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Simultaneous duodenal and colon masses as late presentation of metastatic renal cell carcinoma

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We report a case of pathologically proven simultaneous duodenal and colonic metastases about four years after nephrectomy for mixed clear and granular cell type renal cell carcinoma (RCC). A 76-year-old female patient who had undergone a left radical nephrectomy 4 years previously for RCC presented with a 1-month history of dyspepsia and pain in the right upper abdomen. An abdominopelvic CT scan showed circumferential wall thickening with high enhancement at the second portion of the duodenum and additional enhancement of an irregular protruding mass into the lumen of the ascending colon. A gastroscopy showed a large and ulcerative protruding mass nearly obstructing the second portion of the duodenum. A colonoscopy revealed a polypoid, nodular and purplish mass in the ascending colon. Microscopy of the biopsy specimen showed the features identical to those of the RCC which was resected 4 years earlier in this patient. We believe this to be the first case illustrating a metastatic renal cell carcinoma as simultaneous duodenal and colon masses.

Key Words : *Renal cell carcinoma, Metastases, Duodenum, Colon*

INTRODUCTION

Renal cell carcinoma (RCC) may metastasize to any site of the body, but clinically evident metastatic intestinal involvement by RCC is extremely rare[1-7]. To our knowledge, simultaneous duodenal and colonic metastases have not been reported in the English literature. We report a case of pathologically proven simultaneous duodenal and ascending colonic metastases about four years after a left nephrectomy for RCC.

CASE REPORT

A 76-year-old female patient who had undergone a left radical nephrectomy 4 years previously for RCC (mixed clear and granular cell type, TNM stage III) presented with a 1-month history of dyspepsia, lethargy and pain in the right upper abdomen. Her blood pressure was 110/70 mmHg, pulse rate 80/min, respiration rate 22/min and body temperature 36.8℃. Her conjunctiva were slightly pale and chest was normal to auscultation. Abdominal examination revealed slight tenderness but normal peristalsis; however, a movable mass was palpated in the right upper abdomen.

The hematocrit was 28.1% and the white cell count was 9700/mm^3 with 77% polymorphonuclear cells and 14% lymphocytes. Serum sodium was 134 mEq/L, potassium 3.5 mEq/L, chloride 101 mEq/L and calcium 9.5 mg/L. The result of liver function tests were as follows: total protein 6.5 g/dL, albumin 3.5 g/dL, cholesterol 165 mg/L, bilirubin 0.6 mg/dL, alkaline phosphatase 105 IU/L, AST 28 IU/L and ALT 21 IU/L. The result of a stool examination revealed positive

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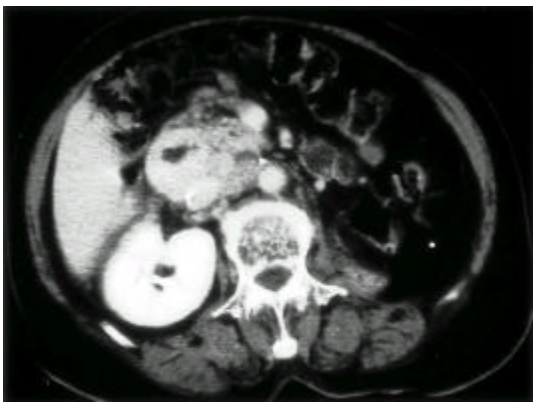

Figure 1A

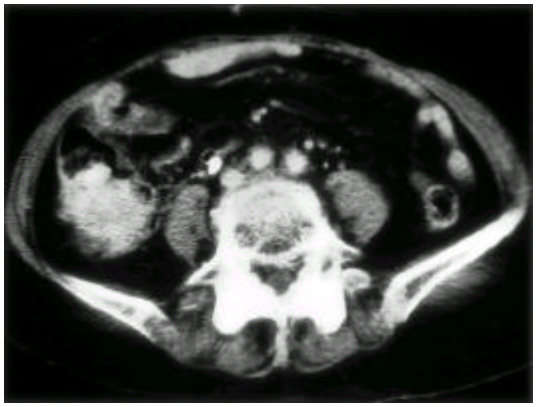

Figure 1B

Figure 1. Abdominopelvic CT scan. (A) Circumferential wall thickening with high enhancement in the second portion of the duodenum. (B) Enhancement of an irregular protruding mass into the lumen of the ascending colon.

