Short reports

Association of a renal papillary carcinoma with a low grade tumour of the collecting ducts

L Daniel, H Zattara-Cannoni, E Lechevallier, J F Pellissier

Abstract
This case report describes a 75 year old man who had a renal papillary carcinoma associated with a low grade tumour of the collecting ducts. These tumours showed different immunohistochemical patterns for epithelial membrane antigen, cytokeratin 19, and Ulex europaeus lectin expression. In addition, cytogenetic findings were 47, XY, +7 <7> and 45, XY, −8, add(12)(q−ter)<10> for the papillary renal carcinoma and the low grade tumour of the collecting ducts, respectively. This is the first report where these two types of tumour are associated and cytogenetically distinguished.

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Case report
A 75 year old man, without urological history, presented with chronic fatigue. Ultrasonography and computerised tomography showed two solid renal tumours. Neither clinical nor biological abnormalities were found. Tumours were located in the upper portion of the right kidney cortex (2.5 cm and 1.5 cm in diameter, respectively) (fig 1). The biopsy of the largest tumour showed a Fuhrman grade 2 renal papillary carcinoma. The patient underwent a radical right nephrectomy. During surgery, the abdominal examination also revealed an ectopic pancreas of the jejunal mesentery.

The cut surface of the 2.5 cm diameter tumour (fig 2, arrow) was grey to yellow, whereas the cut surface of the 1.5 cm diameter tumour was white with a microcystic pattern (fig 2, asterisk). In this second tumour, there was scant chronic inflammation in the interface separating the tumour and normal tissue, but no encapsulation. Neither haemorrhage nor necrosis were seen.

The architecture of the first tumour was almost exclusively tubulopapillary, with foamy cells within the stroma. The cells of the papillae were cuboidal with a finely granular and amphophilic cytoplasm (fig 3A, C). These cells were immunoreactive for cytokeratin 7 (CK7), vimentin, and epithelial membrane antigen (EMA) (fig 3E). No immunoreactivity was seen for the lectin Ulex europaeus (UEA-1), or the cytokeratin 19 (CK19). The architecture of the 1.5 cm diameter tumour was strikingly different, with a microcystic pattern. Hobnail cells lined these cysts and usually showed a lightly eosinophilic cytoplasm without pseudostratified nuclei (fig 3B, D). Nevertheless, neither high grade nuclear features nor mitosis were seen.

Figure 1  Computerised tomography with intravenous contrast showing two hypointense renal tumours.

Figure 2  Macroscopic examination: the two tumours show a grey heterogeneous cut surface (arrow) and a white homogeneous cut surface (asterisk).
The Alcian blue stain was slightly positive within the stroma. The cells were immunoreactive for CK7, CK19, and UEA-1 (fig 3F). No immunoreactivity was seen for vimentin and EMA. Mib-1 immunoreactivity was low, equivalent to that of normal neighbouring tubules. Cyto- genetic analysis showed two different abnormalities, which were 47, XY, +7 <7> and 45, XY, −8, add(12)(q−ter)<10> (fig 4) for the renal papillary carcinoma and the low grade tumour of the collecting ducts, respectively. The patient was followed up for two years without the occurrence of metastasis. He died from cerebral stroke.

**Discussion**

Distinction of collecting duct carcinomas from renal papillary carcinomas can be difficult because both tumours often show a predominant tubulopapillary pattern; this differential diagnosis occurred in this case. For the 1.5 cm diameter right tumour, the location near the medulla, the features of dilated tubules with hobnail cells, and the lack of papillae account for the origin from the collecting ducts. Furthermore, the tumour cells expressed UEA-1 and CK19, which are markers for collecting duct carcinomas and not for renal papillary tumours. In addition, collecting duct carcinomas show variable staining, which is generally more intense for high molecular weight keratins than is seen in other subtypes of renal carcinoma, including papillary renal tumours. We also excluded the diagnosis of cystic nephroma because of the lack of both flattened cuboidal cells and sharp septa within the tumour.

In our case, the well defined borders, the lack of desmoplasia and infiltration into renal parenchyma, and finally the low index of proliferation supported the hypothesis of a low grade tumour. Furthermore, cytogenetic analysis of the largest tumour was in accordance with the diagnosis of renal papillary carcinoma, with a typical trisomy of chromosome 7. Conversely, the cytogenetic characteristics of low grade tumour of the collecting ducts are not well established, but by using polymorphic microsatellite markers Polascik et al found frequent loss of heterozygosity in chromosome arm 8p. Our case supports the involvement of chromosome 8 in such tumours. Ectopic
pancreas was probably an incidental finding, although anecdotal congenital syndromes associate ectopic pancreas and renal cysts.  

Low grade carcinomas of the collecting ducts have already been reported in association with benign tumours, such as the metanephric adenofibroma.  Nevertheless, our present case is of particular interest, showing the previously undescribed association between a renal papillary carcinoma and a low grade tumour of the collecting ducts with cytogenetic findings. This association is a new finding and accounts for the existence of low grade collecting duct carcinomas.


Figure 4 Karyotype of the low grade tumour of the collecting ducts showing monosomy 8 and supplementary material on the long arm of chromosome 12: add(12)(q–ter). Other chromosomal losses are random.