Case Report

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Anaplastic carcinoma showing rhabdoid features combined with ovarian mucinous borderline cystadenoma: a case report and literature review

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Abstract

Anaplastic carcinoma in an ovarian tumor (ACOT) is rare. There have been a few controversial cases illustrating the clinical characteristics and prognostic factors of ACOT, which are not well known. A 60-year-old Chinese woman presented with a large pelvic tumor. A transvaginal ultrasound examination showed a large single ovarian cystic tumor with mural nodules and ascites. A gross ovarian mass with a size of approximately $20 \times 10 \times 15$ cm^3 was found. The content of the ovarian cyst was light yellow and chocolate-like, and a large grayish mural nodule of approximately 10 cm was found on the cyst wall. Histological diagnosis of ovarian mucinous borderline cystadenoma with a mural nodule of anaplastic carcinoma showing rhabdoid features and International Federation of Gynecology and Obstetrics (FIGO) stage IIIa was made. Fifteen months after surgery, the patient had received six courses of paclitaxel and carboplatin. She is still alive without any recurrence of the tumor. Findings from the present case suggest that patients with ACOT and FIGO stage IIIa would benefit from surgery and chemotherapy of paclitaxel and carboplatin. We also review the clinical features and survival rate of patients with ACOT using the Surveillance, Epidemiology, and End Result database, and summarize previously reported treatments.

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Introduction

Mural nodules are rare, and are found in the wall of various ovarian mucinous tumors (including malignant, borderline, and benign tumors).[1] Mural nodules can be classified into sarcoma-like mural nodule (SLMN), anaplastic carcinoma, and true sarcoma. As a malignant nodule, anaplastic carcinoma in an ovarian tumor (ACOT) is an exceedingly rare type, which was first reported in 1982.[2] ACOT is histologically divided into three patterns of rhabdoid, spindle (sarcomatoid), and pleomorphic (combined sarcomatoid and rhabdoid) patterns. These three histological patterns of ACOT have no adverse effects on prognosis, but diversity of the tumor causes a challenging histological diagnosis.[3]

Because of the rarity of this disease, there have been a few controversial case reports on ACOT.[2–5] To date, the clinical characteristics, prognostic factors, and standardized treatments of patients with ACOT are not well known, greatly limiting the diagnosis and treatment of this disease.

We report a 60-year-old woman with ACOT. Microscopic and immunohistochemical findings of this case were analyzed, and the clinical treatments and outcomes of the patient are described. Moreover, we reviewed the clinical features and survival rate of patients with ACOT using the Surveillance, Epidemiology, and End Result (SEER) database, and summarized previously reported treatments. These results may help patients and gynecologists to better understand this disease and make informed decisions for treatment of patients with ACOT.

Case presentation

This case report was prepared following the CARE Guidelines.[6] A 60-year-old Chinese woman with a large pelvic tumor was transferred to the First Affiliated Hospital of Sun Yat-sen University on the suspicion of ovarian malignancy in April 2019. She showed increased abdominal fullness, but no fever, abdominal pain, nausea, vomiting, vaginal bleeding, or dysuria.

A transvaginal ultrasound examination showed a large, single, ovarian, cystic tumor ($186 \times 103 \times 151\,mm^3$) containing anechoic and solid areas. An abundant blood flow signal was observed in the solid part of the tumor (Figure 1). These examination results suggested the presence of a malignant tumor, and therefore, the patient underwent exploratory laparotomy. Computed tomography and magnetic resonance imaging were not performed because of financial constraints. Serum levels of cancer antigen (CA) 125 and CA19-9 were 283.30 U/mL and 2470.74 U/mL, respectively, and both were above the normal range (cut-off values for CA125 and CA19-9: 35 U/mL and 35 U/mL, respectively) and kept increasing. During surgery, a gross ovarian mass (approximately $20 \times 10 \times 15\,cm^3$) with a smooth outer surface was found. The content of the left ovarian cyst was light yellow and chocolate-like, and a large grayish mural nodule of approximately 10 cm in diameter was found on the cyst wall. Resection of the ovarian mass was assessed by intraoperative frozen section analysis, and the patient was primarily diagnosed with ovarian mucinous borderline cystadenoma. Finally, the patient had

