<table>
<thead>
<tr>
<th>Therapeutic model of patient incidents (%)</th>
<th>HHD</th>
<th>OHHD</th>
<th>DHHD</th>
<th>Tx</th>
<th>PD</th>
<th>Movements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ourense</td>
<td>3.8</td>
<td>62.9</td>
<td>0</td>
<td>–</td>
<td>26.9</td>
<td>0</td>
</tr>
<tr>
<td>Therapeutic model prevalent patients (pmp)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Ourense</td>
<td>526</td>
<td>–</td>
<td>5.7</td>
<td>457.8</td>
<td>125.6</td>
<td>–</td>
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<tr>
<td>Galiciaa</td>
<td>436</td>
<td>–</td>
<td>0</td>
<td>489</td>
<td>99.3</td>
<td>–</td>
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<td>Therapeutic model: evolution 1976–2006 (%)</td>
<td>23.1</td>
<td>9.47</td>
<td>0.26</td>
<td>2.763</td>
<td>30.14</td>
<td>33.68</td>
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<tr>
<td>1976–1987</td>
<td>39.18</td>
<td>25.95</td>
<td>0.26</td>
<td>30.14</td>
<td>33.68</td>
<td>3.1</td>
</tr>
</tbody>
</table>

HHD: hospital haemodialysis; OHHD: out-hospital haemodialysis; DHHD: daily home haemodialysis; Tx: renal transplant; PD: peritoneal dialysis.

aGalicia is the region to which Ourense belongs.

management, the TM needs to be diversified and self-service options chosen, whenever so allowed by the clinical situation.

Conflict of interest statement. None declared.

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Angioplasty of the renal artery as a trigger for acute anti-phospholipid syndrome

Sir,

Anti-phospholipid syndrome (APS, Hughes’ syndrome) is a multifaceted systemic autoimmune disease, whose spectrum ranges from tempestuous and deadly to silent and smouldering [1–3]. APS is characterized by recurrent deep venous thrombosis, although the involvement of arterial vessels has been increasingly described [1–3]. The contribution of APS to the development of ‘idiopathic’ renal artery stenosis remains unclear, also because the assessment of antibody status is not a part of the routine diagnostic work-up.

We report here a case in which angioplasty of renal arteries triggered acute APS, a life-threatening complication, never described thus far.

The patient was a 77-year-old woman, affected by type 2 diabetes, moderate hypertension, diffuse vascular disease and recurrent atrial fibrillation. Three years before this report, she was diagnosed with ‘moderate’ right renal artery stenosis. Renal function was normal. The patient was managed conservatively for 2 years, until the right renal artery stenosis progressed to >70%, and left renal artery stenosis <70% was detected. After a severe hypertensive crisis and a rise in serum creatinine, to 1.4 mg/dl, percutaneous transluminal angioplasty (PTA) with stenting of both renal arteries was successfully performed.

A sudden drop of thrombocytes was observed a few hours after the procedure (116 000 pre-angioplasty, 33 000 after 8 h); serum creatinine rose from 1.4 to 3.1 mg/dl. Echo-doppler confirmed the patency of renal arteries, with high intraparenchymal resistances. On the hypothesis of an autoimmune phenomenon, she was empirically started on low-dose corticosteroids and fresh plasma infusion. In the following days, serum creatinine reached 4.7 mg/dl, with nephrotic proteinuria (7 g/day). The immunological tests revealed positivity in the Lupus anticoagulant test, anti-cardiolipin and Anti-GPI: Anti Beta2 glycoprotein 1 antibodies. In keeping with a diagnosis of acute burst of APS, triggered by the procedure, on a pre-existent APS with a smouldering course, the patient was treated with three bolus doses of methylprednisolone 500 mg, and with six sessions of plasmapheresis, with a rapid rise of the platelet count and normalization of coagulation.

At hospital discharge, 25 days later, on chronic Warfarin and oral prednisone, serum creatinine was 2 mg/dl, proteinuria 2.7 g/day, creatinine clearance 35 ml/min, haemoglobin 11 g/dl and platelet count 90–110 000 platelets/mm³.

This case is characterized by the presence of a mechanical trigger, arteriography and renal stenting, which presumably released into the circulation factors that induced a prompt immunological response in the presence of pre-formed antibodies, thus precipitating an acute APS. According to a systematic search on Medline and Embase, this is the first report of a life-threatening acute APS triggered by renal artery stenting.
In the context of systemic vasculitis, the risk of accelerated atherosclerosis is recognized, and systematic echodoppler surveillance is increasingly advised [4,5]. On the contrary, although an increased prevalence of autoantibodies in severe vascular disease has been reported, a systematic search for autoimmune is not routinely performed [6,7].

The present case, describing a new trigger for acute severe autoimmune bursts, suggests that a minimal immunological diagnostic work-up should be considered, at least in high-risk patients in whom an immune-mediated reaction could have severe, even deadly, consequences.

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Metformin-associated lactic acidosis in type 2 diabetes mellitus: incidence and presentation in common clinical practice

Sir,

Metformin is an oral antihyperglycaemic agent used in the treatment of type 2 diabetes mellitus. In 1998, the results of the UK Prospective Diabetes Study [1] indicated that metformin treatment is associated with a reduction in total mortality compared to other antihyperglycaemic treatments.

These and other results led to its progressive widespread use. In the recent ADA-EASD consensus on management of hyperglycaemia in type 2 diabetes, metformin plus lifestyle intervention is the initial recommended therapeutic step [2].

Metformin is considered to be contraindicated in many chronic hypoxaemic conditions that may be associated with lactic acidosis (LA), such as cardiovascular, renal, hepatic, pulmonary disease and advancing age. Nevertheless, this has been a controversial matter. In a recent Cochrane review [3], pooled data from 206 comparative trials and cohort studies revealed no cases of fatal and nonfatal LA in 47 846 patient-years of metformin use. Thus, the causal relationship between metformin use and LA has been questioned. Some authors have even stated that ‘metformin’s contraindications should be contraindicated in people with type 2 diabetes’ [4].

In the past several years an increasing number of LA associated with metformin use has been appreciated. The aim of this brief report is to perform a clinical description of metformin-associated LA in our reference area, triggering medical conditions, possible contraindications and evolution. An epidemiologic study is made in order to determine the incidence of LA and its relation with metformin administration.

Material and methods

A retrospective analysis of all cases defined by hospital discharge with a diagnosis of metabolic acidosis (International Classification of Diseases, ninth revision code), attended in the emergency department of our hospital during the period 2001–2005, was performed. This is a general hospital with a reference population area of 390 000 inhabitants. LA was accepted on the basis of an arterial blood sample with a pH < 7.35 and circulating lactate values > 5 mmol/l. Demographic information, clinical presentation and evolution, biochemical data and risk factors were obtained. During the same period, the metformin consumption in the general population was analysed (dose/1000 inhabitants/day: DHD) in our reference area, this information was obtained from the Pharmacology Department of Catalan Health Institute (Serveis Centrals de l’ICS).

Results

Out of 226 attended cases in which the diagnosis of metabolic acidosis was made, 21 were diagnosed of LA; 13 of them (5.7%) were related to metformin administration. Severe sepsis or septic shock was ruled out in all of them.

There were 10 women and 3 men, between 65 and 77 years. Main biochemical results at admission are shown in Table 1. The trigger factor was an acute gastrointestinal process with relevant dehydration in 85% of cases; 15% (two cases) had overt congestive heart failure. Interestingly, despite a clear worsening of their clinical condition, none of the patients had discontinued metformin treatment in the days preceding admission. All had renal insufficiency at