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Primary collision tumors of the kidney composed of oncocytoma and papillary renal cell carcinoma: A review

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ABSTRACT

Background: There are well known cases of hybrid tumors of chromophobe renal cell carcinoma (RCC) and oncocytoma in kidney, where both tumors have the same cell of origin – intercalated cell of the collecting duct. However, collision tumors composed of neoplasms originating from different cell lineages such as oncocytoma and papillary RCC are extremely rare. Herein, we made a collective literature review of reported cases of collision tumors composed of oncocytoma and papillary RCC, adding a case that we recently experienced.
Material and methods: A PubMed database was search for collision tumors of the kidney composed of oncocytoma and papillary RCC and a collective literature review was made. To this cohort, we also added a recently encountered case with similar, confirmed by immunohistochemistry, morphological features.
Results: To date 8 cases of a collision tumor composed of papillary RCC and oncocytoma have been described in the literature. All of them had a smaller papillary RCC component present within a larger oncocytoma.
Conclusion: Because of a few cases of such a collision tumors reported, it is difficult to make classification and right clinical management of these patients. None of the reported cases had tumor recurrence or progression on a follow-up. The presence of only small portion of papillary RCC in a large oncocytoma raises a possibility of under-sampling of malignant component in large oncocytomas in core biopsy or surgically resected specimens. We recommend better sampling, particularly at the periphery of otherwise classic oncocytomas to unveil this possible association.

1. Introduction

There are well known cases of hybrid tumors of oncocytoma and chromophobe renal cell carcinoma (RCC), where both tumors have the same cell of origin – intercalated cell of the collecting duct. These tumors exhibit three morphological patterns: (1) an admixture of areas typical of oncocytoma and chromophobe carcinoma, (2) scattered chromophobe cells in the background of a typical oncocytoma, or (3) composed of large eosinophilic cells with intracytoplasmic vacuoles [1], perinuclear halos, prominent nucleoli but absent wrinkled nuclear contours [2].

However, to observe a primary renal collision tumor composed of histologically different neoplasms originating from two different linages is rare. True collision tumors are those composed of concurrent but independent tumors with different pathogenesis that have expanded into each other territory and merged with each other [3]. A collision tumor may be represented by a combination of benign–benign, benign–malignant or malignant–malignant tumors. The latter should be distinguished from a phenomenon of tumor to tumor metastasis with a conventional clear cell renal cell carcinoma being the most well-known recipient of metastatic tumors in the kidney [4].

Primary renal collision tumors are infrequent and found in a literature as rare cases reports. Because of a few cases of such a collision tumors reported, it is difficult to make classification and right clinical management of these patients. Herein, we made a collective literature review of reported cases of collision tumors composed of oncocytoma and papillary RCC, adding a case that we recently experienced.

2. Materials and methods

A PubMed database was search for collision tumors of the kidney

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Table 1
Review of the literature.

Authors (Ref. #)	Year	Size of oncocytoma (cm)	Size of p-RCC (cm)	p-RCC type, grade	Chromosomal alterations
1. Al-Saleem et al. [5]	2005	6	Small nests	No type or grade	Both components with trisomy 7
2. Rowsell et al. [6]	2007	1.5	0.7	Type 1, F2	Trisomy 7 in p-RCC
3. Vasuri and Fellegara [7]	2009	3.5	N/A	N/A	N/A
4. Floyd et al. [8]	2011	3.6	0.15	Type 2, F2	N/A
5. Sejben et al. [9]	2013	3.5	1.0	Type 2, F2	No abnormality in both components
6. Özer et al. [10]	2013	5.0	1.7	Type 2, no grade	N/A
7. Goyal et al. [11]	2015	6.4	1.0	Type 1, F2	Trisomy 17 in p-RCC
8. Baydar et al. [12]	2016	4.5	blended	Type 1, no grade	N/A
9. Current case	2016	3.7	Adjacent (25% of tumor)	Type 1, F1	N/A

p-RCC, papillary renal cell carcinoma; F - Fuhrman nuclear grade; N/A, not available.

Table 2
Results of the immunostains of our case.

	CD117	E-cadherin	Vimentin	CD10	CK7	RCCm	MOC31	AMACR
Oncocytoma	+	+	−	−	−	−	−	−
Papillary RCC	−	−	+	+	+	+	+	+