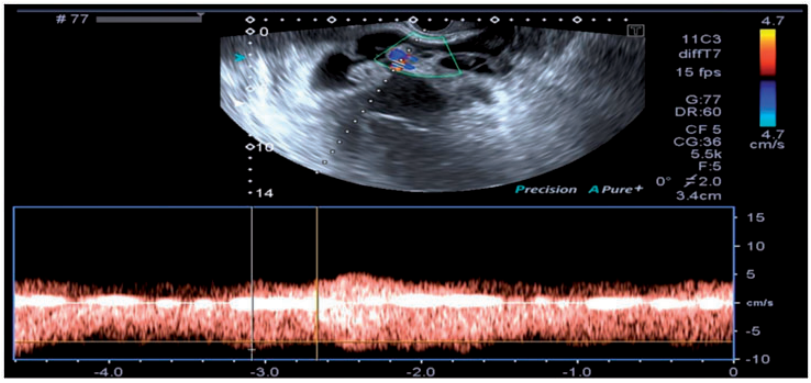


Figure 1. Ultrasonic image of an ovarian tumor with an unclear boundary and irregular shape. The tumor contains anechoic and solid areas. An abundant blood flow signal can be seen in a solid nodule (green box), showing a low resistance arterial spectrum (resistance index = 0.17).

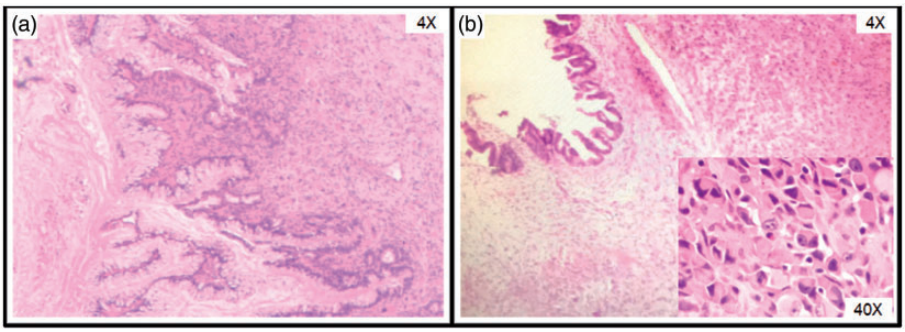


Figure 2. Ovarian borderline mucinous cystadenoma with anaplastic carcinoma (hematoxylin–eosin staining). (a) The bulk of the tumor is composed of borderline mucinous cystadenoma where proliferation of glandular architecture can be seen (4×). (b) The anaplastic carcinoma (rhabdoid pattern) is composed of large anaplastic cells with an ample eosinophilic cytoplasm and prominent nucleoli (4×; insert: 40×).

optimal debulking of the tumor performed, which included bilateral salpingo-oophorectomy hysterectomy, omentectomy, appendicectomy, resection of a superficial tumor of the bladder, resection of a tumor in the Douglas pouch, and peritoneal multipoint biopsy.

A histopathological examination showed cells with dysplasia and cells with a diffuse patchy growth pattern, which were diagnosed as ovarian mucinous borderline cystadenoma and anaplastic carcinoma, respectively (Figure 2). Nodules displayed a rhabdoid pattern, diffuse arrangement of cells with abundant, bright, and eosinophilic cytoplasms, and one or more prominent nucleoli with an atypical ovoid and eccentric shape. The mural nodules were positive for cytokeratin, epithelial membrane antigen (EMA), vimentin, desmin, integrase interactor 1 (INI-1), P53 (90%), and Ki-67 (50%) (Figure 3). These nodules were negative for Wilms tumor 1 (WT-1), paired box 8 (PAX8), estrogen receptor (ER), progesterone receptor (PR), cluster of differentiation 10 (CD10), melanosome (HMB-45), S-