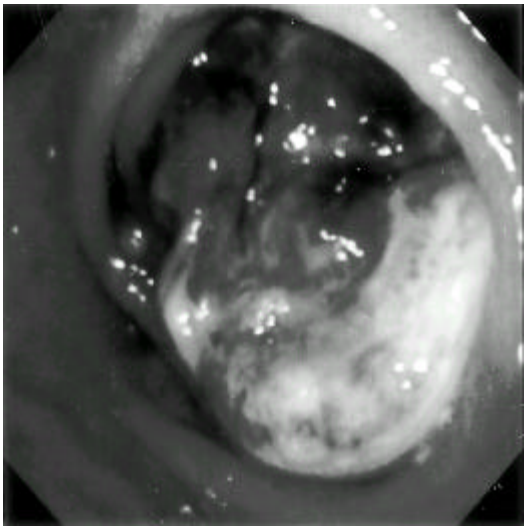

Figure 2A

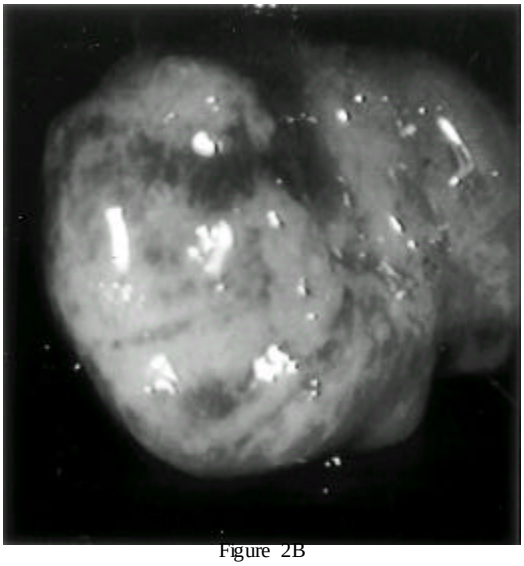

Figure 2B

Figure 2. Endoscopy. (A) A gastroscopic view shows an irregular multi-lobed, partially ulcerative and protruding mass nearly obstructing the second portion of the duodenum. (B) A colonoscopic view shows a purplish-colored, sharp-margined and nodular mass on the ascending colon.

occult blood. A chest X-ray showed no evidence of lymphadenopathy. Tumor marker levels, such as carcinoembryonic antigen (CEA), carbohydrate antigen 19-9 (CA19-9) and α-fetoprotein (AFP), were within normal limits.

An abdominopelvic CT scan showed circumferential wall thickening with high enhancement at the second portion of the duodenum and additional enhancement of an irregular protruding mass into the lumen of the ascending colon with multiple lymphadenopathy in the aortocaval area (Figure 1). A gastroscopy showed a large, irregular multi-lobed, partially necrotic and ulcerative protruding mass nearly obstructing the second portion of the duodenum. A colonoscopy revealed a polypoid, nodular mass in the ascending colon, which was purplish in color, had sharp margins and was pliable

