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Renal artery entrapment by the diaphragmatic crus

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Abstract The aim of this study is to describe renal artery entrapment (RAE) by the diaphragmatic crus and to elucidate the diagnostic and therapeutic approach to this entity. From 1995 to 2002, 15 patients (mean age 65) were found to have a RAE. They were investigated by CT scan (n=14) and/or MRA (n=2) for hypertension (n=7), chronic renal insufficiency (n=4) or aneurysms (n=4). The right (n=11) or the left (n=4) renal artery (RA) was involved. The compression was ostial (n=8) or truncal (n=7), and was ≥50% in eight cases. The course of the RA along the aorta on angiographic views (n=8) or a concentric ostial stenosis in a patient free of atheromatous lesions (n=7) were two findings suggestive of an RAE. Seven RAEs were indicated for treatment but only three were treated, by mean of stenting. Among the latter, two stents were patent at 6-month follow-up and one evolved to restenosis because of a stent fracture. RAEs may be suspected on angiographic views and proved by cross-sectional imaging because of specific imaging features. It is of importance to detect this etiology of RA stenosis because angioplasty with stenting is probably not always advisable.

Keywords Renal artery · Ct, renal artery · MR, renal artery · Angiography, renal artery · Transluminal angioplasty, renal artery · Stenoses or obstruction

Introduction

Renal artery entrapment (RAE) by the diaphragmatic crus was first described by D'Abreu [1] who reported in 1962 two cases that were proven by surgery. Since this first description, fewer than 15 cases [2–11] of RA entrapment by the diaphragm and only one case with percutaneous treatment [2] have been reported in the literature. Investigation of RAs with CT or MRA allows us to have a better analysis of relationships between RAs and the diaphragm. However, since the widespread use of these techniques, RA entrapments by the diaphragmatic crus have not been more frequently reported. We report on a series of RA stenoses related to an entrapment by the diaphragmatic crus and discuss the diagnostic and therapeutic approach to this entity.

Materials and methods

From 1995 to 2002, 15 RAEs were prospectively diagnosed in 15 patients. They included nine women and six men, with a mean age of 65 years (40–79).

CT or MR examinations were required for non-stabilized hypertension (n=7), chronic renal insufficiency (CRI) and hypertension (n=4), aneurysms of the abdominal aorta (n=3) or the superior mesenteric artery (n=1). None of the patients who presented an aneurysm had a clinical context of hypertension or CRI.

The technique used for screening was CT (n=14) and MRI (n=2). Five CT scans were performed with a single-row detectors CT (SD-CT) with 1 mm collimation (High-Speed Advantage, General Electric, Milwaukee, USA) and nine with multi-row detectors CT (MD-CT) with 2 mm

collimation (Sensation 4 and 16; Siemens, Erlangen, Germany). We used a 300–350 mg/ml non-ionic contrast media (Xenetix; Guerbet, Aulnay-sous-bois, France) and protocols for contrast media injection were (1) a bolus of 140 ml at a rate of 4 ml/s with SD-CT and (2) a bolus of 50–90 ml at a rate of 4 ml/s with MD-CT. MIP views were reconstructed in frontal, oblique at −15 and 15°, and caudo-cranial projections. In some cases, frontal and oblique MPR views were obtained in order to analyze relationships between the RA and the diaphragmatic crus. MRI examinations were obtained on a 1.5-T unit (Intera; Philips Medical Systems, Best, The Netherlands). Examinations included frontal and axial T1 and T2 sequences and an angiographic study in the frontal plane with 2.4 mm thick images reconstructed every 1.2 mm, after injection of 15 ml gadolinium (Dotarem; Guerbet). MIP views were reconstructed with the same protocol as for CT.

An angiographic examination was performed in nine patients on digital subtraction units General Electric DG 200 (GE, Milwaukee, USA) and Philips Integris Allura (Philips Medical Systems, Best, The Netherlands). A femoral approach was used with the standard five French catheters. A frontal and two oblique series were acquired (−15 and 15°) with contrast media injection in the aorta and series in a frontal projection with selective injections in RAs. In six patients, another series in expiration was obtained.

The percentage of stenosis was visually estimated on MIP projections with CT or MRI, and straight from angiographic views, with the angulation that displayed the most severe grade of stenosis.

In three patients, intra-arterial blood pressure measurements and/or venous samples were obtained in order to demonstrate the causality of the RAE in patient's systemic hypertension.

Three patients underwent an angioplasty with stenting of the RA. For the first patient, an angioplasty with placement of an auto expandable stent (Wallstent 6×28 mm, Boston Scientific, Galway, Ireland) was carried out. For the others, direct stenting was performed with a balloon-expandable stent (Herculink 6×12 mm, Guidant, Santa Clara, USA and Genesis Cordis 6×15 mm, Johnson and Johnson Company, New Jersey, USA).