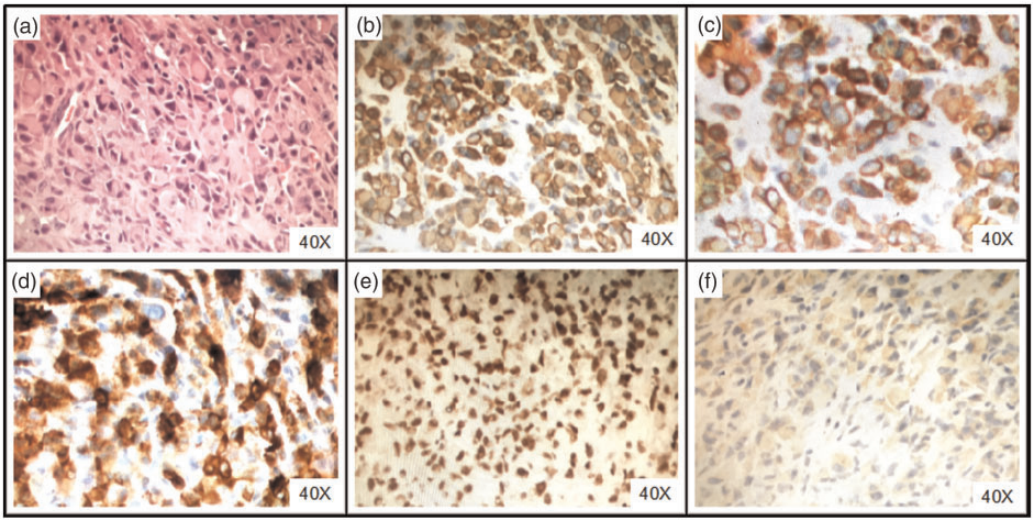


Figure 3. Microscopic and immunohistochemical mural nodule findings. (a) Hematoxylin–eosin stained section showing dense, undifferentiated, polymorphic, and eosinophilic cells with hyperplasia in mural nodules. (b) Mural nodule showing positive immunohistochemical staining for cytokeratin. (c) Mural nodule showing positive immunohistochemical staining for epithelial membrane antigen. (d) Mural nodule showing positive immunohistochemical staining for vimentin. (e) Mural nodule showing positive immunohistochemical staining for integrase interactor 1. (f) Mural nodule showing weak positive immunohistochemical staining for desmin.

100, actin, myogenin, myogenic differentiation 1 (MyoD1), CD56, synaptophysin (Syn), chromogranin A (CgA), and inhibin-α. The same lesions were also observed in the left oviduct, the omentum, and the surface of the bladder. A small number of atypical cells were identified in the ascites. Therefore, we diagnosed the patient as having an anaplastic carcinoma in ovarian mucinous borderline cystadenoma with International Federation of Gynecology and Obstetrics (FIGO) stage IIIa. She received six courses of paclitaxel (240 mg) and carboplatin (120 mg) with an interval of 3 to 4 weeks.

After the adjuvant chemotherapy, the patient developed mild complications of chemotherapy, such as hair loss and leukopenia. Fifteen months after surgery, the patient is still alive without any recurrence of the tumor (Figure 4).

Discussion

ACOT is an infrequent disease, and can arise in any ovarian mucinous tumor. During the past decades, only a few cases of ACOT have been reported.[7–9] Further investigations on the clinical characteristics of ACOT are required to better understand this disease and examine effective treatments.

We report a 60-year-old Chinese patient with FIGO stage IIIA and ACOT. To investigate clinical characteristics of patients with ACOT, a dataset of ACOT from 2004 to 2013 was extracted from the SEER database using the following classification code of International Classification of Diseases for Oncology, the third Edition (ICD-O-3): primary tumor originated from ovarian (C569) and anaplastic carcinoma (8021)[10] (Table 1). A total of 140,487 patients with ovarian

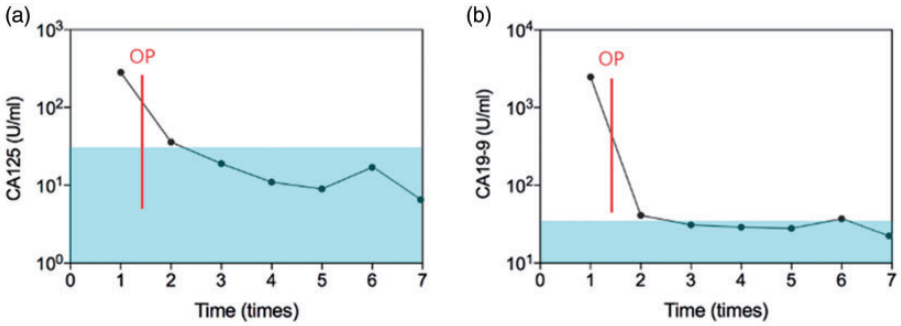


Figure 4. Levels of tumor biomarkers during treatment. (a) Cancer antigen (CA) 125 levels during treatment; (b) CA19-9 levels during treatment.

