CT Images of a Malignant-Transformed Ovarian Mature Cystic Teratoma with Rupture: a Case Report

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A malignant transformation or a tumor rupture is a rare complication of ovarian mature cystic teratoma (MCT). A tumor rupture in a malignant-transformed MCT has never been reported in the literature. We present the CT images of a 39-year-old woman showing a large, predominantly cystic mass in the lower abdomen, with fat-fluid-level ascites. A contrast-enhanced solid component, with regional discontinuity within the cystic lesion, is also demonstrated. The pathologic diagnosis of the ruptured MCT unveils the malignant transformation (squamous cell carcinoma) and mesenteric carcinomatosis.

A mature cystic teratoma (MCT) is the most common type of ovarian neoplasm; however, a tumor rupture of a MCT is rarely encountered (1, 2). The malignant transformation of a mature ovarian cystic teratoma is also a rare phenomenon that usually occurs in postmenopausal women (1). To our knowledge, destitute of reports pertaining to the malignant transformation of an ovarian mature cystic teratoma with rupture were found in the literature. Herein, we present the computed tomography (CT) images of a ruptured mature cystic teratoma with pathologically-proven malignant transformation (squamous cell carcinoma) in a 39-year-old woman.

Case Report

A 39-year-old, gravida 3, para 2, woman suffered from progressive abdominal fullness and intermittent tenderness of the abdomen for the last month. In questioning the patient, we found that she denied any traumatic history and she did not experience nausea, vomiting or changes in bowel habits. The patient’s past history and family history were also unremarkable.

A physical examination revealed a soft, yet distended abdomen with shifting dullness under percussion. The sonographic images revealed massive ascites and a large cystic tumor mass with solid components in the lower abdomen. An abdominal CT scan (Sensation 16; Siemens, Forchheim, Germany; Parameters: SD 5 mm, increment 5 mm, 120 kV at 217 mAs) revealed a huge cystic mass (greater than 15 cm of the maximal linear dimension), mainly in the lower abdomen, and presented with several irregular, enhanced, and solid components. Further, a calcified entity (greater than 1000 HU; Hounsfield Units) and scanty fat-density contents (~120 HU) were measured with a round region of interest (ROI) using a FDA-approved image viewer software (SmartIris, TEDPC, Taiwan) (Fig. 1A). The tumor margin disrupted at one enhanced solid component (Fig. 1B), and the abdomen was filled with moderate fluid.
amounts of ascites with thickened, well-enhanced peritoneum, and multiple fat-fluid levels (fat: less than −130 HU) scattered in the bilateral subphrenic regions (Figs. 1C, D). The patient’s laboratory test results showed leukocytosis (white cell count: 14,280/ml; neutrophils: 83%), low hemoglobin (11.5 g/dl), and high CA-199 levels (87.7 U/ml; normal range, < 37 U/ml). In addition, a chest radiography showed clear lungs.

During the operation, 2,400 mL of bloody ascitic fluid was drained from the patient. The greater omentum was matted, and measured to be about 10 × 10 × 8 cm. In addition, several adhesions were found between the liver and diaphragm. The uterus and right ovary appeared normal; however, tumor seeding was suspected due to some neogrowths on the surface of the bladder and rectum. The intra-operative frozen section showed moderately differentiated neoplastic squamous cells with hyperchromatic and pleomorphic nuclei. Next, we performed suboptimal debulking surgery, total hysterec-

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![Fig. 1. Malignant transformation of mature cystic teratoma.](image)

A. Contrast-enhanced CT scan shows predominantly cystic mass (arrowheads) in lower abdomen with irregular, enhanced solid components (thick arrow; before contrast [not shown], 30–35 HU; after contrast, 75–80 HU), and calcified entity (thin arrow, more than 1000 HU) with scanty fat contents (curved arrows, −120 HU).

B. Contrast-enhanced CT image caudal to A shows disruptive tumor margin (thick arrow) at enhanced solid components (thin arrows) of lesion.

C. Contrast-enhanced CT scan shows thickened, contrast-enhanced peritoneum (thin arrows) and omentum (thick arrow).

D. Contrast-enhanced CT scan shows moderate ascites with multiple scattered fat-fluid levels (thin arrows; fat: less than −130 HU) in bilateral subphrenic regions.
tomy, and an appendectomy.

The microscopic findings of the cystic tumor showed three germ cell components, which revealed some characteristics of mature cystic teratoma and a submucosal layer of solid component filled with differentiated neoplastic squamous cells and keratin pearl formation (Figs. 1E-G).