Patients who were treated were followed clinically at 1 and 6 months and underwent a CT or MR examination at 6 months.

Results

Findings at CT and MR

Muscular entrapments were divided into two groups, according to the length of the compression. (1) In ostial entrapments (n=8) (Figs 1a, 2, 4, 6), the diaphragmatic crus compressed laterally the origin of the artery against the aorta. The lumen of the artery was often oval or ovoid on axial images. The compression was less than 5 mm in length and the artery exited the muscular entrapment anteriorly. (2) In truncular entrapments (n=7) (Figs 1b, 3, 5), the RA went down between the aorta and the diaphragmatic crus and was compressed over a length equal to, or greater than 5 mm. It exited the muscular entrapment anteriorly or inferiorly. In this group, the initial course of the RA along and close to the aorta on MIP views and angiography was very suggestive of the diagnosis (Figs 3, 5).

The right RA was involved in 11 patients (73%) and the left in four. The RAE was located on the main (n=13) or an accessory (n=2) RA. The stenosis was ≥50% in 12 cases and <50% in three. The origin of the RA was proximal to the mid-part of L1 in six cases (40%) and distal to it in the others. The compression was related to a hypertrophic dia-

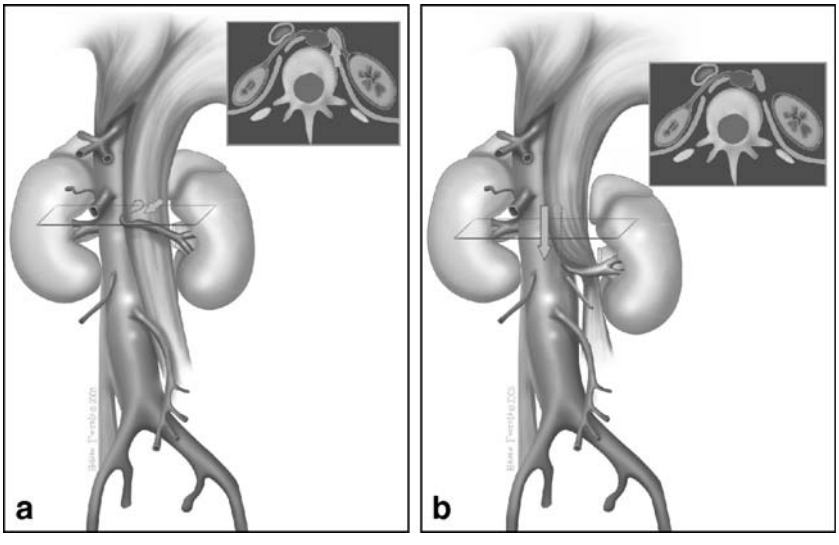

Fig. 1 Classification of compression of RAs by the diaphragmatic crus. **a** Ostial compression. The ostium of the artery is compressed between the diaphragmatic crus and the aorta on a length <5 mm and the artery exits the entrapment anteriorly. **b** Truncular compression. In its proximal course, the artery goes down along the aorta for ≥5 mm and exits the muscular entrapment inferiorly or anteriorly