tumors were recruited, among whom 177 (1.26%) patients with ACOT were chosen to include in this report. The clinical characteristics of the 177 included patients are listed in Table 1. We found that 77.4% (137/177) of the patients with ACOT were diagnosed in the advanced stage. Similar to other types of general ovarian cancer, detecting ACOT cases in the early stage is a challenge. In contrast, Provenza et al.[3] reviewed published case reports of ACOT and showed that only 22.2% (4/18) of patients were in the advanced stage. Despite clinical data of ACOT reported in the literature, statistical significance was limited by the small cohort size.

We report a case of ACOT, which expands our knowledge regarding the behavior and morphological spectrum of ACOT. Mural nodules can present with any ovarian mucinous tumor (benign, borderline, or malignant), and are commonly divided into SLMN, ACOT, and true sarcoma.[1] Distinguishing the three types of lesions is difficult, but important, because they differ in overall survival time and prognosis.[1] Immunohistochemistry can be used as an identification method because of the morphological similarity of these mural nodules.[1] ACOT nodules tend to stain strongly for cytokeratin and are negative for vimentin, whereas SLMN and

Table 1. Clinical characteristics of patients with anaplastic carcinoma in an ovarian tumor.

Variable	Patients' characteristics (n = 177)
Age, median (IQR), years	64 (52–74)
Marital status at diagnosis, n (%)	
Single	25 (14.1)
Married	89 (50.3)
Separated	3 (1.7)
Divorced	12 (6.8)
Widowed	45 (25.4)
Unknown	3 (1.7)
Race, n (%)	
White	163 (92.1)
Black	5 (2.8)
American Indian/Alaskan native	2 (1.1)
Asian or Pacific Islander	7 (4)
FIGO stage, n (%)	
I	24 (13.6)
II	16 (9)
III	126 (71.2)
IV	11 (6.2)
Operation, n (%)	
No operation	49 (27.7)
Operation	118 (66.7)
Unknown	10 (5.6)
Survival, n (%)	
Alive or dead due to cancer	135 (76.3)
Dead	20 (11.3)
Not the first tumor	22 (12.4)

IQR, interquartile range; FIGO, International Federation of Gynecology and Obstetrics.

Table 2. Summary of cases of anaplastic carcinoma in a mucinous cystic ovarian tumor.

