To the Editor:

The formation of pseudoaneurysms in prosthetic vascular accesses is a common event that is associated with fatigue of the prosthetic material secondary to repeated punctures and stenosis-occlusive damage in the venous drainage.1

Treatment is indicated when the pseudoaneurysm undergoes rapid growth, exceeds 2 times the diameter of the prosthesis, produces pain or a threat to cutaneous viability, or in cases of rupture.1

Traditionally, the treatment of this complication has been surgical; however, in recent years, several working groups have incorporated endovascular treatment using the placement of covered stents to exclude this type of lesion.1,2 Thrombin is not widely used as an embolising agent during the treatment of these lesions, with only a few reports of cases in which this type of treatment has been associated with a stent graft.4,5

CASE REPORT

Here we present the case of a 46-year-old patient with chronic renal failure, on trimestral haemodialysis for 23 years secondary to obstructive uropathy, with a left femoral loop (21 months of use) with occluded iliac venous drainage that produced oedema in the leg and three pseudoaneurysms in the therapeutic range. One of these was actively bleeding. The three pseudoaneurysms and occlusion of the left primitive iliac vein were treated using endovascular methods under local anaesthesia. The actively bleeding pseudoaneurysm was excluded with a covered stent, and the other two were percutaneously embolised using balloon-assisted thrombin injections in order to avoid thrombosis in the prosthesis. The iliac venous axis was recanalised and treated with angioplasty balloons; in the final angiographic controls, we observed no pseudoaneurysms, with patency of the venous drainage.

DISCUSSION

Deterioration of the prosthetic material secondary to repeated punctures and stenosis-occlusive lesions in drainage veins represents one of the most common causes of pseudoaneurysms.1 In our case, the prosthetic material was worn down, in addition to a stenosis/occlusive lesion of the venous drainage and bleeding of one of the pseudoaneurysms, with oedema in the leg.

Traditionally, pseudoaneurysms were treated with open surgery; this procedure consisted of replacing the damaged segment of the graft, and if this were impossible, the vascular access was abandoned and another was created. Our patient had exhausted all
were treated using an endovascular approach by placing a stent graft, with a success rate of as high as 100% according to some studies.\(^2,3\)

The use of thrombin as an embolising agent has been widely studied in the treatment of femoral pseudoaneurysms, and this technique currently constitutes the standard practice for treating uncomplicated pseudoaneurysms. In the treatment of pseudoaneurysmal lesions of vascular accesses, we only have case reports of its isolated use\(^4\) or in association with a stent graft.\(^5\) Even so, we have not found any reference in the medical literature to the treatment of 3 pseudoaneurysms in the same patient using different techniques, in addition to revascularisation of the venous drainage system of a femoral loop. Although the treatment of a vascular access with these characteristics may be the subject of debate, we believe that this was the best option in our case, since this was a patient with no other options for a vascular access.

Currently, endovascular treatment offers various alternatives for the treatment of this type of lesion, and so must be considered prior to programming surgery or abandoning the vascular access.

**Conflict of interest**

The authors declare that there is no conflict of interest associated with this manuscript.

5. Keeling AN, Naughton PA, Mc Grath FP, Conlon PJ, Lee MJ. Successful

---

**Figure 1. Fistulographies.**

A and B: left femoral loop fistulography showing 3 pseudoaneurysms in the therapeutic range (arrows) and small pseudoaneurysms in the puncture site of the prosthesis (arrow heads), associated with venous hypertension in the limb. C: collateral circulation through the hypogastric venous branches due to stenosis of the external iliac vein and occlusion of the primitive iliac vein.

**Figure 2. Stent, fluoroscopic images, and fistulography.**

A: stent graft prior to release: lines delineate the proximal and distal ends of the stent. B: fluoroscopic images showing the balloon-assisted embolisation with thrombin technique in one of the non-bleeding pseudoaneurysms. Observe in the upper left quadrant the injection of contrast dye within each pseudoaneurysm prior to embolisation. C: control fistulography following treatment, revealing exclusion of the treated pseudoaneurysms with other small pseudoaneurysms that were below the therapeutic range, and an absence of reflux towards veins of the deep and superficial venous systems, as observed in Figure 1A. D: Patency of the left iliac venous axis following recanalisation and angioplasty, with a notable decrease in collateral circulation.

options of vascular access, for which we decided to preserve the existing one.

