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Case report

Retroperitoneal cystoadenocarcinoma: a case report with a literature review

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Summary. Primary retroperitoneal mucinous cystoadenocarcinoma (PRMC) is an extremely rare clinical entity with about 50 cases described by the literature. Given the rarity of this pathology, the sharing of accurate available informations is important to improve its knowledge. We reported a case of a woman diagnosed with PRMC who received different lines of chemotherapy and radiotherapy and we also performed a review of the literature on the issue. (www.actabiomedica.it)

Key words: retroperitoneal cystoadenocarcinoma; chemotherapy; radiotherapy

Introduction

Primary retroperitoneal mucinous cystoadenocarcinoma (PRMC) is an extremely rare clinical entity. Until 2013 only about 50 cases were reported by the literature (1,2), five of whom were male patients (3-7). Given the rarity of these tumors, it is hard to determine their histogenesis and preoperative diagnosis of these lesions is often difficult because radiological imaging is not always adequate to distinguish their exact origin and nature (8).

The best treatment for this tumor is still controversial, although total resection without rupture and careful investigation of possible primitive origins during surgery seems to be the best strategy.

We report a case of a 69-years-old woman, diagnosed with PMRC with lymph-nodes involvement and spontaneous capsular rupture, who underwent surgery and subsequent systemic and local treatment. We also reviewed the available literature about this rare disease: all published papers were obtained from the Pubmed database, using the subsequent MeSH (Medical Subject Heading) terms: "cystoadenocarcinoma", "retroperitoneal tumours" and "malignant cystic retroperitoneal tumours".

Case report

The patient was a 69 years old woman, never smoker, underwent ultrasound (US) examination on March 8th 2012 for persistent and worsening abdominal pain. The exam showed two periovarian masses: one of 14x9 cm in the right upper abdominal quadrant and another of 11x8 cm in the left lumbar abdominal quadrant; both these lesions had cystic features, with thick walls and internal nodules.

The medical history of the patient was positive for fibromyalgia, spinal disc herniation, surgical removal of pituitary gland for adenoma, hyperprolactinemia, pleuritis, essential hypertension, type II diabetes mellitus, acute diverticulitis, radical hysteroannessiectomy in September 1996 for endometrial G2 adenocarcinoma (Stage Ib FIGO classification), without relapse. The patient was also suffering from a neurological syndrome characterized by ataxia, difficulty in walk-

ing, balance impairment and sensory perypheral neuropathy.

US was followed by a CT scan which evidenced a single large cystic retroperitoneal lesion of 19x12x10 cm in correspondence of the mesogastric region (Fig. 1), with compression and dislocation of the bowel and of the inferior vena cava; peritoneal effusion and two peritoneal lesions suspected to be metastatic sites were also present. Abdominal fine needle aspiration (FNA) was performed with cytological diagnosis of epithelial cystic low grade lesion, cystoadenoma-like. Neoplastic markers were significantly increased with CA19-9 of 796 U/ml (r.v. 0-35 U/ml) and CA125 of 253 U/ml (r.v. 0-35 U/ml).

On March 23th 2012 the patient reported rapidly progressive abdominal pain, so she underwent urgent surgery with resection of the large cystic lesion which was adherent to the transverse colon, showed a rupture and the presence of a relevant quantity of hemorrhagic fluid in the abdominal cavity. A right hemicolectomy extended to transverse colon with terminal ileo-colic anastomosis was performed; the peritoneal lesions previously revealed by the CT scan were not found. The histological examination showed a cystic, unilocular mass of 17x11x4.5 cm, with thick fibrous-connective wall, filled with necrotic-hemorragic material; the final microscopic examination was positive for mullerian type retroperitoneal cistoadenocarcinoma, with moderate differentiation (G2), mucinous and endometrioid pattern, signs of cystic wall infiltration up to the perivisceral tissues (Fig. 2). There was no infiltration of surgical margins. Lymph nodes involvement was present in 8 of 45 resected nodes (Stage IIIC FIGO, considering the disease as an ovarian cancer according with the NCCN guidelines). Immunohistochemical (IHC) examination showed CK7 positivity, weak expression of p53, CK20 and WT1 negativity and

