

Spontaneous Regression of Primary Renal Cell Carcinoma

— A. Case Report —

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Spontaneous regression of metastases or primary lesions of malignancy is a most intriguing phenomenon. However there were no reports of spontaneous regression of primary renal cell carcinoma. We reviewed a case of renal mass which was considered to be a spontaneously regressed renal cell carcinoma. This diagnosis was supported by the size of the renal mass, its total necrosis, and the discovery of a few remaining tumor cells. We cautiously report this case as a spontaneously regressed primary renal cell carcinoma in a 54 year old woman.

Key Words: Spontaneous regression, primary renal cell carcinoma.

Spontaneous regression of cancer has very rarely been documented in patients with neuroblastoma, melanoma, renal cell carcinoma and choriocarcinoma (1). Among these reported instances of spontaneous regression, renal cell carcinoma appeared only in metastatic lesions.

Such a metastatic lesion disappeared following a nephrectomy and with or without additional therapy such as radiation or hormone. We could not find one case of spontaneous regression of primary renal cancer in the literature.

Herein we report a case of renal mass which based upon gross and histological examination, we have diagnosed as a case of spontaneously regressed renal cell carcinoma.

CASE REPORT

In September 1985, a 54 year old woman was referred to our department with a one month history of left upper quadrant discomfort and general malaise. Using ultrasound study and an abdominal C-T scan the diagnosis from the previous hospital was of a left renal mass.

The only abnormal laboratory findings were an erythrocyte sedimentation rate (Wintrobe) of 28mm/h and a urinalysis report of 10-20 WBC/HPF and 2-3 RBC/HPF. Ultrasound study revealed a relatively anechogenic left upper renal mass with a sharp interface distinct from the surrounding renal parenchyma (Fig. 1). When studied by computed tomography the mass had an attenuation value of 14-37 and was not distinctly delineated from the adjacent parenchyma. There was no enlargement or evidence of a mass in the other kidney (Fig. 2).

A nephrectomy was performed. The excised kidney weighed 370gm, measured 14×7×6cm, and had a globular enlargement at the upper pole. A cross section of the upper pole mass revealed a thick, hard, round, cyst-like capsule 6cm in diameter with no demonstrable mucosa. It was filled with a dark reddish brown friable ground-like material (Fig. 3).

Microscopic examination of the capsule revealed that it was composed of thick collagen tissue with irregularly sized and shaped calcifications and had some degree of hyalinization. Macrophages containing lipid droplets and hemosiderin were also present. The capsule's lumen was filled with a mixture of coagulated blood and necrotic tissue. One small piece of tumor tissue was found near the capsule. It contained clusters of tubular structures of varying sizes and a scant amount of intertubular tissue. Most of the tumor cells displayed a condensed nucleus and cytoplasm (Fig. 4).

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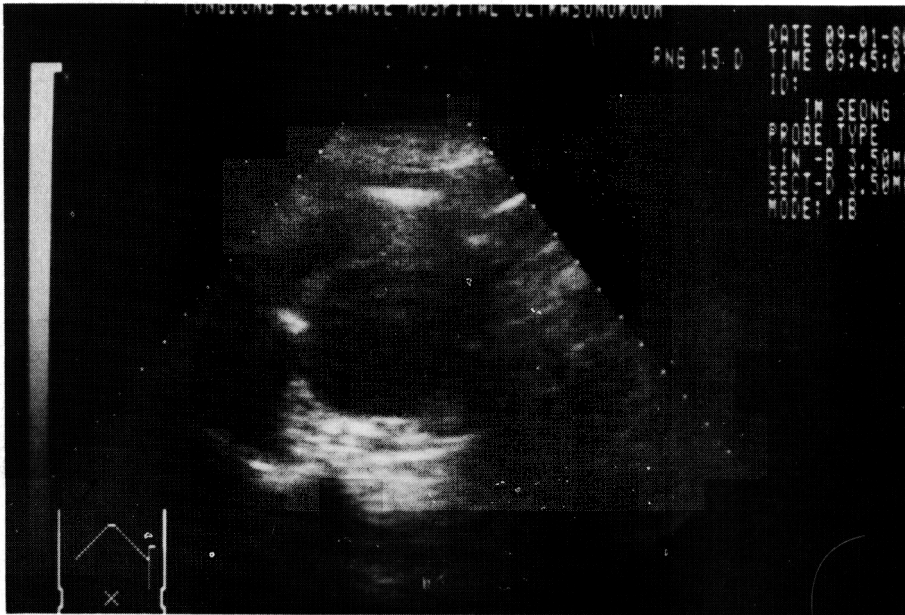


Fig. 1. Ultrasonogram shows a cystic mass with no echoes on the upper pole of L. kidney.