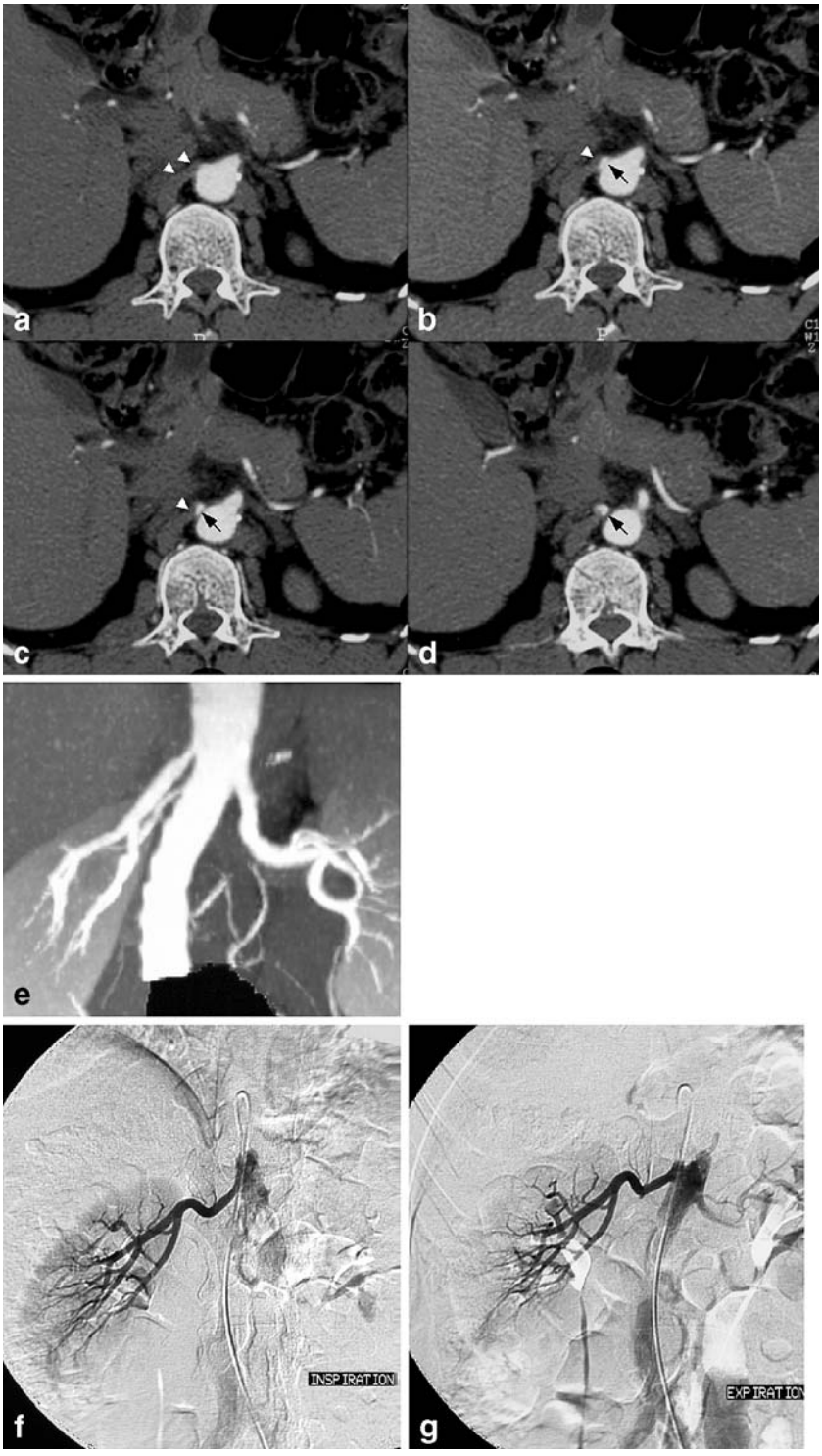

Fig. 2 Ostial compression of the right RA. **a**–**d** MD-CT scan with contrast enhancement, axial slices at the origin of the right RA: the artery (*arrow*) is compressed laterally by the right diaphragmatic crus (*arrowheads*) and then bypasses it anteriorly. **e** Frontal MIP view: stenosis>70% of the right RA. **f**–**g** Renal arteriography in inspiration (**f**) and expiration (**g**): mild stenosis of the right RA that disappears during expiration

phragmatic crus in eight patients (53%) (Figs 3, 6). The coeliac trunk was also compressed by the diaphragm in five cases (33%) and the superior mesenteric artery in three (20%) (Fig. 4).

Findings at angiography

Two angiographic features were suggestive of RA entrapment: (1) an RA descending down along and close to