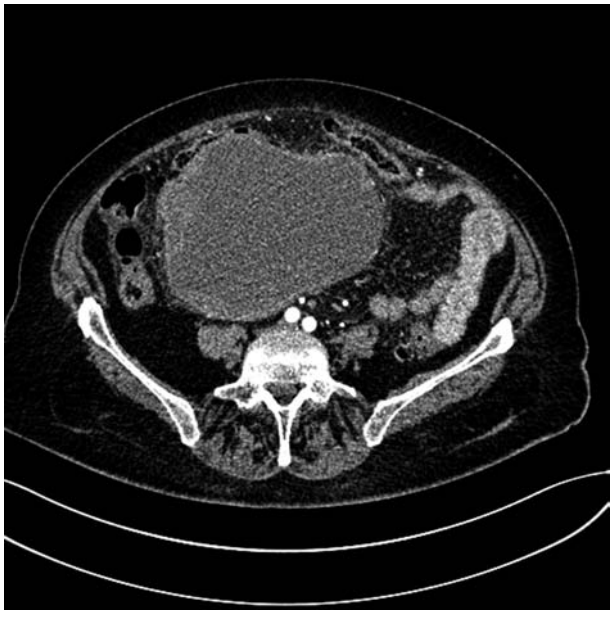

Figure 1. The CT scan evidenced a single large cystic retroperitoneal lesion of 19x12x10 cm in correspondence of the mesogastric region, with compression and dislocation of the bowel and of the inferior vena cava; peritoneal effusion and two peritoneal lesions suspected to be metastatic sites were also present (peritoneal carcinomatosis)

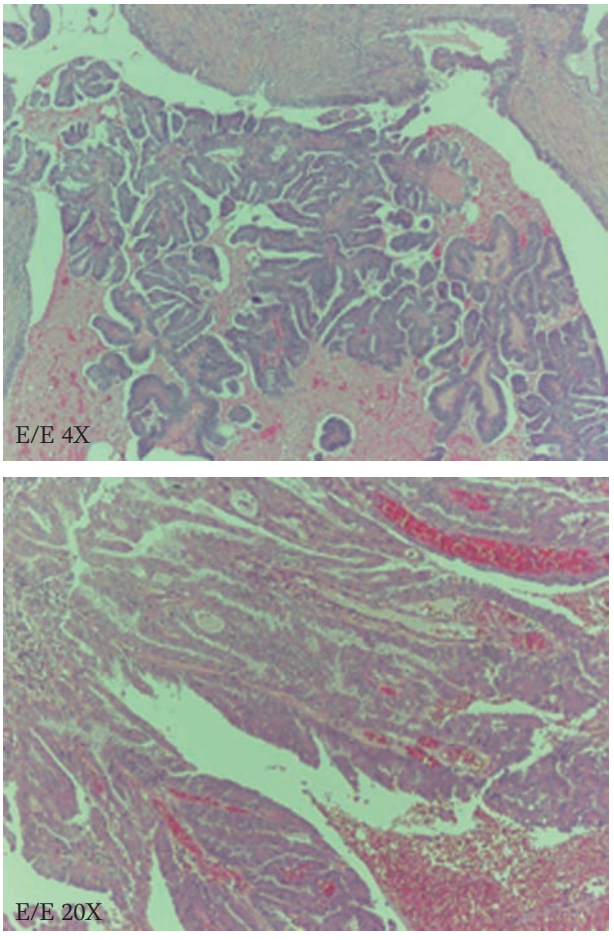

Figure 2. Microscopically: Tubule-like structures with papillae in orderly arrangement covered by stratified epithelium with mild to moderate atypia with few mitoses and abundant eosinophilic cytoplasm (E/E=ematossilina-eosin coloring)

moderate expression of estrogen and progesterone receptors. Neoplastic markers values after surgical intervention fell down (CA 19-9= 25 U/ml and CA 125 = 13 U/ml). The CT-scan performed in May 2012 was negative for disease localizations.

After a multidisciplinary discussion, in consideration of the similarity of this tumor to mullerian tumors, of its rupture into the peritoneal cavity, of the lymph nodes involvement and of the suspicion of peritoneal carcinomatosis at the radiologic revision, basing on the few data from previous case reports and reviews (see below), an adjuvant six cycles chemotherapy with Carboplatin AUC 5 and Paclitaxel 175 mg/mq with prophylactic granulocyte colony-stimulating factors was proposed to the patient. From 22/05/2012 to 24/07/2012, 4 total courses of chemotherapy were administered, with the omission of the last two courses for the worsening of the preexisting neurological syndrome. A new CT-scan was performed at the end of treatment with no evidence of disease; CA19.9 and CA125 values were within the normal range.

