Liver Cell Adenoma Associated with Membranous Nephropathy

Table 1

<table>
<thead>
<tr>
<th>M.J.</th>
<th>Coma-del-Corral</th>
</tr>
</thead>
<tbody>
<tr>
<td>S.</td>
<td>Razquin</td>
</tr>
<tr>
<td>S.</td>
<td>Pascual</td>
</tr>
<tr>
<td>M.P.</td>
<td>Bengoechea</td>
</tr>
<tr>
<td>E.</td>
<td>Gutierrez</td>
</tr>
<tr>
<td>L.</td>
<td>Yuguero</td>
</tr>
</tbody>
</table>

Hospital General Yagüe, Burgos, Spain

M.J. Coma-del-Corral, Servicio de Anatomía Patológica, Hospital General Yagüe, Avenida del Cid s/n, E-09005 Burgos (Spain)

Dear Sir,

Membranous glomerulonephritis is a renal injury caused by immune complexes, among which different antigens associated with tumors have been implicated; most of them are carcinomas. Another, less numerous group corresponds to malignant hematologic diseases [1–3]. Very rarely, benign neoplasms have been reported to be associated with this condition. There are also cases of liver carcinoma with or without HBAg [4, 5], but up to now, we have not found associations between liver cell adenoma and nephropathy.

The membranous nephropathy is characterized by discontinuous deposits of immunoglobulins and complement exclusively in subepithelial distribution along the outer surface of the glomerular capillary wall.

A 62-year-old man who was admitted to hospital with dyspneic cough, blood-stained sputum and pain in the right hemithorax. He had malaise, anorexia and weight loss for 1 month. On examination, there were diminished breath sounds in the lower and posterior right thorax; the liver edge was palpable 4 cm below the costal margin; the patient also had malleolar swelling. X-ray of the chest showed right pleural effusion. The laboratory findings were as follows: serum protein 5.6 g/dl, albumin 1.69 g/dl and α2 globulin 1.145 g/dl (20.44%). Immunoglobulins: IgG 1,250 mg/dl, IgA 419 mg/dl and IgM 145 mg/dl. CEA, α-fetoprotein, HBsAg and HBsAc were all negative. The results of the urine test were: proteins 12.4 g/24 h. No other abnormal substance was found. Urinary sediment was normal. As a secondary nephropathy was suspected, an echography was carried out which revealed a solid growth in the right hepatic lobe; the tumor was hyperechogenic, irregular in shape with hypodense areas inside and arterial supply coming from the hepatic artery. These findings were confirmed by laparoscopy. In view of the relatively good general condition of the patient, the precise diagnosis made, the antecedents of pulmonary embolism and the possibility to resect the mass, a laparotomy was indicated. A few hours before surgery was due to take place, the patient began to suffer from another dyspneic crisis and hypertension, followed by loss of consciousness, cyanosis and cardiorespiratory arrest, which could not be overcome. At autopsy, a massive pulmonary embolism and renal vein thrombosis
contiguous to the vena cava inferior was found. Apart from the massive embolism, which was
the cause of death, there were peripheral infarctions of long standing in the lungs.

Fig. 1. Liver cell tumor: there is a resemblance to normal liver tissue, except for the absence of
portal tracts. Some cells contain fat vacuoles. HE. × 80.

The hepatic neoplasm, situated below the right hepatic lobe, measured 10×10×8 cm, and could
easily be distinguished from the rest of the normal liver tissue. Microscopically (fig. 1) it was a
neoplasm formed by well-differentiated hepatocytes, without nuclear pleomorphism and mitosis.
There were neither biliary ducts nor portal spaces, but some areas of ischemic necrosis and
fibrosis were apparent. Therefore, the tumour was an adenoma.

The kidneys showed diffuse glomerulonephritis with uniform and homogeneous thickening of
the glomerular capillary walls and minimal cellular proliferation. By immunofluorescence,
granular deposits of IgG and complement were seen in the basal capillary membranes.

Ultrastructurally (fig. 2), moderate electron-dense deposits were seen on the subepithelial side of
the basal capillary membrane. There was also extensive pedicelar fusion and hyperplasia of the
foot processes of the podocytes.

No other tumor, amyloid deposits or other alteration was found.

The incidence of nephrotic syndrome associated with neoplasm varies depending on the author:
Papper [2] considers that an average of 10% of the adults with the

References
Pascal RR, Iannaccone PM, Rollwagen FM, Harding TA, Bennett SJ: Electron microscopy and
immunofluorescence of glomerular immune complex deposits in cancer patients. Cancer Res
1976;36:43–47.
Lai KN, Lo STH, Lai FMM: Immunohistochemical study of the membrane attack complex of
complement and S-protein in idiopathic and secondary membranous nephropathy. Am J Pa-thol
Stevens PE, Rainford DJ: Nephrotic syndrome as the marker for underlying malignancy. J R Soc
Helin H, Pasternack A, Hakala T, Pentinen K, Wager O: Glomerular electron-dense deposits and
Rosen RD, Reisberg MA, Hersh EM, Gutterman JU. Measurement of soluble immune

Cosby R, Yamauchi H, Lee JC, Hopper JJ: Tumor related renal lesions – reversal following
Gluck MC, Gallo G, Lowenstein J, Baldwin DS: Membranous glomerulonephritis. Evolution of
Yamada A, Mitsuhashi K, Miyakawa Y, Kosaka K, Takehara K, Iijima M, Tanaka, K, Shibata S:
Membranous glomerulonephritis associated with eosinophilic lymph folliculosis of the skin
mastocytosis associated with membranous nephropathy and peripheral neuropathy. Am J Kidney
Verresen L, Waer M, Verberckmoes R, Morias P, Michielsen P: Primary sclerosing cholangitis
Llach F: Hypercoagulability, renal vein thrombosis and other thrombotic complications of