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Variable kidney position mimicking renal artery branch stenosis in a Tc-99m-DTPA captopril scintigraphy

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Tc-99m-DTPA captopril scintigraphy was performed in a patient with suspected renovascular hypertension. Markedly impaired right renal function (Glomerular filtration rate (GFR) values for the right kidney = 14 ml/min, left kidney = 79 ml/min) was detected in the initial captopril study. Only lower pole activity of the right kidney was observed during the whole study. Since prior ultrasonographic examinations have shown bilateral normal kidney parenchyma, branch stenosis of the right upper pole was suspected. Besides significant function improvement in the following baseline study (GFR values for the right kidney = 59 ml/min, left kidney = 79 ml/min), the right kidney, this time normally shaped, was visibly higher positioned. Because of the possibility of mobile kidney and/or branch stenosis, the patient underwent selective renal angiography. A long pediculed right kidney without renal artery stenosis was found. The final diagnosis was essential hypertension. Kidney position anomalies could influence the reliability of the captopril scintigraphy, particularly when a theoretical kidney depth formula is employed for the attenuation correction.

Key words: captopril, renovascular hypertension, Tc-99m-DTPA scintigraphy

INTRODUCTION

DESPITE A RELATIVE LOW PREVALENCE of 1% to 4% in the general hypertensive population, renovascular hypertension (RVH) is caused by unilateral or bilateral stenosis of the main renal artery, branch arterial stenosis, whole kidney or focal renal infarction, aneurysms and arteriovenous malformations.[1] Numerous tests have been suggested to detect RVH, but many of these tests are of limited value because of their low sensitivity or low specificity.[2] In the recent years, many of the studies have shown that when the renal perfusion is reduced, as seen in renal artery stenosis, the transcapillary pressures which maintain the forces to drive glomerular filtration are sustained by a preferential increase in efferent arteriol resistance. Angiotensin converting enzyme (ACE) inhibitor captopril reduces the angiotensin 2-dependent efferent arteriolar resistance, resulting in a reduction in transcapillary forces, therefore reducing renal function (glomerular filtration rate) in the kidney distal to stenosis.[3] This effect is not seen clinically because of the compensatory function of the other kidney, but can be revealed by radionuclide methods.[4] Significant changes in renal split function and renogram curve patterns in captopril studies compared to baseline scintigraphies are the determining parameters in RVH-diagnosis. Fixed renal position and depth are of paramount importance in this quantitative study. We present a case of mobile kidney mimicking unilateral renal artery branch stenosis.

CASE REPORT

Captopril renal scintigraphy was planned for a 35-year-old female patient with suspected RVH (180/95 mmHg). Plasma renin activities were in the upper limit of normal (2.9 μg/h/l in the lying position and 6.8 μg/h/l in the upright position under a normal sodium diet). Renal ultrasonography was unremarkable. The patient maintained a regular diet for the study and the medication was ceased one week before the captopril study. A baseline study was performed, five days after the captopril study with a circular head digital gamma camera (Philips, Gamma diagnost Tomo). The patient was hydrated for the captopril and baseline study with 10 ml/kg water orally

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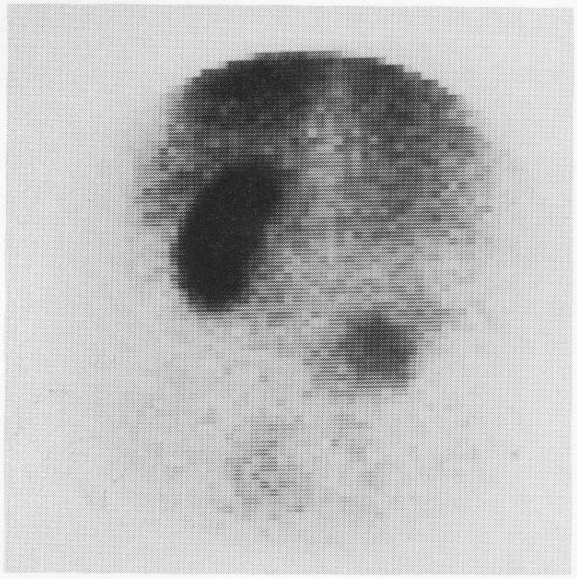

Fig. 1 Captopril study: 2–3 min image, posterior view.

