RENOVASCULAR HYPERTENSION AND ERECTILE DYSFUNCTION SECONDARY TO POLYARTERITIS NODOSA: A CASE REPORT

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Received August 17, 2001

Abstract : We describe a patient with secondary hypertension and erectile dysfunction due to polyarteritis nodosa. A 30-year-old man was admitted because of hypertension and impotence. Blood pressure was 156/94 mm Hg, and was similar in both arms. Superficial sensation was diminished in the soles of the feet. Plasma renin activity was elevated. Intra-arterial digital subtraction angiography (DSA) of the renal arteries showed bilateral multiple microaneurysms in peripheral arterial branches between the interlobular and arcuate arteries of both kidneys. DSA of the internal iliac artery, the internal pudendal artery, and the hepatic artery also showed multiple microaneurysms, as well as focal stenoses. A diagnosis of organic (vascular) erectile dysfunction was made based on findings by the Rigi-scan (Dacomed Inc.). The patient, then, had polyarteritis nodosa presenting hypertension and erectile dysfunction.

Key words: erectile dysfunction, polyarteritis nodosa, renovascular hypertension

INTRODUCTION

Secondary hypertension, arising from various established causes, accounts for fewer than 5 % of all causes of systemic hypertension. Patients identified as having secondary hypertension sometimes can be cured by surgery or other specific treatment. Polyarteritis nodosa, one cause of secondary hypertension, is a necrotizing multisystem vasculitis affecting small and medium-sized muscular arteries, characteristically renal and visceral arteries[1]. Hypertension, a common complication of polyarteritis nodosa, has a reported prevalence of 25% to 71% among patients with this vasculitis[2, 3]. We describe a rare case showing both renovascular hypertension and vascular erectile dysfunction secondary to polyarteritis nodosa.

CASE REPORT

A 30-year-old Japanese man was admitted to our hospital because of hypertension and impotence in July 1996. The patient had been well until July 1994, when he experienced an episode of erectile dysfunction. He was noted to have hypertension at the time of a routine annual medical checkup in July 1995. No significant past medical history or family history was reported. On physical examination, the patient was 175 cm tall and weighed 65 kg. Blood pressure was 156/94 mm Hg, and was essentially the same in both arms. The pulse was regular at 72 beats /min. No abdominal or carotid bruits were detected. Superficial sensation

was diminished in the soles of both feet. Urinary and hematologic laboratory results were normal. The erythrocyte sedimentation rate was 6 mm/h, and C-reactive protein concentrations were normal. Plasma renin activity and plasma aldosterone concentration were elevated to 16.2 ng/ml/h and 360 pg/ml, respectively. Blood urea nitrogen and serum creatinine concentrations were normal. Antinuclear antibody, hepatitis B surface antigen, and anti-neutrophil cytoplasmic antibody were not detected. Arterial digital subtraction angiography (DSA)of the renal arteries showed multiple microaneurysms in peripheral arterial branches between the interlobular and arcuate arteries of both kidneys (Fig. 1). DSA of the internal iliac artery, internal pudendal artery, and hepatic artery showed multiple microaneurysms and also areas of stenosis (Fig. 2). Erectile dysfunction was diagnosed based on recordings of penile tumescence and rigidity with the Rigi-scan (Dacomed Inc.), including a maximum value of 20% upon intracavernous administration of 40 mg of papaverine hydrochloride. The patient was treated with steroid (prednisolone, 20 mg/day) and an antihypertensive drug (nifedipine, 20 mg/day). Antiplatelet agents (ticlopidine at 300 mg/day and beraprost sodium at 120 μg/day)were given to treat impotence. Blood pressure was well controlled, but impotence showed little improvement.

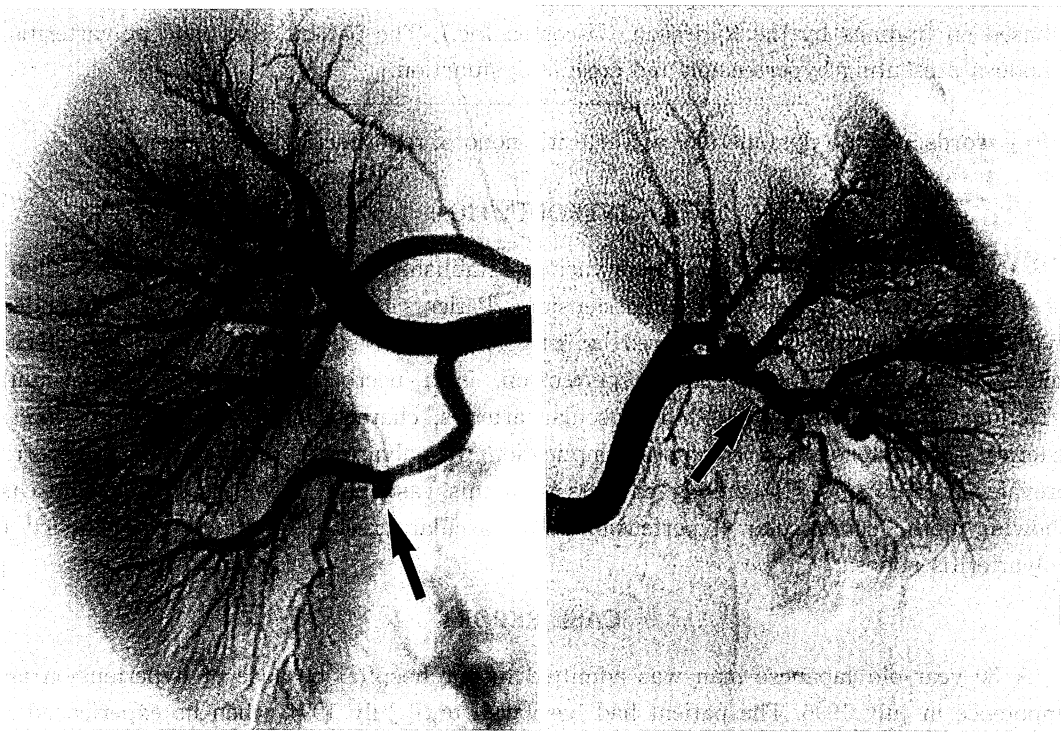

Fig. 1. Arterial digital subtraction angiography of the renal arteries showed multiple microaneurysms bilaterally (arrows). Left panel; right kidney, right panel; left kidney.

