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Incidental Renal Cell Carcinoma (RCC) with Endometrial Cancer: A Case Report

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Abstract

Incidental detection of two primaries call for oncosurgeon's own judgment to decide the best therapeutic approach as no guidelines exist given the rarity of condition.

This is the sixth reported case of incidental renal cell cancer in a patient of endometrial cancer.

Renal cell cancer was detected on preoperative Magnetic Resonance Imaging in a patient who presented as postmenopausal bleeding and histology proven endometrial carcinoma. Both primaries were simultaneously treated surgically. Final histology confirmed dual primaries with uterine primary being endometrioid adenocarcinoma stage Ic, estrogen progesterone receptor positive; where as renal primary was clear cell carcinoma stage Ib, estrogen and progesterone receptor negative.

Estrogen receptors have been identified in Hamster and mouse kidneys as well as in renal cell carcinoma tissues. High plasma estrogen found in some patients of synchronous renal and endometrial cancer may partly explain the association of these two primaries, though not in all cases. Increased serum leptin levels and a common low penetrance susceptibility gene have been reported in both these cancers.

Keywords: RCC, EndometrialCancer, Estrogen Receptors

Introduction

Although the incidence of multiple malignancies in the same patient is less than 4% (1), incidental renal cell carcinoma (RCC) with second primary tumor is common in these patients. That being said, our case of renal cell carcinoma with second primary tumor - endometrial carcinoma, is extremely rare with only five cases reported till now, with Lam et al. (2) reporting first such case.

The reason for the frequent observation of two tumors in same individual is poorly defined. In synchronous tumors a common etiology, such as exposure to the same hormone or carcinogen, is often postulated (3-5).

We here in report case of incidental RCC with endometrial carcinoma successfully managed by us.

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Case Report

A 56 years old multipara (G3P3L3) reported to us in February 2009 with postmenopausal bleeding per vaginum since three months. She had attained menopause ten years ago and never used oral contraceptive pills. There was no history of genital or colonic malignancy in her family. Clinically she appeared normal except obesity (Body mass index - 31.1). Vaginal examination showed normal cervix and vagina, bulky & retroverted uterus with no mass in pouch of Douglas. Her hematocrit and serum biochemistry were normal. Sonography of the pelvis showed bulky uterus with heterogenous myometrial texture. Histology of the endometrial curettage sample reported endometrioid adenocarcinoma (G1). Magnetic Resonance Imaging (MRI) of abdomen and pelvis revealed mass in endometrial cavity with deep myometrial invasion and an additional mass arising from lower half of the left kidney. Later she had Computer Tomography (CT) scan with angiography and three dimensional (3D) reconstruction studies which confirmed 7.2 x 6.8 cm hypervascular mass lesion in relation to lower half of the left kidney and hyper vascular uterine mass (Fig. 1, 1A, and 1B).

Preoperative evaluation including pulmonary, cardiac and hepatic systems was normal. She underwent radical hysterectomy with bilateral pelvic lymph node dissection through an infra umbilical midline incision. The patient's position was then changed to right lateral (45 degree tilt) and through 11th rib bed incision, left radical nephrectomy with para-aortic lymphadenectomy was performed in one sitting (Fig-2 Specimen). Her post operative recovery was smooth and she was discharged on tenth day. Histology of uterus was endometrioid adenocarcinoma of the endometrium FIGO grade 2, with tumor infiltrating more than half the myometrial thickness, pelvic nodes were negative. Estrogen and proges terone