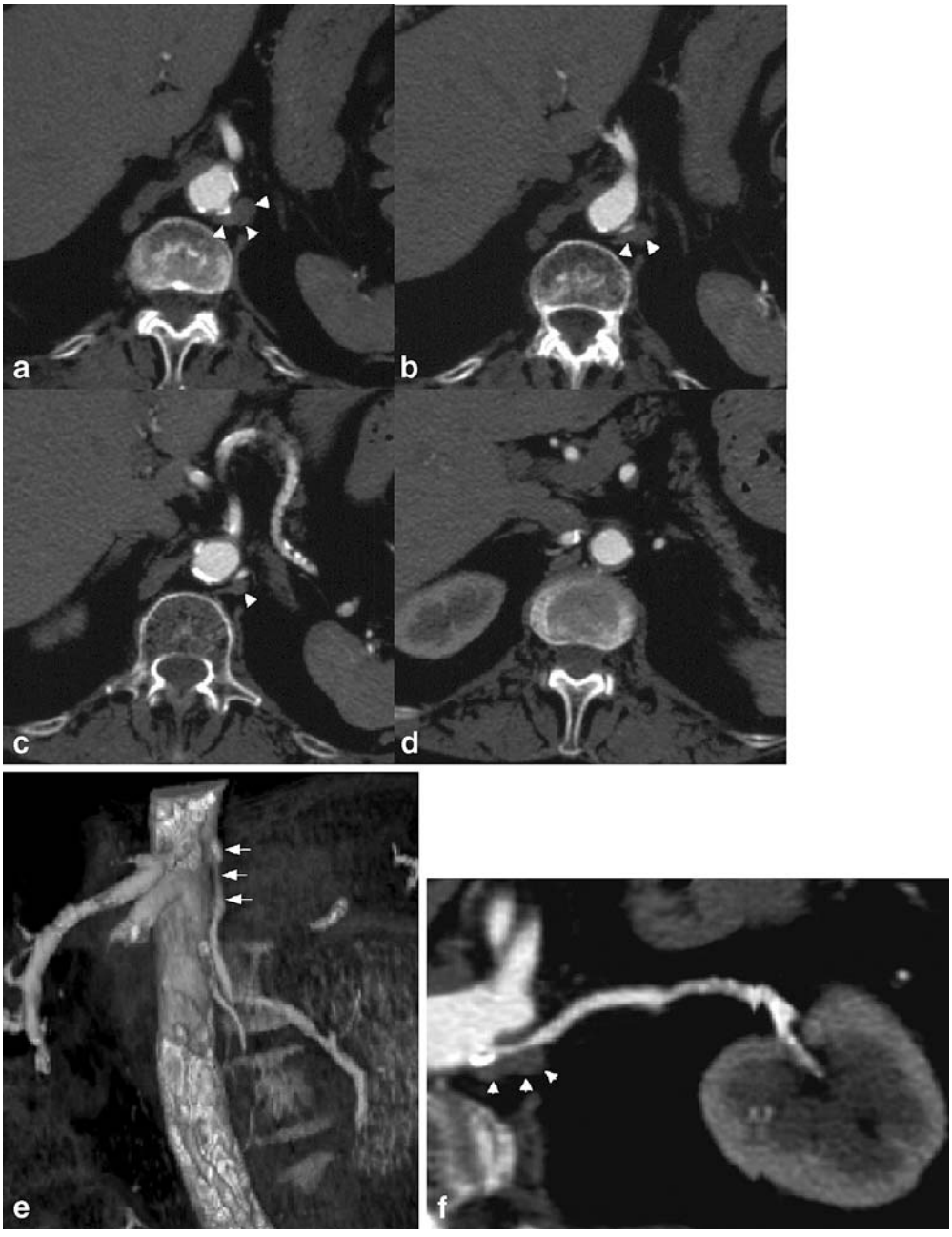

Fig. 3 Truncal compression of the left RA. **a**–**d** MD-CT scan with contrast enhancement, axial slices at the origin of the left RA: the origin of the artery is located above the origin of the coeliac trunk. The artery goes down along the rear of the aorta for more than 10 mm and, in this segment, is compressed between the aorta and the left diaphragmatic crus which is hypertrophic (*arrow-heads*). **e** A 30° oblique anterior VR reconstruction: calcified atherosclerotic lesions involve the aorta and the RAs. The left kidney presents two RAs. The superior artery goes down along the aorta for more than 1 cm (*arrows*). **f** Axial curved MPR along the left RA which demonstrates arterial entrapment between the aorta and the left diaphragmatic crus (*arrow-heads*)

the aorta and (2), a short and concentric ostial stenosis in a patient free of atheroma (Figs 5, 6). Four stenoses were found to be over 50% at CT but less than 50% at angiography; these were ostial (n=2) and truncular (n=2). On angiographic series in expiration, the stenosis was less severe (n=2) (Fig. 2) or unchanged (n=4) (Fig. 6).

Biological results

All venous samples were negative for a hypersecretion of renin and aldostérone.

Treatment

Eight patients did not require treatment because the RA stenosis was <50% (n=7) or the stenosis was located on an accessory artery (n=1).

Three patients were treated by means of stenting the RA. One patient had a truncal compression (Fig. 5) that was treated with a self-expandable stent. At the end of the procedure, the radioscopic control showed that the stent was compressed during inspiration (Fig. 5h) and expanded during expiration. Two years later, the CT scan control and subsequent angiographic examination demonstrated that