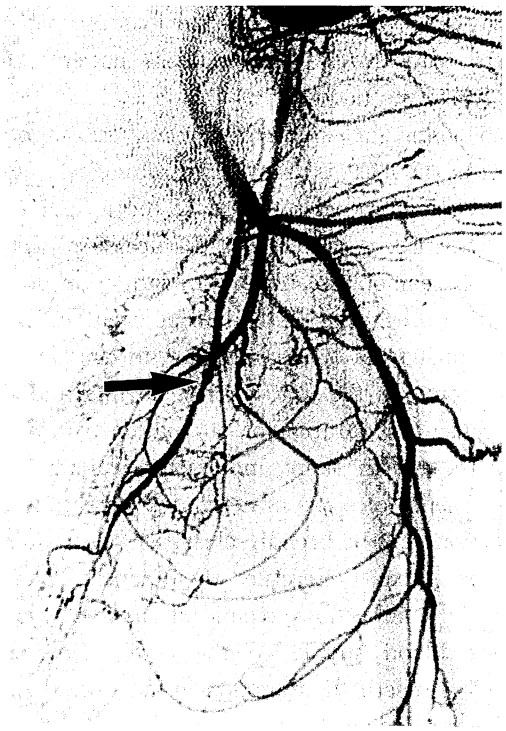

Fig. 2. Intra-arterial digital subtraction angiography of the internal iliac artery showed multiple microaneurysms in the pudental artery (arrow).

DISCUSSION

A diagnosis of polyarteritis nodosa may be confirmed histopathologically, often using a muscle biopsy specimen. Renal biopsy represents a logical choice of procedure in the presence of clinically evident renal involvement, and offers an effective means of diagnosis[4]. However, renal biopsy in suspected polyarteritis carries relatively high risks of bleeding and of formation of an arteriovenous fistula[5]. Further, the predictive value of biopsy for a correct diagnosis of polyarteritis nodosa is surprisingly limited, having been reported as only 15 to 60%[6, 7]. In the absence of easily accessible tissue for biopsy, the angiographic demonstration of involved vessels, particularly renal, hepatic, and visceral arteries, is sufficient for the diagnosis[8-10]. In our patient, multiple microaneurysms were demonstrated in the renal artery, the internal iliac artery, the internal pudendal artery, and the hepatic artery. Polyarteritis nodosa is ordinarily diagnosed according to the American College of Rheumatology 1990 criteria for the classification of polyarteritis nodosa[11].

Polyneuropathy, diastolic pressure exceeding 90 mm Hg, and angiographic abnormalities met these criteria in our patient. Aneurysms due to the medial type of fibromuscular dysplasia and congenital aneurysms also occur commonly, but these noninflammatory aneurysms usually are located between the main artery and lobar artery, and usually are not

multiple[12]. Renal microaneurysms have been reported in some patients with systemic lupus erythematosus[13] and others with drug abuse[14]. These patients can be differentiated by history from those with polyarteritis nodosa.

Our patient showed no signs of inflammation or autoimmune disorder. Arterial inflammation in polyarteritis nodosa is segmental, favoring bifurcations and evolving through four stages[15]. The first of these is edema of the intima and adjacent media. Next, acute inflammation occurs with fibrinoid necrosis, disruption of the elastica, and infiltration by neutrophils and occasional eosinophils. Aneurysm formation or thrombosis may occur at this stage; the inflammatory process may extend to adjacent tissue and veins. In the third stage the cellular infiltrate consists of lymphoid cells; inflammation becomes more chronic, and granulation tissue may form. In the fourth stage healing and fibrosis occur, sometimes obliterating the lumen. Because the duration of our patient's illness was 2 years, he might have been in the third or the fourth stage, in which inflammation diminishes. This time factor may account for the absence of signs of active inflammation in our case.

Causes of erectile dysfunction may be broadly classified as organic (vascular, neurologic, or hormonal), psychogenic, or mixed. Erectile dysfunction has an organic basis in approximately 80% of men[16].A number of systemic diseases may cause erectile dysfunction, mainlyby compromising the blood supply to the penis. Atherosclerotic vasculardisease accounts for 50% of all cases of erectile dysfunction in menover the age of 50[17]. Any disorder that impairs penis blood flow or damages somatosensory or autonomic pathways innervating the penis may cause erectile dysfunction[18]. Intracavernous papaverine injection has become a basic tool in diagnosing and treating erectile dysfunction[19]. Diagnosis of organic (vascular) erectile dysfunction in this case was based on findings in the Rigi-scan, which showed a maximum of 20% upon administration of 40 mg of papaverine hydrochloride. Therefore, the etiology of the patient's erectile dysfunction appears to be vasculitis due to polyarteritis nodosa.

Corticoids are valuable for treatment of polyarteritis nodosa if given before irreversible organ dysfunction develops. Because inflammatory findings had disappeared, our patient received corticoids at 20 mg/day as maintenance therapy, with no initial pulse. In conclusion, our case demonstrates that erectile dysfunction associated with hypertension can present the initial presentation of polyarteritis nodosa.

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