
**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></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±4.4b</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.±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±3.0b</td>
</tr>
<tr>
<td>PVR (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±154b</td>
</tr>
<tr>
<td>SVRI (dyn.Scm⁻²)</td>
<td>2435±209</td>
<td>2550±225a</td>
<td>2595±202b</td>
<td>2710±254a,b</td>
<td>2710±254a,b</td>
<td>2595±202b</td>
</tr>
<tr>
<td>Serum erythropoetin (U/l)</td>
<td>21.3±10</td>
<td>45.31±16a</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>

a*/P < 0.05 vs control.  
b*/P < 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 action of erythropoietin on the physiopathology of CCP may, however, not be limited to its vasomotor effect on the pulmonary circulation; it may also impact on pulmonary vascular remodelling. It is well known that erythropoietin is only one of the many factors activated during the reduction in pO₂, the role of the hormone in the development of pulmonary hypertension and pulmonary vascular remodelling in patients with chronic obstructive broncopathy may therefore contribute to the thickening of the arterial wall of the pulmonary vessels, typical of chronic pulmonary heart disease.

In conclusion, although erythropoietin is only one of the many factors activated during the reduction in pO₂, the role of the hormone in the development of pulmonary hypertension and pulmonary vascular remodelling in patients with cor pulmonale may be of clinical relevance.

Conflict of interest statement. None declared.


doi:10.1093/ndt/gfi098

Advance Access publication 23 August 2005

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 ß-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...
A loss in ultrafiltration or haemoperitoneum [4]. This did increase in permeability of the peritoneum to water causing the peritoneum, but what is available suggests it can cause a differentiating between fibrosis and residual tumour mass [3].

...stases > 5 mm with PET. However, it is not reliable in/C24 100% specificity and positive predictive values for metastatic area. A higher sensitivity (70 vs 40%) has been reported for PET vs CT scanning, with ~100% specificity and positive predictive values for metastases >5mm with PET. However, it is not reliable in differentiating between fibrosis and residual tumour mass [3].

There is a paucity of data on the effects of radiotherapy on the peritoneum, but what is available suggests it can cause an increase in permeability of the peritoneum to water causing a loss in ultrafiltration or haemoperitoneum [4]. This did not occur in our patient and his PD prescription remained unchanged throughout his treatment.

Cisplatin is the most effective agent against seminomas and metastatic disease, and there are reports of successful treatment of renal transplant recipients with this drug [5]. However, due to the well documented nephrotoxicity of cisplatin, we chose to use carboplatin, and this caused no problems with graft function. The sequelae of chemotherapy on a transplanted kidney is not necessarily seen immediately and cases of renal deterioration up to 6 years post chemotherapy have been reported, but after time it becomes difficult to identify whether the chemotherapy agent is the culprit. We also considered renoprotection using sodium thiosulphate and N-acetylcysteine [6], but were worried about reducing the anti-tumour properties of the drugs.

In summary this patient with a germ cell tumour had no PD problems after standard radiotherapy, but had an unusual presentation of recurrence. PET scanning is strongly suggested if recurrence is suspected. Transplantation did not interfere with modified standard chemotherapy, with a good outcome from tumour and transplant.

Conflict of interest statement. None declared.

Brighton and Sussex University Hospitals NHS Trust Royal Sussex County Hospital Brighton BN2 5BE, UK
Email: cally.dean@bsuh.nhs.uk


doi:10.1093/ndt/gfi108

Advance Access publication 23 August 2005

Severe theophylline intoxication: a delay in charcoal haemoperfusion solved by oral activated charcoal

Fig. 1. Fluorodeoxyglucose PET scan.

tacrolimus and prednisolone. Four months post-transplant, a sudden rise in βHCG to 199 iU/l was noted. A CT scan of the chest, abdomen and pelvis was reported as normal, and he had a normal ultrasound scan and biopsy of his remaining testicle. However, a positron emission tomography (PET) scan revealed an intense focus of uptake in the left supraclavicular fossa (Figure 1). A high resolution CT and fine needle aspiration of a tiny lesion in this area showed recurrent seminoma. He was given chemotherapy with etoposide, bleomycin and carboplatin in three cycles, followed by local radiotherapy. Two years later he is well with serum creatinine of ~100 μmol/l, with no evidence of tumour recurrence.

Germ cell tumours are the most common cancers in young men and are highly amenable to treatment, with cure rates of ~90%. Testicular neoplasms are 20–50% more prevalent amongst immunosuppressed patients when compared with controls. Low levels of βHCG are seen in only 10–25% of seminomas. After orchidectomy and radiotherapy a 1–2% relapse rate is quoted. Testicular neoplasms are more prevalent among organ transplant recipients. There is a lower recurrence rate in renal compared with cardiac allograft recipients [1], suggesting that tumour surveillance is impaired proportionally to the degree of immunosuppression. This patient had a very unusual presentation for his seminomatous recurrence, with the rise in βHCG, which could be explained simply by an increase in tumour load. The focus of recurrence was at a distant site from the primary cancer, so it is possible that there were further seeded metastases that were not picked up by the imaging but that contributed to the tumour marker concentration at the time of recurrence. The small proportion of producing βHCG seminomas may behave clinically as non-seminomatous germ cell tumours (NSGCT). The threshold value many clinicians use to suggest NSGCT is a βHCG level of 200 IU/l [2].

The dramatic rise in βHCG prompted careful diagnostic surveillance scans, which failed to detect recurrence at this stage. It was only with PET scanning that we localized the metastatic area. A higher sensitivity (70 vs 40%) and specificity (100 vs 78%) has been reported for PET vs CT scanning, with ~100% specificity and positive predictive values for metastases >5 mm with PET. However, it is not reliable in differentiating between fibrosis and residual tumour mass [3].

There is a paucity of data on the effects of radiotherapy on the peritoneum, but what is available suggests it can cause an increase in permeability of the peritoneum to water causing a loss in ultrafiltration or haemoperitoneum [4]. This did

ADVANCE