The pathological examination revealed tumor involvement in the right ovary, bilateral fallopian tubes, omentum, appendix, as well as the surface of bladder and rectum. In addition, we also noted lymph node metastases around the left adnexa of the uterus.

After surgery, the patient received three courses of chemotherapy and a one time adjuvant radiation therapy. Unfortunately, the patient died four months later due to an uncontrolled urinary tract infection.

**DISCUSSION**

A mature cystic teratoma is composed of a well differentiated derivation of three germ cell layers (endoderm, mesoderm and ectoderm), and accounts for 10–20% of all ovarian neoplasms (1, 2). The complications associated with cystic teratoma cases include torsion (16%), malignant degeneration (2%), rupture (1–2%), and infection (1%) (1, 2). A mature cystic teratoma may rupture into the peritoneal cavity or the adjacent visceral organs (2, 3). Few CT images of an intraperitoneal rupture of a benign cystic ovarian teratoma have been reported (2, 4). Some hypotheses for the causes of tumor rupture include torsion with infarction of the tumor, direct trauma, prolonged pressure from pregnancy, delivery, infection of the dermoid contents, malignant change, pregnancy

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**Fig. 1.** Malignant transformation of mature cystic teratoma.  
**E.** Cystic tumor consists of three germ cell components: squamous cell epithelium (ectoderm: curved black arrow), adipose tissue (mesoderm: thick black arrow), sebaceous gland (mesoderm: black arrowhead) and pseudostratified columnar epithelium (endoderm: thin black arrow) (Hematoxylin & Eosin staining, × 40).  
**F, G.** Cystic tumor contains squamous cell epithelium with indistinct basement (thin black arrow), diffuse submucosal infiltration of differentiated neoplastic squamous cells with hyperchromatic and pleomorphic nuclei (thick black arrows) and keratin pearl formation (arrowheads) (F: Hematoxylin & Eosin staining, × 40; G: Hematoxylin & Eosin staining, × 400).
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hormones, increased internal pressure from the rapid growth of the cyst, and tumor size (3, 5). In our case, the disruptive tumor margin is revealed in the CT images (Fig. 1B). To the best of our knowledge, a tumor rupture due to a malignant-transformed ovarian cystic teratoma has never been reported in the literature.

A malignant tumor transformation arising from an ovarian cystic teratoma is uncommon (1, 6, 7), and usually occurs in postmenopausal women. In contrast, a benign cystic teratoma usually occurs in women of reproductive age (1). The ovarian cystic teratomas reported to be associated with a malignant transformation, consist of a suite of histological differentiations. Squamous cell carcinoma (SCC) arises from the squamous lining of the cyst in the most common supervening type of malignant transformation, accounting for 80-83% of cases, followed by adenocarcinoma (7%), and sarcoma (7%) (6, 7). The typical CT findings of an ovarian cystic teratoma, with malignant transformation finds the tumor to be a fat-containing cystic complex of irregularly lobulated and heterogeneously enhanced solid components (6, 7). However, the imaging findings in the benign and malignant cystic teratomas often overlap. Our case study reveals a disruptive tumor margin at the enhanced solid component, with a pathologically proven squamous cell carcinoma. If these aforementioned findings could provide support for this scenario, while confronting the MCT rupture encountered in the solid component, we could include the malignant transformation into the list of differential diagnoses.

Mature cystic teratomas are easily diagnosed based on imaging studies which reveal their characteristic intratumoral fat and calcification components (8). The typical imaging characteristics of mature cystic teratomas are well known, but some atypical imaging manifestations can be misleading, such as tumors with scanty or without a fat component, pure fat in the cyst without the other component, a collision tumor, torsion, infection or rupture of teratoma, and association with a malignant transformation (1). In our case, scanty fat content and a small calcified entity are well depicted from the admission CT images. Further, the numerous intraperitoneal fat-fluid levels are considered to be a consequence of tumor rupture. Herein, the presence of scanty-fat cystic teratomas, associated with extra-tumoral fat, may imply tumor rupture with fat expulsion.

Diffuse peritonitis resulting from a benign cystic teratoma rupture may mimic carcinomatosis due to advanced ovarian malignancy (2, 3). The CT images in our case study revealed diffuse peritonitis, moderate ascites, and omental thickening. These findings suggest a more complicated radiological impression of the mixed benign and malignant processes, which are proven by a later pathologic report. In assembling all the clues, we found that the combination of scanty-fat ovarian cystic teratomas with disruptive solid parts, intraperitoneal fat-fluid levels, peritoneal thickening, and omental caking, should arouse the possibility of malignant tumor transformation with episodes of tumor rupture, even in a pre-menopausal woman, such as in our case.

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