Since the first publication by Sapoval in 1996, several cases have been reported in which aneurysms and pseudoaneurysms of vascular accesses were treated using an endovascular approach by placing a stent graft, with a success rate of as high as 100% according to some studies.\(^2,3\)
Senile adult with acute post-infectious glomerulonephritis

Nefrologia 2012;32(5):689-90

To the Editor:
Acute post-infectious glomerulonephritis (APGN) is primarily a disease found in infants that occurs following infection of the respiratory tract or impetigo.\(^1,2\)

This disease is uncommon in older patients, but aging has become an important risk factor. Four decades ago, only 4%-6% of adults with APGN were older than 65 years of age, compared to 34% in recent reports.\(^3\)

Comorbidities such as decreased renal function, diabetes mellitus, arterial hypertension, hyperlipidaemia, cardiovascular disease, and neoplasia contribute to the modification of the clinical presentation and natural progression of this condition. APGN is present in 3%-6% of biopsies, and is often an unexpected finding. Renal function is recovered within 3-4 weeks in 95% of cases, although this rate is much less predictable in the elderly.\(^1,3,4\)

The treatment of APGN is based on antibiotics, diuretics, hypotensive drugs, and occasionally haemodialysis.\(^2\)

We present four cases of patients older than 55 years with acute renal failure and nephritic syndrome, three of which were preceded by a respiratory infection and substantially elevated creatinine levels, and the fourth of which had nephritic/nephrotic syndrome with no prior infection and conserved renal function.

Case A: A 59-year-old male with chronic kidney disease (CKD) and rheumatoid arthritis, hospitalised due to nephritic syndrome in the context of a cutaneous infection by methicillin-re-

### Table 1. Clinical and laboratory values

<table>
<thead>
<tr>
<th>Age</th>
<th>59 years</th>
<th>64 years</th>
<th>80 years</th>
<th>83 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial hypertension</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Oedema</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Haematuria</td>
<td>250µl</td>
<td>250µl</td>
<td>Macroscopic</td>
<td>250µl</td>
</tr>
<tr>
<td>Leukocyturia</td>
<td>Negative</td>
<td>Negative</td>
<td>100µl</td>
<td>100µl</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>150mg/dl</td>
<td>2.6g/24 h</td>
<td>500mg/dl</td>
<td>13g/24 h</td>
</tr>
<tr>
<td>Creatinine</td>
<td>4.3mg/dl</td>
<td>1mg/dl</td>
<td>5.8mg/dl</td>
<td>6.1mg/dl</td>
</tr>
<tr>
<td>C3</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>ASLO</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>FR/ANCA/ANA/anti-MBG</td>
<td>Negative</td>
<td>p-ANCA (+) 1/320</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Infection</td>
<td>Respiratory and cutaneous</td>
<td>None</td>
<td>Respiratory</td>
<td>Orchitis/epididymitis and respiratory</td>
</tr>
<tr>
<td>Treatment</td>
<td>Prednisone 1mg/kg</td>
<td>None</td>
<td>ATB + HD</td>
<td>ATB + prednisone 1mg/kg + HD</td>
</tr>
<tr>
<td>Evolution of creatinine</td>
<td>2.4mg/dl</td>
<td>1mg/dl</td>
<td>7.8mg/dl HD</td>
<td>HD</td>
</tr>
<tr>
<td>Evolution of proteinuria</td>
<td>300mg/24 h</td>
<td>1.2g/24 h</td>
<td>HD</td>
<td>HD</td>
</tr>
</tbody>
</table>

ANA: anti-nuclear antibodies; ANCA: anti-neutrophil cytoplasmic antibodies; HD: haemodialysis