Anti-Klebsiella, Chlamydia, Neisseria, Campylobacter, Yersinia, Toxoplasma, and Epstein Barr virus antibodies were negative. The phenotype of the HLA antigen showed B27 positivity. Repeat X-rays, computer tomograms and skeletal scintigraphy showed only irregularity of the left sacroiliac joint and increased uptake of contrast medium in the same area.

The patient was treated with non-steroidal anti-inflammatory agents and gentamicin by parenteral route, therapy that resulted in immediate and sustained regression of fever, and the elbow arthritis. However, there was clinical evidence of restrictive cardiomyopathy over the course of 2 weeks.

Three months after hospital admission, the patient was enjoying excellent health.

We have described an unusual case, not previously reported, of reactive arthritis as a result of Staphylococcus epidermidis. In our patient, three positive blood cultures established the presence of a true bacteremia caused by Staphylococcus epidermidis; moreover, the excellent clinical response to gentamicin, with subsequent negative blood cultures, confirmed the diagnosis.

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Paradoxical acute brain thromboembolism during prostacyclin (PGI₂) acute challenge for primary pulmonary hypertension

Acute testing with vasodilators to detect a reversible component in primary pulmonary hypertension (PPH) has been associated with several serious complications[12], but not with thromboembolism.

We report a patient with severe PPH and patent foramen ovale (PFO) who developed paradoxical cerebral thromboembolism during withdrawal of a Swan-Ganz catheter inserted for PGI₂ acute testing. In October 1993, a 34-year-old woman was referred to our institution to explore the pharmacological reversibility of PPH under invasive haemodynamic monitoring, using PGI₂ as a screening agent. Six months earlier, a PFO had been documented by contrast-enhanced echocardiography. Her only treatment had been acenocoumarol which was discontinued 7 days before hospitalization. Internal jugular puncture and progression of the catheter in the cardiac chambers were uneventful. Prostacyclin, from 5 to 10 ng kg⁻¹ min⁻¹, was infused with perfect clinical tolerance. Haemodynamic parameters and PaO₂ in supine position, at rest and breathing room air, at baseline and under PGI₂ are reported in Table 1.

As a result of the increase in mean pulmonary artery pressure (PAP) and vascular resistance index (PVR), with an associated decrease in systemic vascular resistance index (SVR), PGI₂ infusion was stopped and the Swan-Ganz catheter immediately removed. A few minutes later, the patient developed asphasia and right-sided hemiparesis. A transthoracic cardiac echocardiogram was obtained immediately and showed absence of intracardiac thrombi and a patent foramen ovale. Doppler examination of the deep veins of the legs was normal. The patient was treated with intravenous heparin and the neurological deficit completely resolved within 12 h. Brain CT scan performed 36 h after the onset of symptoms showed spontaneous hypodensity in the left capsule-lenticular area consistent with the diagnosis of ischaemic vascular accident. Oral anticoagulant therapy with acenocoumarol was restarted and the patient was discharged on day 5 with a normal neurological examination.

In contrast, with long-acting, non-titratable agents[3], PGI₂, a potent systemic vasodilator with a very short half-life, has been shown to be safe during acute testing for PPH[4]. In the case that we report here, the close time relationship between removal of the Swan-Ganz catheter and the onset of neurological symptoms makes the hypothesis of migration of a catheter-related thrombus into the systemic circulation through the PFO likely. The alternative hypothesis of venous air embolism seems unlikely since the catheter was inserted and removed through an indwelling Cordis sheath introducer. In our patient, changes in pulmonary and systemic haemodynamics induced by PGI₂ may have promoted the systemic embolization of a right-sided thrombus.

The administration of PGI₂ resulted in a substantial rise in PAP and right atrial pressure (+22% and +80% from baseline respectively).

Table 1 Acute haemodynamic response to PGI₂

<table>
<thead>
<tr>
<th>HR (beats min⁻¹)</th>
<th>Baseline (10 ng kg⁻¹ min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAP (mmHg)</td>
<td>83/60/68</td>
</tr>
<tr>
<td>RAP (mmHg)</td>
<td>1</td>
</tr>
<tr>
<td>PWP (mmHg)</td>
<td>7</td>
</tr>
<tr>
<td>CI (l min⁻¹ m⁻²)</td>
<td>3.1</td>
</tr>
<tr>
<td>PVRi (U m⁻²)</td>
<td>19.7</td>
</tr>
<tr>
<td>SAP (mmHg)</td>
<td>110/60/74</td>
</tr>
<tr>
<td>SVRi (U m⁻²)</td>
<td>23.8</td>
</tr>
<tr>
<td>PaO₂ (mmHg)</td>
<td>83</td>
</tr>
</tbody>
</table>

HR=heart rate; PAP= pulmonary artery pressure (systolic/diastolic/mean); RAP=right atrial pressure; PWP=pulmonary wedge pressure; CI=cardiac index; PVRi=pulmonary vascular resistance index; SAP (s/d/m)=systemic artery pressure (systolic/diastolic/mean); SVRi=systemic vascular resistance index.

References

Eur Heart J, Vol. 17, January 1996
with an increase in the mean interatrial pressure gradient (Table 1). This apparently paradoxical response can be explained by the existence of a fixed obstruction in the pulmonary vascular bed and a concomitant increase in cardiac output due to a prevailing systemic vasodilator response (assessed by the increase observed in our patient PVRi/SVRi ratio).

This report indicates that right-sided heart catheterization for acute vasodilator testing in patients with PPH carries unpredictable risk of a systemic embolic event when PFO is present. In patients with known PFO, this procedure should therefore be performed under anticoagulant therapy, such as standard heparin, unless it is contra-indicated. Furthermore, systemic thromboembolism may be promoted by a deleterious haemodynamic response to a systemic vasodilator.

Such a complication, occurring with one of the safest non-selective vasodilators available, favours the use of inhaled nitric oxide, a potent and selective pulmonary vasodilator\(^\text{151}\), when screening for acute vasodilator responsiveness in PPH.

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