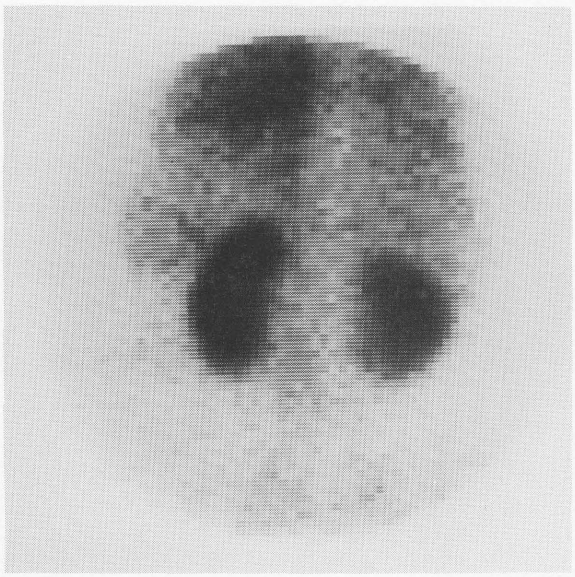

Fig. 2 Baseline study: 2–3 min image, posterior view.

and 250 m*l* serum physiologic intravenously (i.v.). 50 mg Captopril was administrated orally 45 minutes before the study. In the supine position, an i.v. bolus injection of 555 MBq Tc-99m-DTPA was given and serial 2 second interval blood perfusion images as well as 1 minute interval sequential images were recorded in the computer during 30 minutes for both studies. Each kidney was outlined by regions of interest and glomerular filtration rate (GFR) values were calculated by means of the Gates[5] and Tonnosen[6] formulae. The changes in relative DTPA uptake and GFR values for the captopril and baseline studies were 80%, 79 m*l*/min to 58%, 79 m*l*/min for the left kidney and 20%, 14 m*l*/min to 42%, 59 m*l*/min for the right kidney, respectively. The calculated relative DTPA uptake and GFR values were pathological in the captopril study and normal in the following baseline study for the right kidney, supporting the diagnosis of RVH (Fig. 1), however a different localization of the right kidney was observed during the baseline study (Fig. 2). A retrospective evaluation of the whole study strongly indicated a floating right kidney. Nevertheless the possibility of a coexisting upper branch stenosis could not be excluded with certainty. The following selective renal angiography actually showed a long pediculed right kidney without renal artery stenosis (Fig. 3). The final diagnosis was essential hypertension.

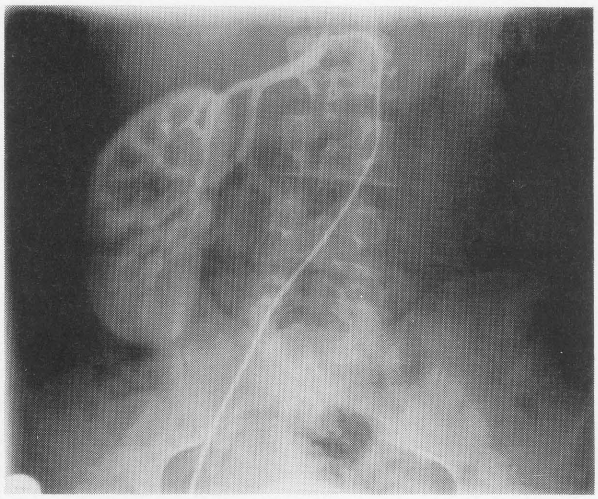

Fig. 3 Selective renal angiography showing mobile right kidney.

DISCUSSION

Renovascular hypertension is a disease potentially curable by surgery (renovascular intervention or nephrectomy).[1] For this reason true (+) results are extremely important. In the presence of a mobile kidney, changing kidney position could cause variable soft tissue attenuation, which consequently results in different GFR values between the baseline and captopril studies. In our case, this was probably the reason for the changes in the renal split function. A partial enhancement of the tissue attenuation over the lower pole by the pelvic bone during the captopril study could explain the marked degree of the function diminution. It may also be speculated that this could be due to a coincidental transient function change in the floating kidney on the day of the captopril study. Considering the fact that the position of the right kidney was different in each of the three studies (captopril, baseline study and renal angiography), the most reliable method in known mobile kidney cases seems to be the measurement of the kidney depth by means of a cobalt marker at the end of the acquisition without moving the patient. Even ultrasonographic depth measurements before scintigraphy were not reliable enough because of

patient movement between the two studies. In our opinion, any case of partial renal hypoperfusion in captopril studies should be examined in additional views to avoid such difficulties in subsequent interpretations.

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