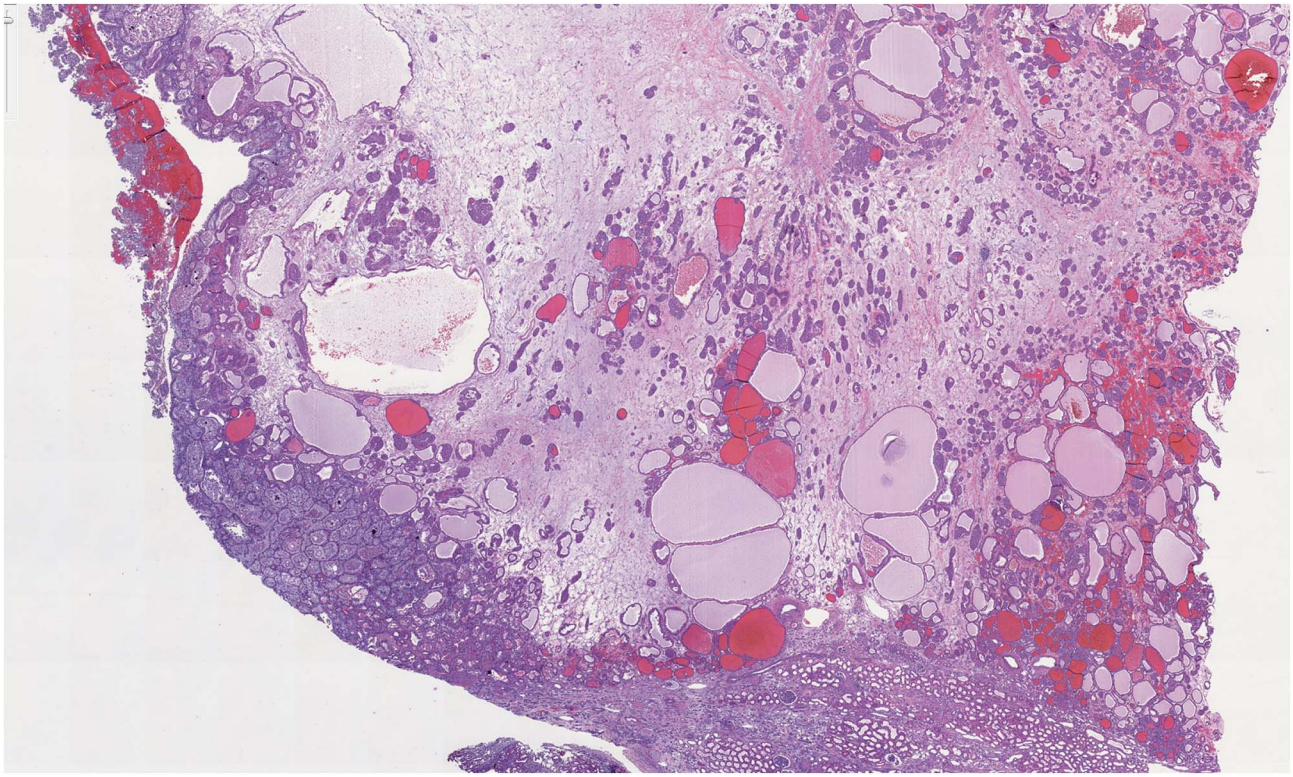

Fig. 1. Oncocytoma – papillary RCC collision tumor.Section of the kidney shows a tumor with two distinctively different tumor cell morphologies. The predominant tumor component is composed of oval-round oncocytic cells arranged in nests and small cystic structures embedded in a loose edematous stroma. Adjacent to this, at the periphery of the tumor nodule (lower left corner of the image), there is a second component exhibiting tubular and papillary architecture and composed of cells with columnar shape and clear-foamy cytoplasm. Non-neoplastic kidney parenchyma is seen at the bottom right portion of the image. (H & E stain, scanning magnification.)

composed of oncocytoma and papillary RCC. From every manuscript, the following data were recorded: patient's prior history of papillary RCC, sizes of the tumors, morphologic characteristics and immunohistochemical confirmation of two different tumors, type and grade of papillary carcinoma, chromosomal abnormalities if any detected. In the case that we recently encountered representative sections of the tumor were formalin fixed, paraffin embedded, sectioned and stained with hematoxylin and eosin (H & E) and later with immunohistochemical stains. Immunohistochemical stains for vimentin (predilute antibody, 20-minute incubation, clone V9, Ventana, Tucson, AZ), CD117 (predilute antibody, 12-minute incubation, clone 9.7, Ventana, Tucson, AZ), E-cadherin (predilute antibody, 8-minute incubation, clone 36, Ventana, Tucson, AZ), CD10 (predilute antibody, 16-minute incubation, clone SP67, Ventana, Tucson, AZ), CK7 (predilute antibody, 24-minute incubation, clone SP52, Ventana Tucson, AZ), RCCm (predilute antibody, 20-minute incubation, clone PN-15, Ventana, Tucson, AZ), AMACR (1:100 dilution, 32-minute incubation, clone p504S, Dako, Santa Clara, CA), and MOC31 (predilute antibody, 24-minute incubation, clone MOC31, Ventana, Tucson, AZ) were performed on 4 μm paraffin embedded sections. The slides were deparaffinized in xylene, then rehydrated through graded alcohol series. The endogenous peroxidase was blocked using 3% hydrogen peroxide. Slides were