière. Les concepts actuels et les traits caractéristiques de la FMD sont discutés brièvement.

Introducton

Renovascular occlusive disease is now generally recognized as the commonest form of surgically remediable secondary hypertension and is estimated to account for about 10% of all cases of hypertension in the western literature (Hillman, 1982).

In Nigeria, the literature on RVH is very scanty. To-date, only four cases have been documented (Abrahams & Parry, 1962; Adi, 1965; Akinkugbe & Jaiyesimi, 1968). Three of these four cases of RAS were associated with the presence of abdominal aortic aneurysms thought to be the result of a peculiar non-syphilitic aortitis often seen in tropical Africa. The fourth was a case of FMD of a renal artery in a 26-year-old Nigerian woman who had a nephrectomy with cure of her hypertension. However, Aderele and Seriki (1974) did mention three paediatric cases of RVH in their review of 138 cases of hypertension in Nigerian children attending the UCH during the 9-year period 1964–1972.

Eight new cases, seen in the last 5 years, in UCH are being added (Table 1). This report does not attempt to define the prevalence of RVH in the population at large as it is confined to patients with hypertension admitted to UCH who had radiological investigation done.

It is the purpose of this communication to direct the attention of clinicians to this curable form of hypertension and to highlight the current ways of detecting and managing RVH so that fatal consequences will be averted in future. Three representative cases are briefly presented to illustrate the practical clinical implications raised in this article while Table 1 is a summary of details of all twelve cases.

Case reports

Case 1

A 30-year-old woman was referred to UCH,

Table 1. Summary of details of twelve cases of renovascular hypertension

Case no.	Age (years)	Sex	Clinical presentation	Initial blood pressure (mmHg)	Angiographic findings	Fate
1	30	F	Recurrent left loin pain, recurrent mid-trimester abortions	170/120	Smooth, tight narrowing of the proximal portion of left renal artery (Fig. 2)	Left nephrectomy Post-op. normotension
2	23	M	Pulsatile abdominal mass and abdominal pain	230/150	Large abdominal aortic aneurysm with a small stretched much underperfused left renal artery & kidney aortitis (Fig. 3). Also iliac artery aneurysms	Left nephrectomy Post-op. normotension
3	24	M	Renal failure	200/150	Bilateral fibro-muscular dysplasia of the middle and distal portions of both renal arteries (Fig. 4)	'Died'
4	21	F	Routine medical test pre-employment	160/100	Smooth left renal artery stenosis and its proximal portion	Left nephrectomy Post-op. normotension
5	16	F	A proven case of Takayasu's arteritis with cerebral and renal involvement	190/130	Complete occlusion of left renal artery at its origin	
6	25	F	Congestive cardiac failure, malignant hypertension	240/150		
7	26	F	Renal failure	180/120	Fibromuscular dysplasia involving the middle and distal portions of both renal arteries	'Died'
8	22	F	Cerebro-vascular accident and renal failure	210/130		
9	21	F	Pulsatile abdominal mass	220/150	Abdominal aortic aneurysm with involvement of the left renal artery. Aortitis	Defaulted
10	26	F	Severe pre-eclamptic toxaemia — twice + 2 stillbirths. Hypertensive between pregnancies	250/140	Stenosis of the main trunk of right renal artery with post-stenotic dilatation. FMD	Right nephrectomy Post-op. normotension
11	21	M	Abdominal pain and pulsatile abdominal mass	240/140	Abdominal aortic aneurysm with left renal artery involvement, aortitis	Left nephrectomy Post-op. normotension
12	21	N		240/130		

Cases 1–8: present series; Cases 9–12: series from 1957 to 1968.

Ibadan in October 1979 because of a 3-year history of poorly controlled high blood pressure and recurrent left loin pain. She had been treated with analgesics and various anti-hypertensive drugs but to no avail. She had experienced three second trimester recurrent abortions due to severe hypertension in pregnancy. Examination confirmed the moderately severe hypertension with a blood pressure of 170/120 mmHg; an epigastric bruit was heard at first but only intermittently on subsequent examinations. Fundoscopy was normal. Urine analysis and culture, haematological profile, electrolytes and urea, electro-cardiogram and chest X-ray were also normal. Excretion urography (Fig. 1) showed a small left kidney, smooth in outline, with considerable delay and increased concentration of contrast excretion. The collecting system showed reduction in size but no ureteral notching was evident. The right kidney showed no abnormality. These findings were highly indicative of a left renal artery stenosis. Angiography revealed a tight smooth narrowing of the proximal portion of the left renal artery with slight post-stenotic dilatation (Fig. 2).