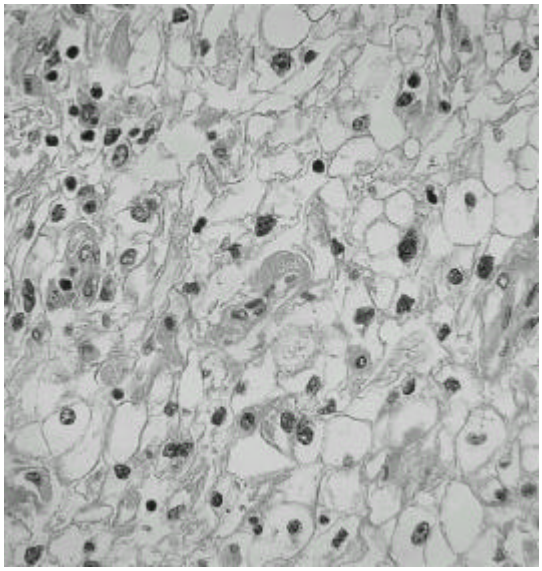

Figure 3A

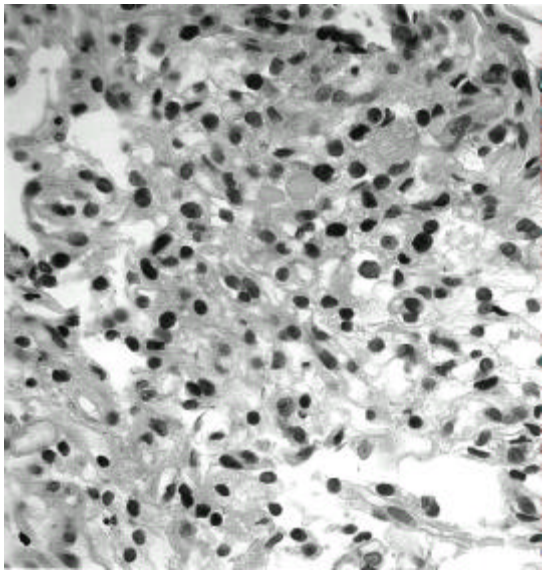

Figure 3B

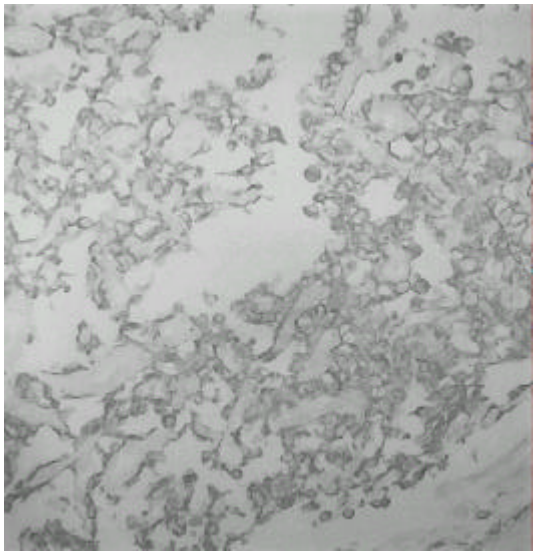

Figure 3C

Figure 3. Pathology. (A) Mixed clear and granular cell type renal cell carcinoma in the nephrectomy specimen 4 years ago (H&E X200). (B) Renal cell carcinoma in duodenal tumor (H&E X200). (C) Positive immunohistochemical stain for vimentin in duodenal tumor X200).

(Figure 2). Microscopic findings of biopsy specimens showed features identical to those of the renal cell carcinoma which was resected 4 years earlier in this patient and immunohistochemical stainings for vimentin was positive (Figure 3).

Interferon was given at a dose of 2 million IU per square meter three times a week. However, interferon therapy was discontinued after three months due to anemia, anorexia and general weakness. She is still alive 1 year later.

DISCUSSION

The biological behavior of RCC is characteristically variable and the prognosis is unpredictable[8]. The clinical course of the disease ranges from months to several decades and even spontaneous regression has been documented[9]. Renal cell carcinoma can disseminate locally by contiguity and metastatize to distant sites. Approximately 25% of patients with RCC have metastatic disease at the time of diagnosis[2]. The delayed

occurrence (as late as 31 years after a nephrectomy) of metastatic RCC is well known[3]. Solitary metachronous metastases from RCC are rare; however, they can occur very late in the course of the disease[1]. Renal cell carcinoma may metastasize to almost every organ of the body, but 95% of the metastatic lesions involve the lung, lymph nodes, liver, bone, adrenal glands and the opposite kidney[3]. However, there are scattered reports in the literature of clinically evident intestinal metastasis from RCC[1-7]. To the best of our knowledge, simultaneous duodenal and colonic metastases have not been reported in the English literatures.

A secondary tumor involving the intestinal tract may be caused by direct extension, lymphatic spread, peritoneal or hematogenous dissemination[4]. In the present case, duodenal and colonic metastases are not generalized carcinomatosis because any other metastasis is not occurred in common metastatic sites and duodenal and ascending colonic involvement are remote from a primary cancer of the left kidney.

Metastatic RCC, like the primary tumor, is frequently hypervascular[1, 5]. Hypervascularity of the present tumor, as suggested by a CT scan, gastroscopy and colonoscopy, was compatible with the characteristics of metastatic renal cell carcinoma. Also, positive immunohistochemical stain for vimentin elucidated that the duodenal and colonic masses are compatible with carcinoma rather than adenocarcinoma.

Surgical excision of the local recurrence is the best procedure for therapy, but this can be radical only when the recurrence can be completely excised[3]. Chemotherapies, including hormonal and interferon therapies, are effective in some patients with metastatic renal cell carcinoma[8]. In our case, only interferon treatment was done without surgical excision. Although Interferon treatment was used to treat metastatic RCC, it is not easy to evaluate its effectiveness because of premature discontinuance of the treatment.

In our opinion, endoscopists should consider the possibility of intestinal metastasis of RCC when endoscopic and CT studies show a hypervascular mass in a patient with a previous history of RCC.

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