Author	Age (years)	FIGO stage	Surgery	Disease	Size of nodule (cm)	Adjuvant therapy	Chemotherapy regimen (cycles)	Follow-up (months)
Prat et al.[2]	46	Ia	TH+BSO+OM	Carcinoma	1.7	Chemo	Alkeran	DOD (4)
	46	Ia	TH+BSO	Carcinoma	4.5	–	–	DOD (7)
	72	III	TH+BSO+OM	Carcinoma	2	Chemo	Adriamycin (6); Cyclophosphamide +cisplatin (9); Cyclophosphamide (12)	NED (18)
	17	III	USO+OM+LN	Carcinoma	11	Chemo	–	–
Czernobilisky et al.[13]	75	Ia	TH+BSO+OM+LN	Carcinoma	3	Rad	–	NED (12)
Yamana et al.[14]	27	Ia	TH+BSO	Carcinoma	–	None	–	DOD (120)
Hayman et al.[7]	50	Ia	TH+BSO	Carcinoma	4	None	–	DOD (12)
Fujii et al.[15]	29	Ia	TH+BSO	Borderline	2	None	–	NED (22)
Chan et al.[16]	30	IIIb	TH+BSO+OM	Borderline	2	Chemo	Cisplatin +cyclophosphamide	NED (4)
Kessler[17]	22	Ia	TH+BSO	Carcinoma	–	–	–	–
Nichols et al.[11]	66	Ia	TH+BSO+OM	BIEC	0.5	Chemo	Cisplatin/cytoxan (6)	NED (12)
	74	Ia	TH+BSO+OM	BIEC	4	Chemo	Alkeran (2)	DOD (12)
	45	Ia	TH+USO	Carcinoma	4	None	–	NED (47)
Sondergaard et al.[4]	66	Ic	TH+BSO+OM+A	Carcinoma	12	–	–	DOD (3)
	29	Ia	TH+BSO	Carcinoma	1.5	–	–	NED (24)
	37	Ia	TH+BSO+OM+A	Borderline	22	–	–	NED (18)
Tsuruchi et al.[12]	18	IIb	TH+BSO+OM	Carcinoma	4.5	Chemo	Cisplatin+adriamycin +cyclophosphamide (1); cisplatin+adriamycin (9)	DOD (41)
Hellemans et al.[18]	38	Ic	TH+BSO+OM+LN	Carcinoma	–	None	–	NED (30)
Nakamura et al.[19]	28	Ia	TH+BSO+OM+LN	Carcinoma	1	None	–	NED (24)
Baergen et al.[20]	37	III	TH+BSO+OM	Carcinoma	1	Chemo	–	DOD (6)
Hillesheim et al.[21]	40	Ia	TH+BSO+A	Carcinoma	8	–	–	NED (12)
Yamazaki et al.[9]	45	Ia	TH+BSO+OM	Borderline	3.6	None	–	NED (15)
Mhawech-Fauceglia et al.[1]	36	Ia	TH+BSO+A	Borderline	2.2	None	–	DOD (3)
Okumura et al.[22]	53	IIIb	TH+BSO+OM	Borderline	4	Chemo	Paclitaxel+carboplatin (6)	NED (36)

(continued)

Table 2. Continued.

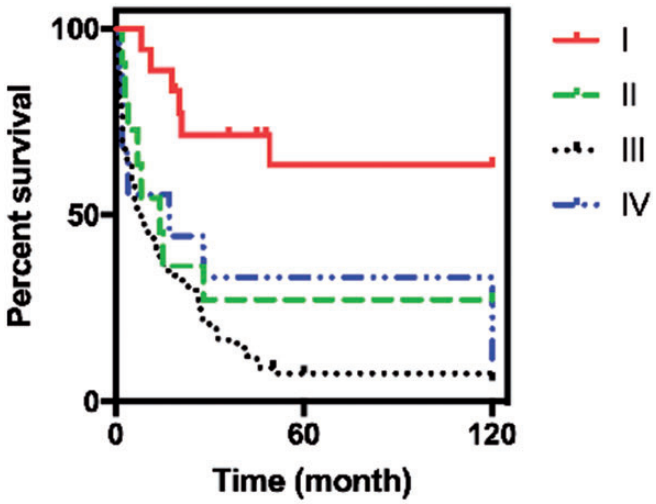
Author	Age (years)	FIGO stage	Surgery	Disease	Size of nodule (cm)	Adjuvant therapy	Chemotherapy regimen (cycles)	Follow-up (months)
Ardakani et al.[23]	48	IV	–	Carcinoma	1.4	–	–	AWD (10)
	22	IIIB	–	Carcinoma	0.5	–	–	NED (17)
	30	Ia	–	Borderline	3	–	–	–
	68	IIIa	–	Carcinoma	4	–	–	DOD (11)
	43	Ic	–	Carcinoma	0.5	–	–	DOD (15)
	37	IIIb	–	Carcinoma	1.9	–	–	–
	39	Ic	–	borderline	0.8	–	–	–
Zheng et al.[8]	48	Ia	BSO+OM+A	Carcinoma	4.5	Chemo	Paclitaxel+carboplatin (6)	NED (12)
Kihara et al.[24]	64	IIb	TH+BSO+OM +LN+A	Carcinoma	3.5	Chemo	Paclitaxel+carboplatin (6); gemcitabine (1)	DOD (9)

FIGO, International Federation of Gynecology and Obstetrics; TH, total abdominal hysterectomy; BSO, bilateral salpingo-oophorectomy; OM, omentectomy; Chemo, chemotherapy; DOD, died of disease; NED, no evidence of disease; USO, unilateral salpingo-oophorectomy; LN, lymph node sampling; Rad, radiotherapy; BIEC, borderline tumor with intraepithelial carcinoma; A, appendectomy; AWD, alive with disease.