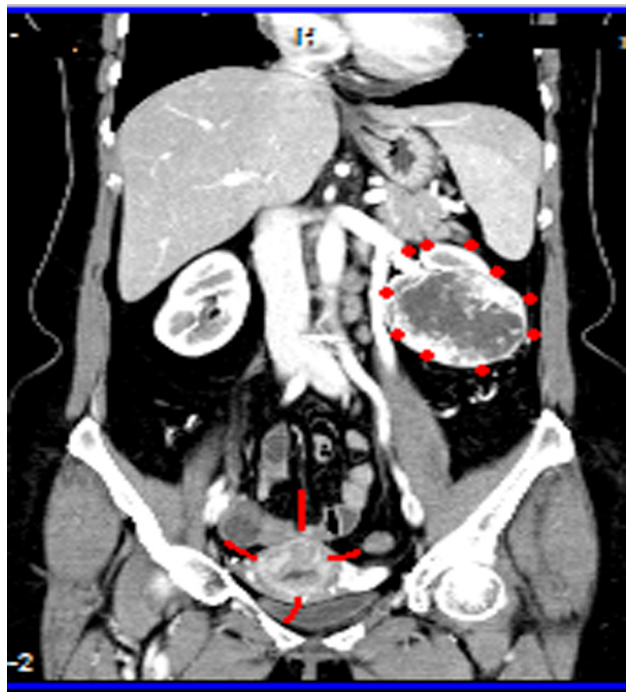

Figure 1. CT scan image of uterus and left kidney. Uterus marked by red arrows and left renal mass marked by red dots

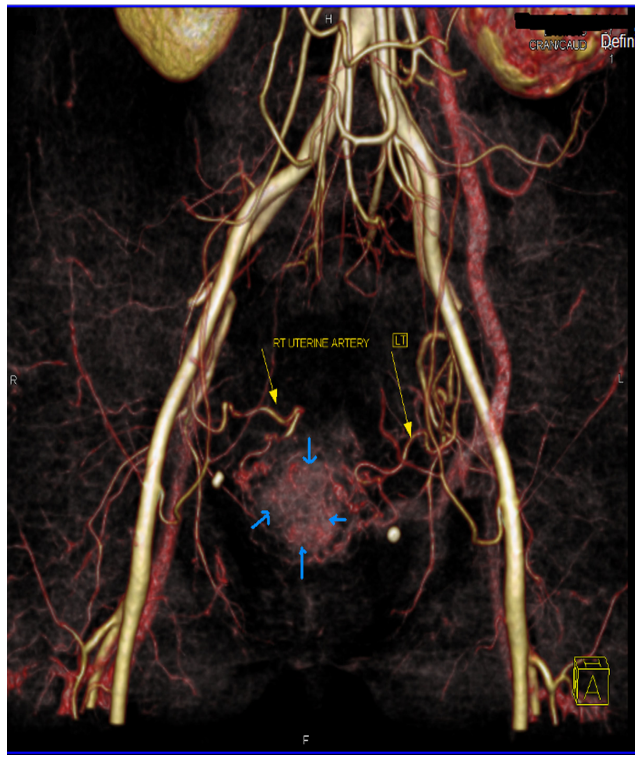

Figure 1A. CT angiography showing uterine mass (marked with blue arrow) and uterine arteries (marked by yellow arrows)

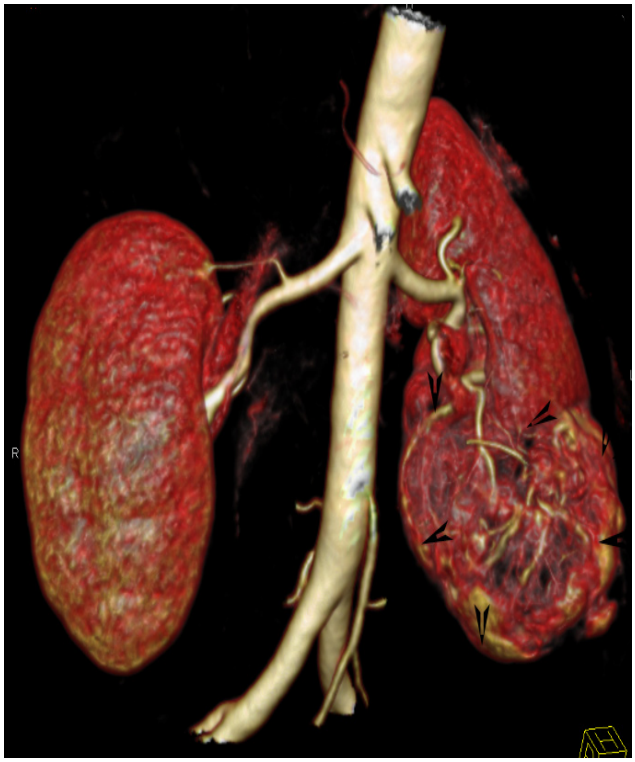

Figure 1B. 3dimentional CT scan showing left renal mass (marked by black arrow heads)