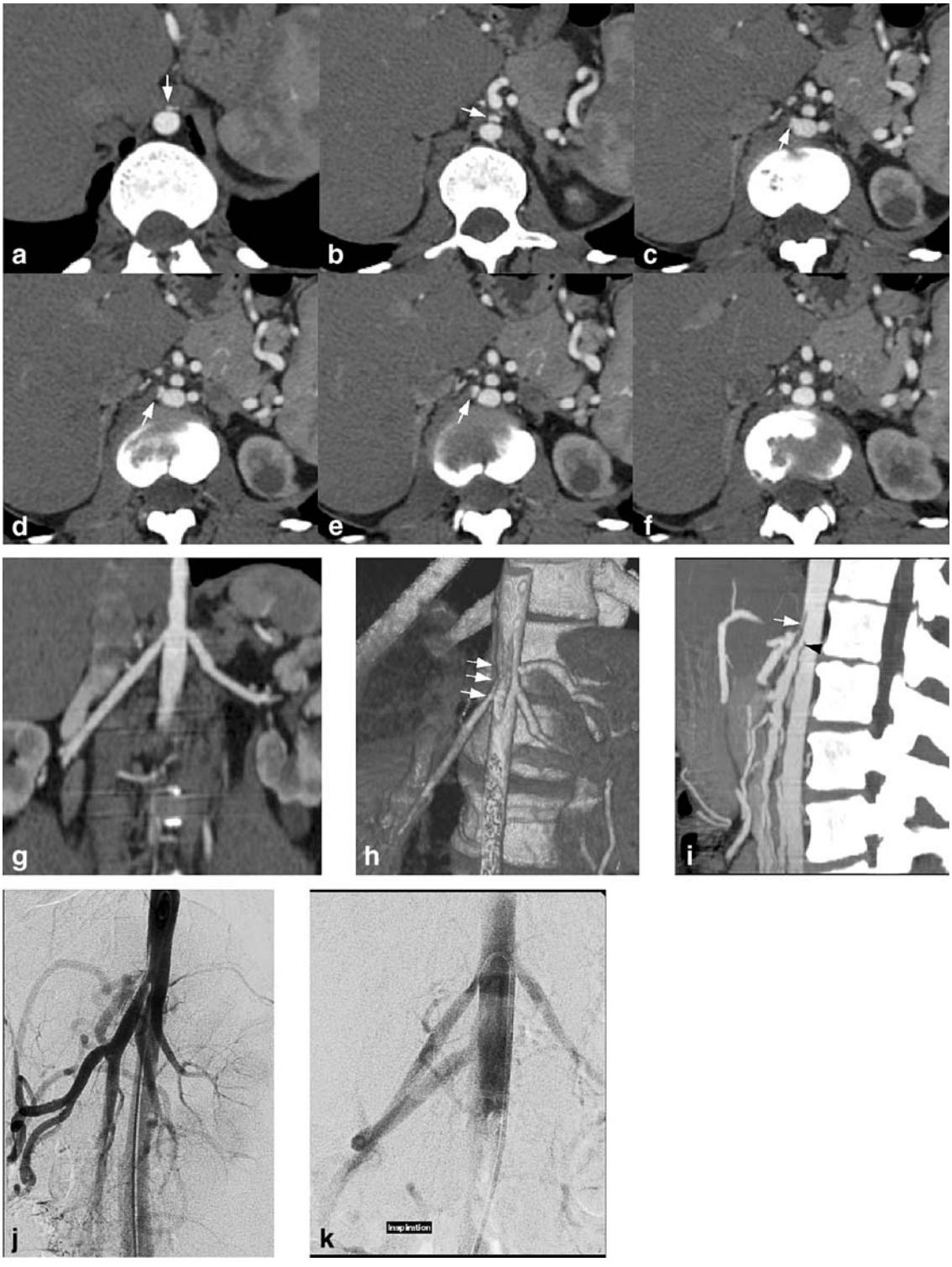

Fig. 4 Entrapment of the celiac trunk, superior mesenteric and right RA. **a**–**f** MD-CT scan with contrast enhancement, slices focused on the origin of the visceral arteries: **a** the coeliac trunk is compressed by the diaphragm (*arrow*), **b** compression of the origin of the mesenteric artery (*arrow*), **c**–**f** short ostial compression of the right RA (*arrow*). Note a round low level attenuation mass of the left adrenal gland. **g** Frontal MPR view showing 50% stenosis of the right RA. **h** Frontal VRT view of the aorta: distortion of the right aortic border and narrowing of the right RA's ostium (*arrows*). **i** Sagittal MIP view: long and tight stenosis of the coeliac trunk (*arrows*) and mild ostial stenosis of the superior mesenteric artery (*arrow-head*). **j** Aortography, sagittal plane: compression of the CT and SMA. **k** Renal arteriography that confirms a 50% stenosis of the right RA. In this case, venous samples were obtained in the inferior vena cava and renal veins. They did not demonstrate any hypersecretion of renin or aldosterone and thus, the left adrenal adenoma was considered to be non-secretant and the right renal stenosis non-hemodynamically significant

