Renovascular hypertension (RVH) is defined as the elevation of arterial pressure precipitated by a haemodynamically significant stenosis of a renal artery or arteries (that is, a stenosis greater than 75% of the vessel lumen or 50% with post-stenotic dilation). When the lesion affects both renal arteries, or a single functioning kidney, and is accompanied by renal failure (plasma creatinine concentration above 1.5 mg/dl), it is called ischemic nephropathy or ischemic renal disease [1, 2].

The rate of renovascular hypertension is less than 1% when a mild-moderate hypertension population is assessed, but this increases according to the severity of lab hypertension and with population age [3].

Two well-differentiated of renal artery lesions have been described. Fibromuscular dysplasia is a non-inflammatory lesion that affects young women between 15 and 20 years of age, and its incidence is less than 10% of all RVH cases. Progression of lesions from the angiographic point of view is defined by the appearance of new focal lesions, or a worsening of the existing stenosis grade, and is produced when the intima layer of the artery is affected [4–5].

The most prevalent mechanism underlying lesion of the renal arteries (90%) is atherosclerosis. This increases with age, especially in elderly patients with diabetes, hyperlipidaemia, aortic occlusive disease, and lesions in the coronary artery. Atherosclerosis of the renal artery is a progressive disease that may cause ischemic renal disease, also known as ischemic nephropathy. The prevalence of ischemic nephropathy is poorly quantified, and may vary from 30% in patients with coronary disease to 50% in those with diffuse arteriosclerotic disease [5]. It has been estimated that it may be responsible for 5% to 22% of cases of end-stage renal failure in dialysis programs [6].

**Diagnosis**

The signs and symptoms that suggest RVH include sudden onset of hypertension, especially in young women (fibrodysplastic lesions), existence of hyperkalaemia, abdominal vascular murmurs and asymmetry in renal size (> 1.5 cm) according to ultrasonography criteria. When the lesion is due to atheroma plaque in the ostium of the renal artery it affects men over the age of 60 and is accompanied by lesions in other vascular territories. Table 1 shows the most frequent clinical characteristics according to our experience [7–9] in renal arterial lesions due to atherosclerosis.

**Screening tests**

According to the recommendations of the American College of Cardiology/American Heart Association [10], the following techniques are recommended:

- **duplex Doppler ultrasonography:** in addition to evaluating renal size, it also assesses the morphology of the renal artery and the characteristics of intrarenal flow. In experienced clinical centres, sensitivity and specificity of this test may exceed 96% [11]. Measurement of the intrarenal resistance index (IRI) is an indirect evaluation of the integrity of the circulation of intrarenal vessels and intraparenchymatous lesion. IRI values greater than 0.80 indicate severe parenchymatous disease and reveal little clinical benefit in the control of blood pressure and recovery of kidney function if revascularization is performed;

- **magnetic resonance angiography (MRA):** The test specificity increases with three-dimensional MRA with gadolinium. The sensitivity and specificity of the technique are 97% and 93%, respectively, in the diagnosis of stenosis greater than 50%. In recent months, a serious disease called Nephrogenic Systemic Fibrosis [12], secondary to the administration of gadolinium, has been described in some patients with severe kidney failure;

- **angiography:** If IRI values are greater than 1.0, a renal arteriogram is performed, which will demonstrate an RVH.

**Table 1.** Clinical findings consistent with atherosclerotic renal artery stenosis

<table>
<thead>
<tr>
<th>Abrupt onset at age &gt; 60 years old</th>
<th>Severe hypertension</th>
</tr>
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<tbody>
<tr>
<td>Smoking</td>
<td>Occlusive vascular disease (cerebrovascular, coronary, peripheral)</td>
</tr>
<tr>
<td>Abdominal bruist, flank bruist or both</td>
<td>Atoxemia</td>
</tr>
<tr>
<td>Unexplained azotemia</td>
<td>Azotemia induced by treatment with ACEI/ARB</td>
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<tr>
<td>Flash pulmonary oedema</td>
<td></td>
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</tbody>
</table>