On February 19th, 2013 a new CT scan showed a relapse of disease with a new peripancreatic lesion of 2 cm and the increase of 2 pulmonary micronodules. The value of CA19-9 was a little increased (46 UI/ml) while CA 125 was still normal. On the basis of the tumor similarity with ovarian cancer, a new chemotherapy with liposomal Peg-Doxorubicin was started at the dose of 30 mg/m^2 on March 13th 2013; 5 courses were totally administered. The CT scan after 3 courses of chemotherapy showed a stable disease, with a little increase of the neoplastic markers value. The patient continued chemotherapy with liposomal Peg-Doxorubicin at the same doses. A new CT scan at the end of treatment showed stable disease. A total body PET/CT scan was then performed, evidencing a single peripancreatic uptake of ^{18}FDG. Surgical revaluation or stereotactic radiotherapy were both proposed to the patient and she chose the latter. Therefore, from December 6th to December 16th, 2013 stereotactic radiotherapy was administered to the pancreatic lesion. At the end of treatment markers values were decreased to CA19-9= 38 U/ml and CA125=15.37 U/ml. The patient continued the follow up.

On February 20th, 2014 the CT scan evidenced the presence of 3 new lymph nodes near the little gastric curve, suspected for metastasis, while the peripancreatic lesion was stable. Another PET/CT scan was performed, with finding of metabolic activity in perihepatic lymph nodes, involvement of both hepatic lobes and negativity of the peripancreatic lesion and the perigastric lymph node stations. Considering the discordance between CT scan and PET/CT findings, a magnetic resonance (MR) was performed: the exam confirmed the liver and lymph nodes disease progression. Neoplastic markers values showed a little decrease of CA 19-9 and a stability of CA 125. The table 1 describes the marker's trend before and after the treatments.

Considering the disease free survival of the patient after adjuvant therapy, longer than 6 months, chemotherapy with Carboplatin was administered until July 2014, when the CT scan documented a nodal and hepatic disease progression. A chemotherapy regimen with Gemcitabine was therefore initiated but suddenly discontinued after one cycle for grade 4 haematological toxicity.

In August 2014, given the clinical progression of disease, the patient started a therapy with megestrol acetate in order to obtain a possible therapeutic effect (in view of the moderate IHC expression for estrogen and progesterone receptors) over and above the effect against anorexia and neoplastic cachexia.

Discussion

Adjuvant chemotherapy

Due to the rarity of this tumor there is no evidence of benefit from surgery and chemotherapy as first choice of treatment after diagnosis of PRMC. Nevertheless, as reported by the literature, different chemotherapies were attempted (Tab. 2).

In adjuvant setting, Andres Anibal Roma and Anais Malpica (9) described two cases treated with adjuvant chemotherapy after radical surgery; one of these had a component of high-grade sarcoma and it was treated with doxorubicin and ifosfamide, but the disease relapsed after only 6 months and the death of the patient occurred 9 months after the diagnosis. The second patient had an histologic diagnosis of mucinous carcinoma with areas of sarcomatoid carcinoma;

he received 3 adjuvant cycles of ifosfamide and Cisplatin; the disease relapsed and a new chemotherapy with Paclitaxel was performed; the patient was still alive at 26 months of follow up, with hepatic metastatic disease. In another report (1) a patient with PRMC, showing an area of infiltrating adenocarcinoma associated with areas of dedifferentiation in desmoplastic stroma, was treated with 6 courses of adjuvant chemotherapy with Carboplatin AUC 6 and Paclitaxel 175 mg/m^2, but disease relapsed 8 months after the end of chemotherapy.

Tjalma and Vaneerdeweg (10) described a case of PRMC treated with 4 cycles of Carboplatin after radical surgery, but this patient had a recurrence of disease after 8 months and he died 31 months after the initial diagnosis.

Sun Ah Lee et al. (11) described a case with PRMC with sarcomatoid component which was treated with adjuvant Cyclophosphamide 600 mg/m^2 and Cisplatin 60 mg/m^2 for 6 courses. The patient was still alive after 42 months of follow up, without suspicion of disease recurrence at the CT-scan.

Tenti et al. (12) treated a patient with Cisplatin and Cyclophosphamide in adjuvant setting, with no recurrence of disease after 33 months since the diagnosis. In contrast, Ulbright et al. (13) described a carcinoma of the retroperitoneum with serous papillary characteristics which was treated with doxorubicin, cyclophosphamide and cisplatin; a second look surgery showed microscopic residual disease and the patient was treated with radiotherapy. Gotoh et al. (14) described a case of infiltrating PRMC treated with Tegafur-Uracil after surgery: the patient developed metastasis only 3 months after surgery. Dierickx et al. (15) treated a cystadenocarcinoma with a sarcoma-like mural nodule with adjuvant chemotherapy composed by 6 courses of Carboplatin AUC 7 in monotherapy every 4 weeks (follow up time not known). Ayaka Yura et al. (16) treated a retroperitoneal serous adenocarcinoma with Paclitaxel 175 mg/m^2 and Carboplatin AUC 6, with recurrence of disease 18 months after surgery.

