grew *Aspergillus flavus*. Acute fungal prostatitis was diagnosed, and treatment with itraconazole (200 mg i.v. od) was initiated. Subsequently he developed a perinephric collection, which on drainage grew *Escherichia coli* and *Aspergillus*. The antimicrobial regime was changed to vancomycin (1 g i.v. bd) and imipenem (1 g i.v. qds). The itraconazole was empirically changed to voroconazole (400 mg i.v. od) and caspofungin (50 mg i.v. od). The patient’s pyrexia and symptoms took a further 8 weeks to resolve. He received voroconazole for a total of 75 days (28 days i.v. and 47 days orally) and caspofungin for 23 days.

Six months following discharge, the patient presented again with similar symptoms, reduced urinary flow and raised inflammatory markers. A further TRUS of his prostate demonstrated a cystic area involving the left seminal vesicle. He restarted anti-aspergillus treatment of voraconazole and caspofungin for 23 days.

Conflict of interest statement. None declared.

1Department of Urology and Transplantation
2Department of Radiology
Hammersmith Hospital
London
UK
Email: banksfrederick@yahoo.co.uk

Discussion

Prostatitis is a rare presentation of invasive aspergillosis in the transplant setting, although previous cases of aspergillus prostatitis in immunocompromised patients have been reported [1–4]. This patient’s only predisposing factor was the post-transplantation immunocompromising therapy, and no steroid boluses for rejection were required as the graft function remained stable throughout, at a creatinine of ~150 μmol/l. The infection disseminated to the transplanted kidney in spite of supposedly effective treatment using older antifungal agents. Vorconazole and caspofungin were effective treatment agents, and this is the first report of caspofungin being used for the treatment of fungal prostatitis. This report highlights the requirement for an aggressive, prolonged multi-agent approach to the treatment of fungal prostatitis in immunocompromised patients.

Possible role of erythropoietin in the pathogenesis of chronic cor pulmonale

Sir,

As has been well demonstrated, the role played by erythropoietin, the growth factor of erythroid precursors, in general homeostasis is far wider than the simple regulation of the erythropoietic mass. This growth factor may also play a role in the development of several diseases, including chronic cor pulmonale (CCP).

CCP is a pathological condition characterized by right ventricular dilation and pulmonary hypertension. The pathophysiology of pulmonary hypertension is extremely complex. While hypoxic vasoconstriction appears to govern the entire process, the real cause of this condition is unknown; it is probably linked to the release of mediators acting on the vascular tone and leading to neurohormonal vascular hyperactivity that would, in the long term, cause chronic vasoconstriction [1]. Moreover, pulmonary hypertension and chronic hypoxia trigger muscular hypertrophy of the small artery walls with a further increase in vascular resistance [2].

Few studies have investigated the role of erythropoietin, the main growth factor of erythroid precursors, in the onset and progression of CCP. To assess the potential effect of erythropoietin on pulmonary artery pressure, we studied the effect of i.v. erythropoietin administration by means of a Swan–Ganz catheter and peripheral arterial catheter in 10 male subjects with CCP (mean age 68 ± 3 years, history of chronic bronchitis and evidence of right ventricular dilation, pulmonary artery pressure >35 mmHg), and 10 healthy male non-smokers (69 ± 2 years). Written informed consent was obtained from all subjects.

Subjects received an intravenous bolus of rhHuEpo (erythropoietin β 70 U/kg). At 0, 10 and 30 min we evaluated the mean arterial blood pressure, mean pulmonary arterial blood pressure, pulmonary vascular resistance index and the systemic vascular resistance index. The results are shown in Table 1. Statistical analysis was done using the ANOVA one-way test. The value *P* < 0.05 was considered significant. Data were expressed as mean values ± SD.

Our data appear to confirm that erythropoietin has an effect on vascular pulmonary resistance, causing a significant increase in pulmonary vascular resistance index (PVRI) both in healthy people and in CCP patients. We made a haemodynamic evaluation in two patients with hypervolaemic shock using a catheter placed in the pulmonary artery. Erythropoietin administration determined an increase in pulmonary vascular resistance and in mean pulmonary artery pressure [3].

