time of presentation revealed normocytic, normochro-
mic anemia, moderately elevated ESR and dyslipi-
demia. The urine gave a +/- test for protein and he-
moglobin. The echocardiogram revealed symmetrical
left ventricular hypertrophy, and the ejection fraction
was 60%. Specific laboratory investigation included
measurement of 24-hour urinary fractionated meta-
nephrines (HPLC), total catecholamines and vanillyl-
mandelic acid (VMA, spectrophotometry) on three
consecutive days. In addition, twenty-four hour uri-
inary excretion of dopamine and homovanillic acid
(HVA, spectrophotometry) were measured. All the
results were within normal range (Table 1). Plasma
catecholamine or metanephrine levels were not de-
termined because pertinent methodology was not
available in our institution. Despite the normal levels
of urinary catecholamines and their metabolites, sus-
picion of pheochromocytoma remained very strong on
clinical grounds. An imaging study with 131I-metaiodo-
benzylguanidine (131I-MIBG) scintigraphy was per-
formed and revealed selective concentration of the
radiotracer, corresponding to the CT mass, on the 24-
to-72-hour images (Figure 2). After adequate adren-
ergic blockade and hydration, successful surgical ex-
cision of the tumor was performed through a transab-
dominal incision. Immediate dissection of the mass
revealed extensive cystic degeneration. Macroscopi-
cally, pathological examination revealed a specimen
19.2gr in weight containing a deep red neoplasm 2cm
in maximum diameter. Microscopical examination
revealed a neoplasm of the left adrenal medulla, with
morphologic and immunophenotypic characteristics
of pheochromocytoma (Syn +, Chrom +, S-100 + on
supportive cells) with central hemorrhagic infiltration.
Indications of malignancy (mitosis, necrosis, vascular
or capsular infiltration) were not detected. The spec-
imen contained a peripheral rim of adrenal cortex.
The patient was discharged a few days later without
any antihypertensive medication. She experienced no
spells of hypertension and her blood pressure re-
mained normal with no clinically important postural
drop. On the follow-up three months later, she had
mild recurrent hypertension, asymptomatic orthostatic
hypotension and reported panic attacks. Repeated
biochemical and imaging studies (131I-MIBG scan, tho-
racic and abdominal MRI) showed no evidence of re-
mainig or recurrent disease. Psychiatric assessment
resulted in the diagnosis of persistent depression and
the patient was started on appropriate treatment with

Table 1. 24-hour urinary outputs of catecholamines and their metabolites in 3 consecutive collections ($V_1$, $V_2$, $V_3$)

<table>
<thead>
<tr>
<th></th>
<th>$V_1$: 1820ml</th>
<th>$V_2$: 2280ml</th>
<th>$V_3$: 2880ml</th>
<th>Normal values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total catecholamines</td>
<td>28</td>
<td>39</td>
<td>29</td>
<td>14-108μg/24h</td>
</tr>
<tr>
<td>Fractionated metanephrines</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normetanephrine</td>
<td>93</td>
<td>343</td>
<td>313</td>
<td>88-444μg/24h</td>
</tr>
<tr>
<td>Metanephrine</td>
<td>26</td>
<td>91</td>
<td>89</td>
<td>52-341μg/24h</td>
</tr>
<tr>
<td>VMA</td>
<td>2.7</td>
<td>2.9</td>
<td>2.9</td>
<td>1.8-6.7mg/24h</td>
</tr>
<tr>
<td>Dopamine</td>
<td>126</td>
<td>131</td>
<td>122</td>
<td>65-400μg/24h</td>
</tr>
<tr>
<td>HVA</td>
<td>4.1</td>
<td>4.5</td>
<td>4.4</td>
<td>&lt;6.2mg/24h</td>
</tr>
</tbody>
</table>

VMA: Vanillylmandelic acid, HVA: Homovanillic acid

Legends