In our case report the patient was treated as suspected metastatic disease without parameters with a Carboplatin and Paclitaxel regimen. She relapsed 7 months after the end of this treatment; only 4 courses were administered of the 6 initially planned. Actually it isn't possible to identify an adjuvant chemotherapy which seems to be effective in this type of tumor; more data about the treatment of this disease are needed.

Advanced/not resectable disease treatment

In the literature there are few data about the treatment of retroperitoneal cystadenocarcinomas in case of not resectable (locally advanced or metastatic) disease. A great part of the studies provide the administration of chemotherapy regimens based on Carboplatin and Taxanes (Tab. 2). In one study Tamoxifen and oral Etoposide were also used (1) without response. In our case radiotherapy and Gemcitabine were administered in the metastatic setting, with a temporary disease control. Also the last treatment with megestrol acetate showed a moderate clinical benefit.

Histopathological features

Retroperitoneal Cystadenocarcinomas are unilocular or multilocular cystic lesions composed by a

Table 1. Variation of markers during the treatments

	CA 19-9 (range 0-35 U/ml)	CA 125 (range 0-35 U/ml)
Initial value	796 U/ml	253 U/ml
After surgery	25 U/ml	13 U/ml
Pre-CT with liposomial Peg-Doxorubicin	55 U/ml	16 U/ml
After third cycle of liposomial Peg-Doxorubicin	73 U/ml	16 U/ml
Post-CT with liposomial Peg-Doxorubicin	73 U/ml	16 U/ml
Pre-RT	46 U/ml	21 U/ml
Post-RT	38,3 U/ml	15,3 U/ml
Pre-CT with Carboplatin	34,3 U/ml	20,81 U/ml

Table 2. Previous case reports of patients affected by PRMC

Authors	IHC	Tumoral markers	AC	Follow up after AC	Unresectable disease treatment	Follow up/ Follow up for MD
De Leòn DC et al. (1)	No No	CA 125 – CA 15-3 – CA 19-9 – CA 125 -	Carboplatin + Paclitaxel No	Relapse after 8 months No	Oral Etoposide; Tamoxifen No	No 6 months without relapse
Feng JF et al. (3)	No	CA 19-9 – CEA – αFP -	No	No	No	13 months without relapse
Thamboo TP et al. (4)	CK 7-20 + MUC2 + MUC5AC + MUC1 -	CA 19-9 + CEA +	No	No	No	18 months without relapse
Hrora A et al. (6)	No	CA 19-9 -	No	No	No	6 months without relapse
Shiau JP et al. (7)	No	CA 19-9 – CEA -	No	No	No	79 months without relapse
Kanayama T et al. (8)	CK 7 + CK20 – Vimentin + EMA -	CA 125 – CEA -	No	No	No	6 months without relapse
Roma AA et al. (9)	CK 7 + (6 cases) CK 20 + (4 cases) Vimentin, ER/PR + (1 case)	No	Doxorubicin + Isofosfamide (1 pt) Isofosfamide + Cisplatin (1 pt)	Relapse and death after 9 months 26 months without relapse	No	1-148 months (mean 40 mo, median 22 mo) without relapse
Tjalma WA et al. (10)	NA	NA	Carboplatin	Relapse after 8 months	No	Death after 23 months from the recurrence
Lee SA et al. (11)	CK + Vimentine – Desmine – S-100 protein -	CEA – CA 125 +	Cyclophosphamide + Cisplatin	42 months without relapse	No	No
Dierickx I et al. (15)	NA	NA	Carboplatin	NA	No	No
Iura A et al. (16)	CK 7 + ER + CK 20 -	CA 19-9 + CA 125 +	Paclitaxel + Carboplatin	Relapse after 18 months	Chemotherapy not specified	14 months with MD
Kaku M et al. (22)	No	CA 19-9 + CEA – CA 125 +	No	No	Docetaxel + Carboplatin	16 months with MD

(continued)

Table 2. Previous case reports of patients affected by PRMC

Isse K et al. (23)	EMA + Calretinin + ER/PR +	CA 19-9 +	No	No	No	No
Suzuki S et al. (24)	EMA + Vimentin -	CA 19-9 +	No	No	No	No
Uematsu T et al. (25)	No	CEA + CA 15-3 -	No	No	No	6 years without relapse
Lee WI et al. (26)	No No	CA 125 - CEA – CEA – CA 125 -	No No	No No	No No	30 months without relapse 15 months without relapse
Dore R et al. (27)	No	No	No	No	No	16 months without relapse
Youssef C et al. (28)	No	No	Cisplatin + Paclitaxel	2 years without relapse	No	No
Tamura T et al. (29)	No	No	No	No	No	13 months without relapse
Pearl ML et al. (30)	No	CEA – CA 19-9 -	No	No	No	10 months without relapse
Yadav R et al. (31)	No	No	No	No	No	One and half year without relapse
Navin P et al. (32)	CK 7 + CK 20 +	No	No	No	No	No
Mikami M et al. (33)	NA	NA	No	No	No	Death after 18 months from surgery