A left nephrectomy was done in January 1980, and histology confirmed fibromuscular dysplasia of the left renal artery and an ischaemic left kidney. The blood pressure gradually returned to normal 130/80 mmHg. In March 1981, 15 months post-operatively, she was booked into the ante-natal clinic. She remained normotensive throughout her pregnancy, which she carried to term, and successfully delivered a live baby in November 1981.

Case 2

A 23-year-old male taxi driver was first seen at the UCH General Out-patient Clinic in December 1978 with a 6-month history of recurrent colicky abdominal pain and headaches and had in the previous 6 weeks been conscious of a pulsating mass in the left side of his abdomen. On examination, his blood pressure was 230/150 mmHg and there was slight cardiomegaly. A pulsating swelling was palpable to the left of the midline in the epigastrium, and there was a bruit over it. Fundoscopy showed minimal retinal changes of arteriolar narrowing and straightening. A chest X-ray and an electrocardiogram confirmed slight left ventricular enlargement and hypertrophy. A right transfemoral abdominal aortogram (Fig. 3) showed

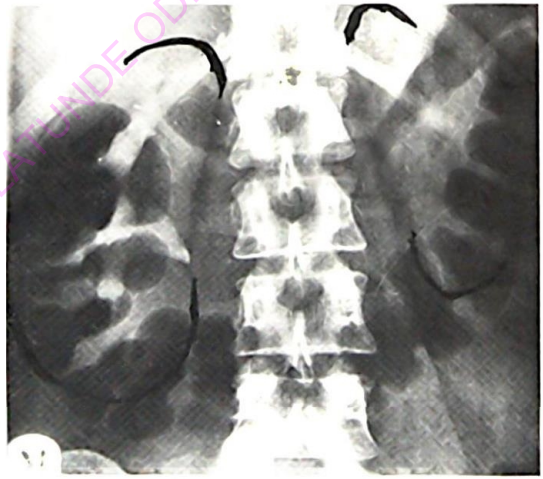

Fig. 1. Excretory urogram on Case 1. File at 5 min. (a) Reduced left renal size; (b) differential contrast excretion — the left pelvi-calyces not yet outlined.

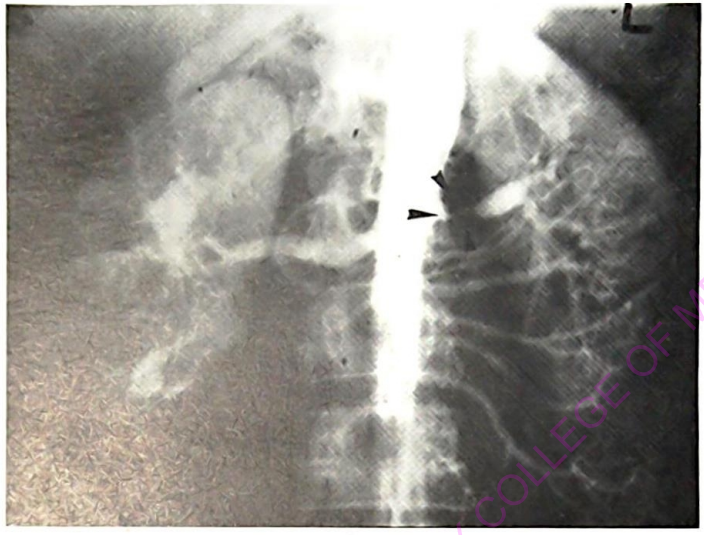

Fig. 2. Angiogram on Case 1. Tight stenosis of proximal half of left renal artery (arrows).

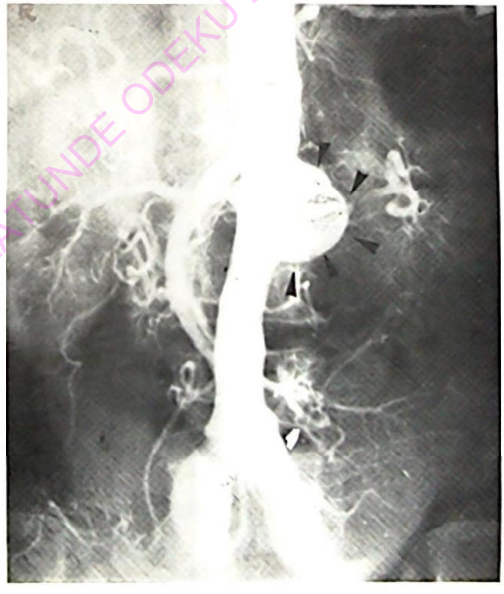

Fig. 3. Case 2: flush aortogram showing aortic aneurysm involving the origin of the left renal artery (arrows)

aortitis and multiple aneurysms of the iliac arteries and abdominal aorta with involvement of the left renal artery; the left kidney was shrunken and contracted. A delayed abdominal film after the angiography showed a poorly functioning small left kidney with faint opacification and a slightly hypertrophied but normal right kidney.