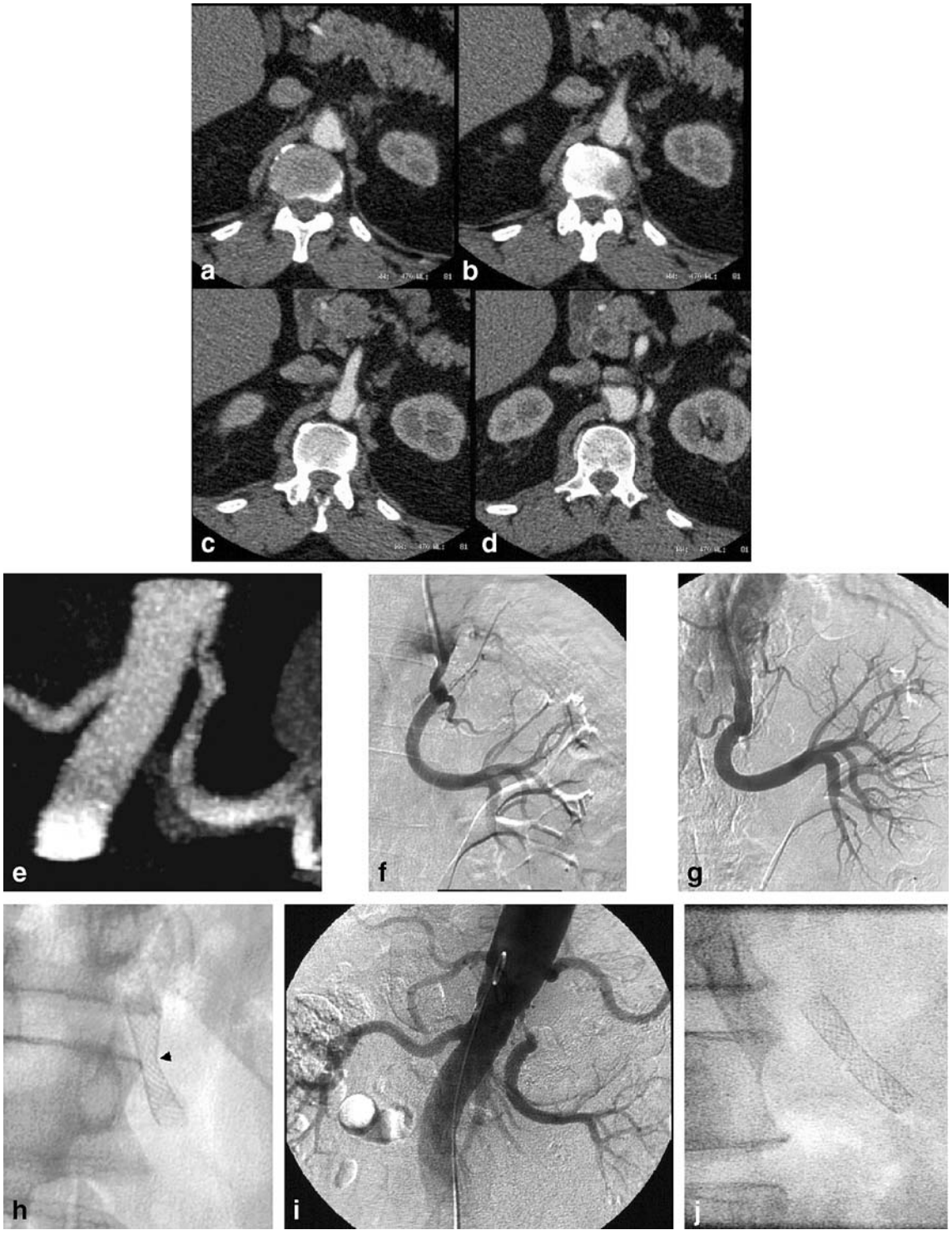

Fig. 5 A 74-year-old man with hypertension. **a**–**d** SD-CT scan with contrast enhancement: long compression of the left RA which originate from the aorta above the SMA. **e** Frontal MIP view: long stenosis of the left RA. **f** Renal arteriography: 50% long stenosis of the left RA with tortuosity of the artery below. **g** Angiographic control after stenting of the stenosis with a self-expandable stent: improvement of the arterial diameter. **h** Radioscopic control of the stent showing compression during respiratory motions (*arrowhead*). **i** Angiographic control 2 years later: restenosis in the stent. **j** Radioscopic control of the stent which is fractured in its middle part