sarcomas are negative for cytokeratin, but positive for vimentin.[11] As reported previously, in ACOT nodules, cytokeratin (AE1/3) and CAM5.2 are typically positive, vimentin, desmin, and PAX8 are variable, and ER and PR are usually negative.[1,11,12] In our case, the nodules were positive for cytokeratin (AE1/3), epithelial membrane antigen, vimentin, and integrase interactor 1 (Figure 3), and negative for Wilms tumor 1, PAX-8, ER, and PR. This led to the diagnosis of ACOT.

The foci/nodules of ACOT can be divided into rhabdoid, spindle, and pleomorphic patterns.[3] Our case was rhabdoid ACOT, and showed diffuse arrangement of large cells containing one or more prominent nucleoli, and a bright, eosinophilic cytoplasm and eccentric nuclei. Although these categories of foci/nodules do not have an effect on patients' outcomes and prognosis, they can make histological diagnosis difficult.[3]

Currently, there is no standard treatment for ACOT owing to the lack of knowledge on ACOT. Previous reports have highlighted the importance of adjuvant chemotherapy in postoperative management of ACOT. We reviewed and summarized previous literature on ACOT[1,4,7–9,11–24] (Table 2), and found that adjuvant chemotherapy was used in postoperative management for some stage I patients and most patients at a higher stage (stage II, III, or IV). Among cases of ACOT in the literature, regarding the 22 patients in stage I, two of four patients died after chemotherapy, three of eight patients died without chemotherapy, one patient with radiation treatment was still alive at 12 months, and information on chemotherapy was not available for nine patients. Therefore, determining whether adjuvant chemotherapy improves the overall survival rate of stage I patients is difficult. Furthermore, platinum-based chemotherapy was used for most of the ACOT cases (7/9) and resulted in favorable



No. at risk	0	60	120
Stage I	18	12 (66.7%)	12 (66.7%)
Stage II	11	3 (27.3%)	3 (27.3%)
Stage III	94	6 (6.4%)	5 (5.3%)
Stage IV	10	3 (30.0%)	1 (10.0%)
All	133	24 (18.0%)	21 (15.8%)

Figure 5. Kaplan–Meier estimates of the overall survival curve in 133 patients with anaplastic carcinoma in an ovarian tumor stratified by the International Federation of Gynecology and Obstetrics stage (four groups). Stage II vs. I, $P = 0.013$; stage III vs. I, $P < 0.001$; stage IV vs. I, $P = 0.003$; stage III vs. II, $P = 0.055$; stage IV vs. II, $P = 0.475$; and stage IV vs. III, $P = 0.183$.

clinical outcomes. Among these seven patients, five survived without evidence of disease and two died of the disease (Table 2). In the present ACOT case, platinum-based chemotherapy was performed. To date, the current patient is still alive without relapse. Because the therapeutic regimens were not available for most cases in the previous reports, more investigation on treatment for ACOT is required.

We also analyzed the survival data of ACOT cases from the SEER database (Figure 5). Statistical analysis was carried out using IBM SPSS version 22.0 (IBM Corporation, Armonk, NY, USA). We found that patients with ACOT in stage I had a high rate of overall survival. This finding is consistent with a previous report[5] in which patients without metastasis or infiltration beyond the ovaries showed a favorable prognosis. Provenza et al.[3] also found that unruptured stage I cases had a better prognosis than cases at other stages.

Conclusion

We report a patient with ACOT and FIGO stage IIIa. Our findings suggest that these patients would benefit from surgery and adjuvant chemotherapy of paclitaxel and carboplatin. This is the first study to investigate the clinical feathers and survival rate of patients with ACOT using the SEER database. Using data from a literature review, effective treatments for ACOT were also summarized. Our research

findings may help patients and gynecologists to make informed decisions for treatment of patients with ACOT.

Ethics statement

The clinical data were approved by the Ethics Committee of the First Affiliated Hospital of Sun Yetsen University (ethics approval no. 308-2016-03-01). Consent for treatment was obtained from the patient and written informed consent for publication was signed by the patient. Part of the original data of our study were provided by the SEER database.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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