A left nephrectomy was subsequently performed. The left renal artery was stretched, narrowed and thrombosed and the left kidney was ischaemic and shrunken. Post-operatively the blood pressure gradually returned to normal. However, 3 years after surgery the patient died of rupture of his aneurysm.

Case 3

A 24-year-old male undergraduate was admitted into UCH as an emergency in February 1982, with features of chronic renal failure. He had suffered from hypertension for over 5 years and had been on various anti-hypertensives, but the hypertension had responded poorly to drug therapy. Serum electrolytes and urea, chest X-ray and an excretion urogram had been recorded as normal. On examination he was febrile and pale with moderate bilateral pedal oedema. His blood pressure was 200/150 mmHg and there was clinical cardiomegaly. His blood urea was 34 mmol/l with a packed cell volume of 19%.

He was managed as a case of acute on chronic renal failure with hypertension. This included repeated peritoneal dialysis. Renal angiography, using minimal quantity of contrast medium, revealed a beaded appearance of the middle and distal portions of both renal arteries characteristic of medial fibroplasia (a variety of fibromuscular dysplasia) with tiny mural aneurysms (Fig. 4). Both kidneys were small. Three weeks after admission, the patient, who had not shown any clinical improvement, had lapsed into a deep coma and died. An autopsy confirmed fibromuscular dysplasia of both renal arteries.

Discussion

In the western world the commonest cause of RAS is atherosclerosis, which usually occurs after the age of 40 and may or may not be associated with an elevated blood pressure. Two-thirds of renal artery lesions are actually caused by atherosclerosis with a male:female ratio of approximately 2:1 (Holley *et al.*, 1964). In the Co-operative Study of RVH, atherosclerosis and FMD accounted for approximately two-thirds and one-third, respectively, of cases. The rarer causes (aneurysms, thrombosis, emboli, arteriovenous fistulae, miscellaneous arteritides and renal artery narrowing by extrinsic bands or renal or extra-renal masses)

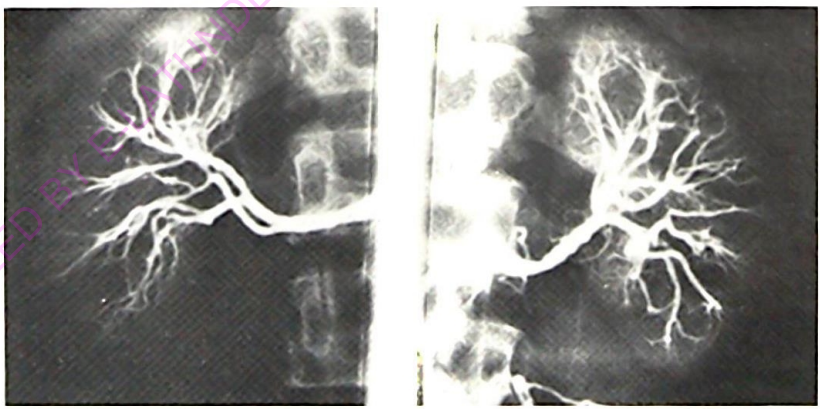

Fig. 4. Case 3: renal arteriograph showing 'beaded' appearance of both renal arteries (typical of fibromuscular disease).

accounted for about 5% (Holley *et al.*, 1964).

However, in this series of twelve cases in indigenous Nigerian patients (Table 1), seven were due to FMD; four were due to involvement of the renal arteries by aortic aneurysm and one was due to Takayasu's arteriopathy.

All patients were young Nigerians, aged between 16 years and 30 years. All the four cases (Cases 2, 9, 11 and 12) of RVH associated with aortic aneurysm had nephrectomy with cure of their hypertension. The single case of Takayasu's arteriopathy was the only patient in a series of four cases reported who had renal artery occlusion with consequent renal hypertension (Ogunbiyi, Falase & Sekoni, 1981). She died, not from the hypertension but from severe extensive involvement of carotid and vertebral arteries. Of the seven cases of FMD, the three with unilateral lesion had surgery (nephrectomy) and all became and remained normotensive, but all the four patients with bilateral renal artery disease died from cardiac, renal and/or central nervous system complications.