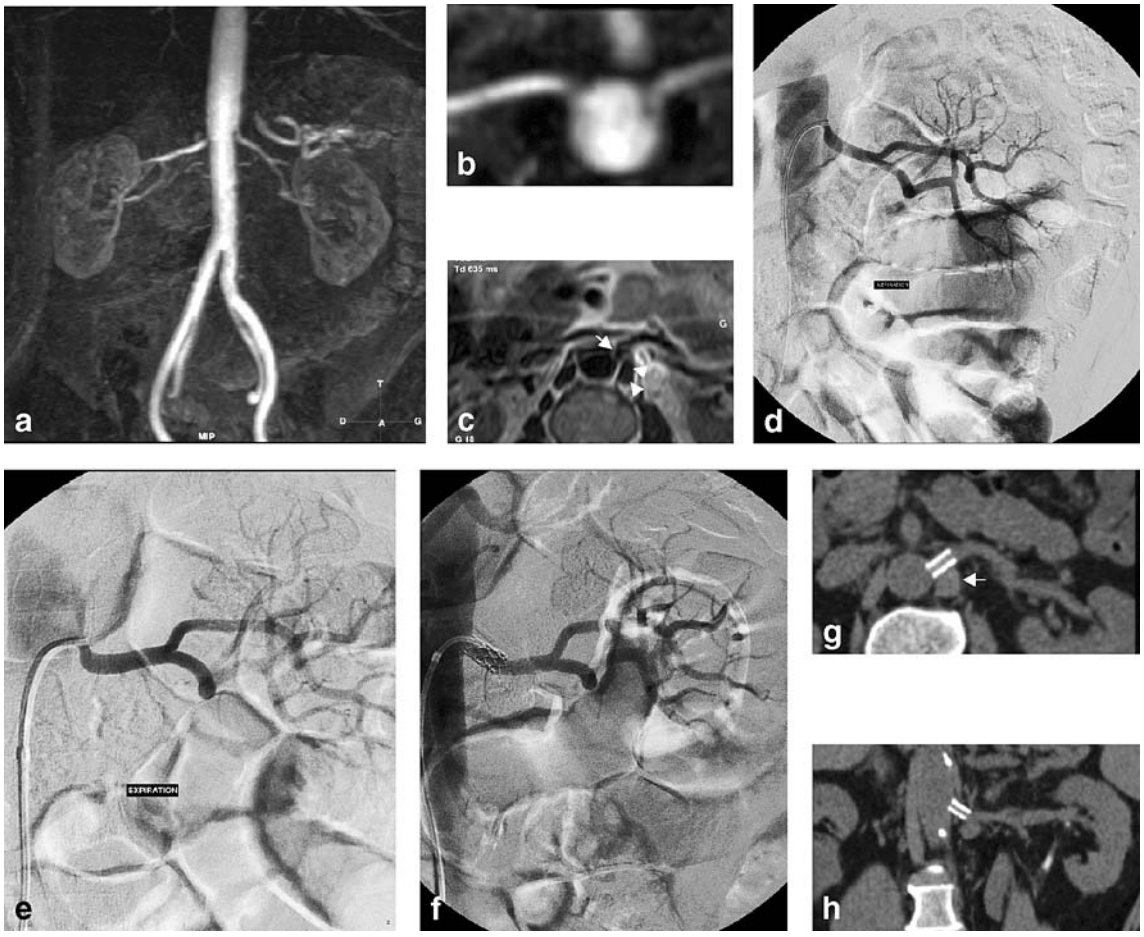

Fig. 6 A 68-year-old man, with hypertension and chronic renal insufficiency. **a** MRA, frontal MIP view: the left RA is compressed against the aortic border over a short segment. **b**–**c** Axial slices at the level of the left RA with MRA (**b**) and T2 TSE (**c**): the left RA bypasses (*arrow*) the left diaphragmatic crus (*arrow-heads*) anteriorly. **d**–**e** Renal arteriography in inspiration (**d**) and expiration (**e**): short stenosis >50% without any change in expiration. **f** Angiographic control after treatment of the stenosis with a balloon-expandable stent : full expansion of the left RA. **g**–**h** MD-CT scan control without enhancement, after treatment, axial (**g**) and frontal (**h**) MPR views: the stent straightens the RA curve. Note the hypertrophy of the left diaphragmatic crus (*arrow*)

the stent was fractured (Fig. 5j) in its middle part and that a restenosis had occurred (Fig. 5i). This patient then underwent a successful surgical aorto-renal prosthetic bypass. This observation has already been published [12]. Two patients had an ostial compression. They were treated with a balloon-expandable stent (Fig. 6). These stents did not collapse during the respiratory cycle. They remained patent on CT follow-up controls at 6 months. Among the three treated patients, the first experienced a significant improvement of his hypertension before the stent collapse and after surgery, but the two latter did not had any significant decrease in their blood pressure level or improvement of their renal function that was previously impaired.

Two patients were indicated for surgical treatment of their RAE but the treatment was not performed. In one patient (Fig. 3), the artery went down along the posterior side of the aorta with a long stenosis and a curve at the end of the muscular entrapment. Considering the clinical context of this 77-year-old women with diffuse atheromatous lesions, stenting the RA through the subclavian artery was at risk for stroke. Moreover, placement of a stent was thought to be at risk for fracture or collapse because the diaphragmatic crus was hypertrophic. Thus, a surgical repair was discussed but, due to the bad condition and the advanced age of this patient, it was decided against. The other patient was planned for a surgical cure of an aneurism of the abdominal aorta. He presented a RAE with a long stenosis involving >70% of the right RA. In this patient too, a precise deployment of the stent at the ostium of the RA appeared to be difficult and this long muscular entrapment was at risk for collapse and fracture of the stent. Thus, a surgical bypass was indicated during the intervention for the aortic aneurism but was not performed because of technical difficulties encountered during surgery.