**Suspicion of renovascular hypertension**

**Duplex Doppler ultrasonography**

- **Positive**
  - M.R.A. or C.T.A.
  - Repeat the same examination after 4 months

- **Negative**
  - Angiography

**Other screening tests**

- **Renal scintigraphy following ACE inhibitor:** The sensitivity and specificity of this test are 78–90% and 88–98%, respectively. This decreases when the lesion is bilateral and in kidney failure. In patients with ischemic nephropathy, only renal scintigraphy is used to demonstrate kidney viability.

- **Renal vein renin measurements:** This is used on rare occasions in patients with lesions in both renal arteries.

- **Renal revascularization:** Angioplasty and surgery is indicated in severe and refractory hypertension, and fundamentally, when there is high clinical suspicion of RVH due to fibrodysplasia, renal arteriography can be used directly to confirm the lesion and perform a possible angioplasty. When suspicion is moderate, Doppler duplex should be used, followed by MRA or CTA, depending on the results and experience of each centre.

**Treatment**

The fundamental purpose of the treatment of renovascular hypertension is to control blood pressure and preserve or improve kidney function. Given the different aetiologies and courses of the vascular lesions, both diseases, fibromuscular dysplasia and atherosclerosis, should be analyzed separately.

**Fibromuscular dysplasia**

Blood pressure can be controlled with angiotensin converting enzyme inhibitors (ACEI) or angiotensin II receptor blockers (ARB), together with thiazide diuretics. If blood pressure control is not optimal, a calcium antagonist or beta-blocker may be added [10]. The use of ACEI/ARB in patients with severe and bilateral lesions may cause haemodynamic intraglomerular alterations that deteriorate the glomerular filtration rate. This makes it necessary to monitor plasma creatinine and serum potassium.

Renal revascularization (angioplasty and surgery) is indicated in severe and refractory hypertension, and fundamentally, when there is progression of the lesions with a loss of renal function and mass. Intraluminal angioplasty is the technique of choice: the morphological results according to angiographic criteria show a beneficial grade of dilation between 83% and 100% [14–16]. The percentage of restenosis is 12% to 25%, with an evolution time of two years [14–15]. Hypertension is
controlled in 22% to 59% of these patients, improves in 22% to 74%, and is not modified in 2% to 30% of them [14–17]. Revascularization by surgery is limited to cases with aneurysms in the renal artery or angioplasty of the iliac arteries [28]. The specific complications of the technique include bruises in the puncture zones (20%), cholesterol arterioles (10%), contrast-induced nephropathy and dissection of the renal and iliac arteries [28].

Surgery: This is considered to be a technique of choice (1) in those patients with pathology in the aorto-iliac arteries who will require a combined revascularization, (2) in very severe ostial lesions, and (3) in complete renal artery thrombosis. The results published describe improvement or stabilization of the renal function in 79% to 90%, and progressive deterioration in 10% to 20%, of these cases [24, 19]. The mortality was 4.6% and was associated with older age and symptoms of heart failure [29]. Some authors describe good results with surgical revascularization in cases of acute thrombosis of the renal artery (non-functioning kidneys) as long as some minimum criteria are fulfilled for the surgery and it is possible to place a bypass [26, 27]. Figure 2 shows the algorithm for the treatment of patients with renovascular hypertension.

In conclusion, ischemic renal disease is a complex disease with extrarenal vascular lesions that increase cardiovascular morbidity and mortality. The treatment of renal arteries is difficult to achieve and it is recommended to begin with noninvasive techniques. Initially, medical treatment is indicated and if this does not control the hypertension and the renal function, angioplasty without stent should be used, especially in fibromuscular dysplasia. In ischemic renal disease with atherosclerotic causes, revascularization is indicated if there is a progression of the lesions with loss of renal mass and function. The decisions should be based on individualized analysis of each patient, according to the complexity of their lesions and the experience of each centre.

References