It is noteworthy that two of the three cases of RAS due to FMD had severe hypertension in pregnancy with consequent foetal wastage.

Detection of a renovascular cause for hypertension depends on a high index of clinical suspicion. There are no absolutely reliable distinguishing clinical characteristics to aid the physician in making a diagnosis. Young hypertensives (i.e., under the age of 40), especially females or those poorly controlled on multiple drugs, should be suspected. Hypertensives in whom abdominal bruit is heard, even if intermittently, as in Case 1, must be further investigated, as should patients with recent acceleration of their hypertension. Useful investigations that should be carried out include excretory urography, isotope renography, renal arteriography and renal vein renin studies. Angiographic demonstration of an arterial abnormality in a patient with hypertension is no guarantee of causality. However, the search for renovascular disease terminates when renal angiogram is interpreted as normal. Current thinking holds that all renovascular lesions causing hypertension do so via hypersecretion of renin. In order to prove the cause, evidence of increased renin secretion must be looked for by sampling renal venous blood for renin activity. Final proof rests on long term cure or improvement of hypertension after clinically adequate reconstructive surgery, nephrectomy or angioplasty — a proof on which the diagnosis in this series was based since estimation of renin activity was not done pre-operatively due to lack of facility at the time.

Treatment of renovascular occlusive disease is either surgical or by interventional radiology by means of percutaneous transluminal angioplasty (PTA). Operative intervention is usually recommended following failure of drug therapy and operative options include bypass grafts, reconstructions, enderterectomy and transluminal dilation. Primary nephrectomy is undertaken only for irreparably diseased ischaemic kidneys. Renal angioplasty using Gruntzig flexible double-lumen balloon catheter is a recent development to treatment of renovascular occlusive disease. RAS of arteriosclerotic origin has been most commonly chosen for angioplasty but those due to FMD and graft stenosis have been performed with moderate success. Renal angioplasty which may be repeated, if need be, is used to control hypertension or preserve renal function (Schwarten *et al.*, 1980).

Fibromuscular dysplasia was first described in the late 1950s (Poutasse & Dustan, 1957; De Camp & Birckett, 1958). The aetiology remains unknown. It was thought to be present from birth, a form of generalized arterial dysplasia, but recent reports have described the development of FMD in patients who had undergone renal angiography with demonstration of normal vessels 12 years previously, suggesting an acquired rather than a congenital disorder (Aurell, 1979). The higher incidence in smokers suggests that non-specific vascular damage can promote the development of this condition (Mackay *et al.*, 1979). Women are more often affected than men with a female to male ratio of 4:1, multiparous women being particularly at risk. FMD is bilateral in 30–60% of patients. In the present series 57% showed bilateral lesion and the female to male ratio is 5:2. It is estimated that 40% of renal hypertension is secondary to FMD.

The diagnosis is made by angiography. FMD primarily affects the mid and distal portions of main renal artery but recently it has been shown that in approximately 24% of cases both the primary and secondary branches of renal artery were also involved (Houck, 1979). A similar

condition can be found in other medium-sized arteries, especially in the proximal external iliacs and carotids but when symptomatic they have been found with the greatest frequency in the renal and carotid arteries, which receive 20% and 14% of cardiac output, respectively, suggesting increased blood flow in the development of the lesions (Wylie & Wellington, 1960). FMD of the renal artery is associated with an increased incidence of intracranial aneurysms (Houck, 1979).

Fibromuscular dysplasia has been sub-grouped according to cross-sectional histologic involvement as primarily intimal, medial, or adventitial. Intimal and medial involvement predispose to spontaneous renal artery dissection and thrombosis. Of all fibromuscular stenoses the most common (60–70%) is medial fibroplasia with aneurysm, causing the familiar 'string of beads' appearance of the renal artery on angiography (Youngsberg, Shops & Strong, 1977).

The natural history of the condition is obscure but is usually progressive. Lumping all sub-groups, it appears that progression occurs in at least 40–50% of patients but is especially common with predominant intimal fibroplasia, medial hyperplasia and perimedial fibroplasia. Progression occurs less commonly with medial fibroplasia (string of beads). However, patients with renal artery FMD may be normotensive, the lesion remaining dormant, just like patients with atherosclerotic stenosis of the renal artery (Youngsberg *et al.* 1977).

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