In two patients, stenting of the RA was thought to be the best treatment because the muscular entrapment was short but a severe atheroma of the aorta contra-indicated it (n=1), or the patient was lost from follow-up (n=1).

Discussion

Since the first report by D'Abreu [1], several cases of RAEs have been reported in the literature [2–11]. Congenital abnormalities such as abnormal musculo-tendinous fibres [1, 2, 6], high ectopic RA origin [5, 6] or hypertrophic diaphragmatic crus [11] were found to be responsible for these entrapments. Demonstrating whether abnormal bundles of fibres are present or not is difficult with cross-sectional imaging; however, RA entrapments seem rather related to an abnormal relationship between the RA and the diaphragmatic crus, the origin of the artery being covered by the diaphragmatic crus. Moreover, in our series, the occurrence of this disease in elderly patients (mean age 65 years) suggests that these compressions are not congenital but may be favoured by changes in relationships between the aorta and musculo-skeletal structures over time.

The best way to detect these arterial compressions is to systematically analyse relationships between RAs and the diaphragm when looking for RA stenoses. Although Duplex ultrasound is an accurate examination for screening RA stenoses [13], it does not allow the analysis of the relationship between RA and muscular structures. CT and MRI allow for depiction of these compressions; however, reports of RAEs detected with these imaging tools are few [3, 5]. This entity may also be suspected on angiographic examination or angiographic-like reconstructions with CT or MRI, when the initial course of an RA is parallel to the aorta or compressed against it.

In our series of RA entrapments, there were discrepancies between CT scan and angiographic results regarding to the grading of stenoses. CT scan examination may overestimate these stenoses because the artery is often deformed (oval shape of the arterial lumen) without significant narrowing of the lumen (Figs 2, 4). Moreover, some of these compressions disappear during expiration. Conversely, angiography may underestimate these stenoses because it may be difficult to bring out of the aorta an initial segment of the RA that is compressed against it. Therefore, it may be sometimes difficult to assess the role of a RAE in generating systemic hypertension. In these cases, Duplex ultrasound and intra-arterial blood pressure measurements may be useful (Fig. 4). Venous sampling should also be of interest.

A review of the literature found only one case report of balloon angioplasty [2] to treat a RA entrapment but no data about the use of stenting. We have treated three patients by mean of stenting. This treatment failed in a case of truncular stenosis (Fig. 5) because the stent was compressed during respiratory motions and this led to a fracture. A balloon-expandable stent would have probably been a better choice because its radial strength is higher. Stenting RAEs with long stenoses exposes to several technical problems. First, the course of the RA along the aorta implies a humeral approach for stenting and in this condition, it may be difficult to precisely control the positioning of the stent in the RA. Second, because the RA is parallel to the aorta, it is difficult to cover the ostium of the RA without a significant protrusion of the stent in the lumen of aorta. Third, at the end of the muscular entrapment the RA is sometimes curved and stenting this angulation exposes to a fracture of the stent because of the movements are important at this point of the RA during the respiratory cycle. Fourth, a hypertrophic diaphragmatic crus and a long compression may probably favour the stent collapse. Two patients with ostial compression of the RA were treated with balloon-expandable stents in order to expand and straighten the artery (Fig. 6). The treatment was successful and we did not notice any collapse of the stent at the 6 month follow-up. Short RA entrapments seem more suitable for endovascular treatment with stenting, first because angulation between the RA and the aorta is less acute. Second, the stent should not be compressed between the aorta and the diaphragmatic crus but should push back the diaphragmatic crus and expand in the retroperitoneal space and thus would be probably less prone to collapse.

With our limited experience, we have observed that, in one case stenting of a long RAE failed and in two cases stenting of a short RAE was successful. Despite it seems more logical to stent short entrapments and indicate for surgery long entrapments, we need for more experience and longer follow-up to define the place of these two treatments.

In summary, RAEs should be suspected each time an angiography or an angiographic reconstruction shows a RA parallel to the aorta in the proximal part of its course or an ostial concentric stenosis is discovered in a patient free of atheroma. A cross-sectional imaging is necessary to demonstrate the muscular compression of the artery. As the lumen of the artery is sometimes deformed but not narrowed, it may be difficult to affirm the causality of the RAE in the hypertension of a patient. The indication for endovascular treatment with stenting or surgical bypass needs to be further defined.

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