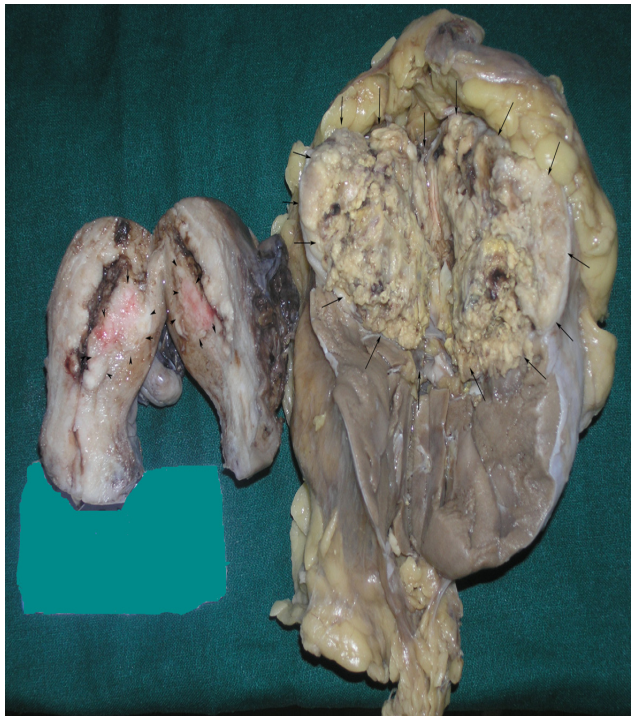

Figure 2. Cut open specimen of uterus and kidney. Endometrial mass marked by small black arrow heads and renal mass marked by black arrow

receptors were strongly positive. Histology of renal mass was clear cell carcinoma, Fuhrman nuclear grade 4. The renal capsule was intact; the tumor was infiltrating the sub-epithelial tissue of pelvicalyceal system. The vascular and ureteric cut margins were negative. Three left renal hilar nodes and nine left para-aortic nodes were negative for metastasis. Estrogen and progesterone receptors were negative.

She received adjuvant external beam pelvic radiotherapy with brachytherapy to the vaginal vault. At the latest follow up (three months) she is disease free.

Discussion

Although the etiology of endometrial and renal cell cancer (RCC) is unknown, there is some evidence that hyper estrogenic state, obesity and genetic predisposition may be high risk factors. Estrogen receptors have been identified in Hamster and mouse kidneys (6, 7) and RCC tissues (8, 9). Di Silverio F. (10) reported high plasma estrogen levels in 1 out of his 4 cases of RCC with endometrial carcinoma supporting the causal association. Obesity, which is associated with high leptin levels (cytokine derived from adipocyte) in Postmenopausal women with endometrial cancer (11). Leptin also promotes endometrial growth and invasiveness (12). In addition, Horiguchi et al. (13) found 38% of RCCs with renal vein invasion had high leptin receptor expression and high levels of serum leptin possibly due to leptin stimulated cell proliferation and induced activation of signal transducers (14). We have not done leptin levels in our patient; however, it is possible that obesity in our patient must have contributed to high leptin levels. Lastly, genetic predisposition in obese patients with malignancies has been studied over the years and there is some evidence that PPARG (Peroxisome proliferators activator receptor gamma)

is a receptor that under genetic control regulates differentiation and cell growth. Smith et al. (15) observed that PPARG serves as a common low penetrance susceptibility gene for cancers associated with obesity and high fat intake (such as endometrium, ovary, prostate, kidney and cervix).

In our patient preoperative imaging lead to the detection of incidental RCC as a second primary that altered the management and we believe that tracking or treating two primaries independently in one sitting is beneficial to the patients as it avoids morbidity of repeated operation theatre pursuits and hospital admissions. Adjuvant therapy in such scenario will depend on the histology of tumor in terms of local invasion, nodal status and adequacy of margins. Prognosis in these cases depends on biological potential of either primary. However, in our case we believe that endometrial cancer is likely to be more aggressive.

Conflict of Interest

None declared.

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