In transgenic mice that constitutively over-express the human erythropoietin gene in an oxygen independent manner, it has been demonstrated that pulmonary artery pressure is increased *in vivo*. Likewise, in heterozygous Hypoxia-Inducible Factor-1α-deficient (HIF1α +/−) mice, the key factor in erythropoietin regulation, following hypoxia a significantly delayed development of polycythemia, pulmonary hypertension, right ventricular hypertrophy, and pulmonary vascular remodelling are seen [4]. The action of erythropoietin on vascular pulmonary resistance could be determined by the capacity of erythropoietin to influence vascular tone.

In our previous experience on studying the blood flow of the pre-tibial muscle, we found an erythropoietin induced...
Table 1. Mean arterial blood pressure, mean pulmonary arterial blood pressure, pulmonary vascular resistance index, systemic vascular resistance index, serum EPO and HT in CCP subjects and in healthy controls after administration of 70 UI/kg rHuEPO

<table>
<thead>
<tr>
<th>Parameter</th>
<th>T0 Control</th>
<th>CCP</th>
<th>10min Control</th>
<th>CCP</th>
<th>30min Control</th>
<th>CCP</th>
</tr>
</thead>
<tbody>
<tr>
<td>MABP (mmHg)</td>
<td>106.5±3.6</td>
<td>105.0±3.0</td>
<td>111.6±4.4a</td>
<td>113±3.9b</td>
<td>113±4.9b</td>
<td>114±4.5b</td>
</tr>
<tr>
<td>MPABP (mmHg)</td>
<td>16.2±1.7</td>
<td>38.3±6.1a</td>
<td>17.5±1.5b</td>
<td>47.6±2.1a,b</td>
<td>17.0±0.9b</td>
<td>47.1±3.0a,b</td>
</tr>
<tr>
<td>PVRI (dyn.Scm⁻²)</td>
<td>166±19</td>
<td>480±155a</td>
<td>195.1±20b</td>
<td>578±141a,b</td>
<td>199.1±19b</td>
<td>544±154a,b</td>
</tr>
<tr>
<td>SVRI (dyn.Scm⁻⁵)</td>
<td>2435±209</td>
<td>2550±225a</td>
<td>2595±202b</td>
<td>2710±254ab</td>
<td>3450±289b</td>
<td>3510±310b</td>
</tr>
<tr>
<td>Serum erythropoetin (U/l)</td>
<td>21.3±10</td>
<td>45.31±16b</td>
<td>3450±289b</td>
<td>3510±310b</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HT (%)</td>
<td>44±4</td>
<td>47±7a</td>
<td>43.1±9</td>
<td>47±8a</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*aP < 0.05 vs control.

bP < 0.05 vs T = 0.

MABP, mean arterial blood pressure; MPABP, mean pulmonary arterial blood pressure; SVRI, systemic vascular resistance index. All parameters show higher values in CCP group, even at basal condition. Data are expressed as mean±SD.

The challenge of germ cell tumour therapy in dialysis and transplantation

Sir,

We report an unusual pattern of recurrent seminoma and highlight the choices in curative treatment in the context of peritoneal dialysis and transplantation. A 53-year-old man with end-stage renal failure secondary to autosomal dominant polycystic kidney disease on continuous ambulatory peritoneal dialysis was noted to have an enlarged right testicle. Investigation revealed a stage I classical seminoma entirely confined to the testis and he underwent orchidectomy. He received adjuvant radiotherapy to a para-aortic strip (20 Gy in 10 fractions). Human chorionic gonadotrophin (HCG) and α-fetoprotein were negative and there was no evidence of lymphadenopathy or of metastatic spread on CT. Two years later, with no evidence of tumour recurrence after repeated staging investigations, he received a cadaveric renal transplant. Immunosuppression was with basiliximab, followed by

Advance Access publication 23 August 2005

Conflict of interest statement. None declared.