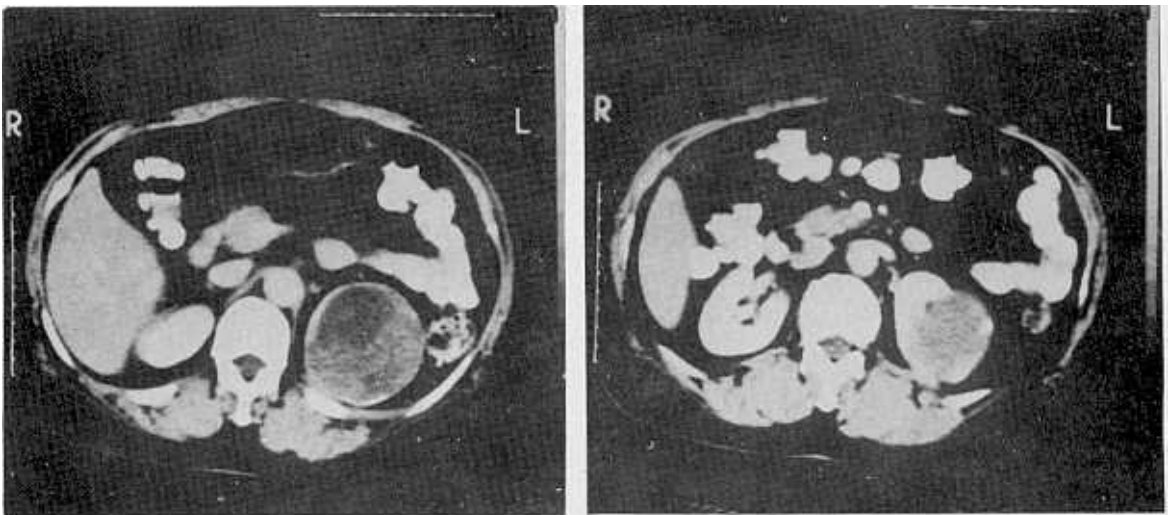


Fig. 2. CT shows an indeterminate mass on the upper pole of L. kidney. (Hounsefield No. 14-37).



Fig. 3. A well circumscribed mass with thick capsule and filled with hemorrhagic necrotic tissue in the upper pole.

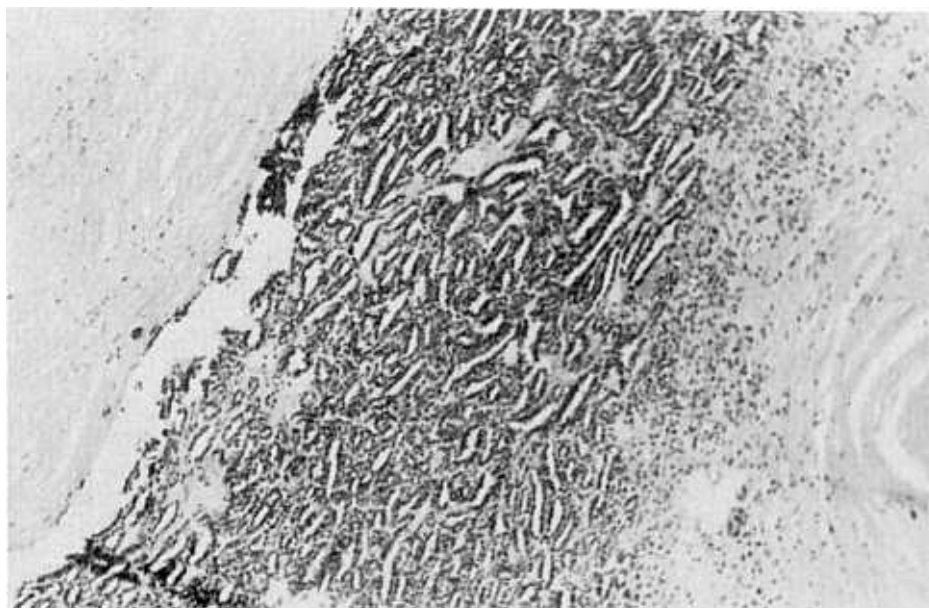


Fig. 4. Cluster of well differentiated tubular structures with areas of tumor necrosis is noted.

DISCUSSION

C-T scanning is a valuable investigative method that can be used to study the nature and extent of renal masses which have been discovered by screening urography (2). In this case the renal mass was characterized by poor delineation from the surrounding normal tissue, a thickened wall, scattered capsular calcification, and an attenuation value higher than that of a typical benign cyst. These findings were compatible with a hemorrhagic or inflammatory cyst, an abscess or a tumor. As renal angiography is of little help in making a definite diagnosis in cases of this type, Balfe etc. (3) recommended ultrasonographic guided aspiration or surgery. In our case, an inflammatory cyst or abscess could be excluded preoperatively based on the clinical symptoms and routine blood test. A metastatic lesion could also be excluded without the evidence of detectable primary site due to the symptoms, lab examinations and X-ray examination.

Robinson (1969) reported that regression changes in lesions of hypernephroma commonly show fibrosis, hyalinization, calcification, and ossification. Everson & Cole (1966) suggested that the tumor invaded the vein. This invasion led to regression of the tumor and replacement with a reactive fibrosis. Also, tumor cells were found only in hyalinized connective tissue.

Our case was characterized by a thick, hyalinized connective tissue capsule containing scattered calcifications, ferritin laden macrophages, necrotic tissue and a cluster of tumor cells. We concluded that it was a spontaneously regressed primary renal cell carcinoma.

In the case of renal cell carcinoma, the first reported case of spontaneous regression of metastases was by Bumpus in 1928. Since then there have been about 70 documented cases, but all of them have been metastases to the lungs. Bashford estimated a frequency of 0.001% for spontaneous regression of renal cell carcinoma in 1935. Bloom (1973) documented 3 acceptable instances of regression in a study of 1100 renal cell carcinoma patients who had undergone a nephrectomy. Bloom (1973) also reported a regression rate of 15-20% for metastatic lesions which were treated with a progestational agent following nephrectomy. Bracken etc. (1975) reported 2 cases of regression of metastatic lesions among 3 cases with a preoperative embolic infarction and a postoperative treatment with a

progestational agent.

Although there have been about 70 cases of spontaneous regression of metastatic lesion, until now we could not find any documentation of spontaneous regression of primary renal cell carcinoma.

The mechanisms thought to be involved in spontaneous tumor regression include a walling off mechanism which block the blood flow, a hormonally induced change, an infection and/or fever, and alterations of the immune status (11).

The term "spontaneously regressed renal cell carcinoma" can be applied to a tumor which has been confirmed and then regressed. In this case the diagnosis of a spontaneous regression of renal cell cancer is based upon the gross appearance and histology of the renal mass.

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