IHC = immunohistochemistry; AC = adjuvant chemotherapy; MD = metastatic disease; NA = not available

thin single or multiple layers of mucinous columnar epithelium of adenocarcinoma mixed with thick layer or nodules of atypical border–line cells, benign cells or malignant cells, sometimes sarcomatoid-like or with areas of adenocarcinoma with papillary and cribiform architecture. At the IHC analysis, CK7 is almost ever expressed; CK 20 is often positive, with some exceptions. Hormone receptors could be positive and this could suggest an endocrine sensitivity of the disease, but we have no data about hormonal therapy effectiveness. Sometimes markers of sarcomatoid differentiation like Vimentin, Desmin or S-100 protein could be present.

Histogenesis

The possible pathogenesis of PRMC is of interest. Although the exact origin of this tumor remains unclear, several hypotheses have been suggested to explain its histogenesis. One of the most accepted is that of ectopic supernumerary ovarian tissue (17), even though some cases in male patients have been reported and accessory and supernumerary ovaries in females are rare. Other Authors have proposed that PRMC may arise from a teratoma in which the lining mucinous epithelium has overridden all the other components to survive as the single cell component (18). Another hypothesis is that the tumor arises from peritoneal epithelium which possesses the potential for Müllerian differentiation (19). Finally, the hypothesis that has gained increasing support is that of coelomic metaplasia, in which some clusters of coelomic epithelial cells are deposited in the retroperitoneal area, developing an inclusion cyst with subsequent mucinous metaplasia (12,20,21). This last theory is also of interest because it may explain occurrences in male patients.

Although the exact origin of this neoplasm is still controversial, in the future a better and more accurate IHC evaluation will facilitate the histological determination and the establishment of histogenetic correlations of the tumor.

Prognostic factors

The rupture of cystic mass is deemed to be a negative prognostic factor for the high risk of recurrence after surgical resection; several authors affirm that if cystic rupture or extracapsular invasion occur, adjuvant chemotherapy could be recommended (11). Another prognostic factor is a pathological finding related to the histopathological features of the intramural nodules, with a worse prognosis if they are composed by more aggressive histologies with sarcoma-like or anaplastic features (9). The behavior of this neoplastic disease is very variable and dependent by the histological differentiation of the intracystic nodules and of the cystic wall.

Neoplastic markers utility

Neoplastic markers levels are usually normal in this disease, but sometimes they could be increased. In our case, CA19.9 and CA 125 values were both increased and they were consistent with the clinical course of the disease, so they were used to monitor the response to treatment.

In the literature we can find 4 cases with CA 19-9 abnormalities (16,22-24). The other neoplastic marker resulted to be altered was CA 125, reported to be increased in 4 cases (1,11,16,22). In some reports also CEA value was increased (4,25). No abnormalities of CA 15-3 and α-fetoprotein have been reported.

In conclusion, there isn't actually a marker which could be considered highly sensitive for this tumor and all the neoplastic markers considered lack of any specifity. If increased, serum markers could be a good parameter for the clinical follow-up and the evaluation of responses to therapies. CA 19-9 and CA 125 levels could be increased more frequently than others and they could be helpful for evaluating the response to treatments and for early detecting relapse after surgery (Tab. 2).

Conclusions

PRMC is a very rare neoplasm whom origin seems likely to be the coelomatic epithelium. There is no clinical neither radiological, histopathological, laboratoristic or pathognomonic feature which could help the diagnosis of this tumor.

Its behavior is variable and it could depend on the differentiation of tissues which can develop inside the cysts.

Radical surgery, whether possible, is actually the first choice of treatment. The few data concerning adjuvant therapy after surgery didn't show a reduction of risk of relapse and the role of this approach remains uncertain.

In local advanced or metastatic disease no evidence of benefit from chemotherapy is still demostrated, so we can conclude that PRMC seems to have a poor chemosensitivity.

We didn't found data about the effectiveness of radiotherapy; in our case radiotherapy didn't show a clear benefit with a progression of disease only three months after treatment.

Further data about this tumor are needed to improve treatment and survival of the affected patients.

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