Omental metastasis with malignant ascites: an unusual manifestation of prostatic adenocarcinoma

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Abstract

Omental metastasis with malignant ascites from prostatic adenocarcinoma is rare. This case report is about a patient who presented with a 24-hour history of a swollen right leg. Clinical examination revealed a hard prostate and blood biochemistry demonstrated an elevated prostate specific antigen level. A Doppler ultrasound scan excluded deep venous thrombosis, but a CT scan of the abdomen revealed marked para-aortic lymphadenopathy and prostate gland biopsy confirmed prostatic adenocarcinoma. The patient was treated with goserelin. Three years later, he presented with ascites and an omental mass. Histology of the omental mass showed metastasis from the prostatic adenocarcinoma. He was treated with second-line hormonal therapy but died after 4 months. We discuss the clinical progression, with a review of the literature.

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Introduction

Every year in the United Kingdom nearly 32 000 cases of prostate cancer are diagnosed. Prostate cancer causes more than 10 000 deaths in the United Kingdom each year.[1] Prostate cancer has a tendency to metastasize to bones and lymph nodes. It rarely gives rise to intra-abdominal secondary cancer.[2] We report a case of omental metastasis and malignant ascites secondary to the prostate cancer, which is a rare manifestation of this disease. There is only one previous report of a case of prostate cancer that metastasized to the omentum and was accompanied by gross ascites.[3]

Case Report

A 75-year-old man presented acutely to the accident and emergency department with a 24-hour history of a swollen right leg. His past medical history was unremarkable. The right leg was markedly swollen, with pitting edema up to the thigh. On digital rectal examination, the patient was found to have a malignant feeling prostate. Routine blood tests were unremarkable. Prostate specific antigen (PSA) was elevated at 92.9 ng/mL. A Doppler ultrasound of his leg showed no evidence of a deep venous thrombosis. An abdominal ultrasound revealed extensive para-aortic lymphadenopathy and right hydronephrosis. CT scan confirmed extensive retroperitoneal lymphadenopathy involving mainly right para-caval and inter-aortocaval nodes, markedly compressing the inferior vena cava and causing right hydronephrosis. A right percutaneous nephrostomy was performed followed by the insertion of an antegrade JJ stent to manage right renal obstruction because an attempt at retrograde stenting was unsuccessful. A possible diagnosis of prostate cancer was made, which was subsequently confirmed by transrectal ultrasound guided prostate biopsy that revealed prostatic adenocarcinoma with a Gleason sum score of 9. The patient was started on hormone therapy with goserelin.

Two months after starting treatment, the patient's leg edema had resolved. A repeat CT scan at that time confirmed that the right hydronephrosis had resolved. His PSA had dropped to 5.4 ng/mL. The JJ stent was removed. A further CT scan 12 months later revealed almost complete resolution of abdominal and pelvic adenopathy. His PSA had dropped to 0.2 ng/mL at 12-month follow-up.

Three years after the patient's initial presentation, he was readmitted with a 10-day history of abdominal bloating but had no other symptoms. On examination, he had gross ascites. His PSA had risen to 10.3 ng/mL. Abdominal ultrasound confirmed a large volume of ascites with no focal liver lesions. There was a 7 × 9 cm irregular mass in the upper abdomen. Abdominal paracentesis was performed, with a large volume of hemorrhagic fluid aspirated. Cytological examination of the fluid revealed areas of atypical epithelial cells, which were suspicious of neoplasia. Reactive mesothelial cells were also seen. CT scan confirmed ascites with multiple nodular mass lesions within the omentum and mesentery, the largest in the omentum measuring 9 × 9 cm (Fig. 1). A CT-guided biopsy of the omental mass was performed and histology of the omentum biopsy showed fibro-fatty omental tissue infiltrated by metastatic cribriform carcinoma consistent with secondary prostate cancer (Fig. 2). The patient was started on diethylstilboestrol (1 mg/d) and ASA (75 mg/d) as a

second-line therapy. However, he developed recurrent ascites and required multiple admissions for therapeutic paracentesis and packed cell transfusions for anemia. The patient died 4 months after his first presentation with ascites.

Discussion

Malignant effusions may occur in the pleural or peritoneal cavities, but they are very uncommon manifestations of prostate carcinoma.[2] Although these effusions may constitute the initial presenting feature of prostate cancer, they may be the only sign of recurrence of prostatic disease.[4–6] Ten cases of malignant ascites secondary to prostatic carcinoma have been reported in the literature (Table 1). Table 1 shows that patients have a poor survival rate after presentation with ascites. Two of these had chylous ascites, while 1 was a case of hemorrhagic ascites. The mechanism of ascites may include peritoneal seedlings or lymphatic obstruction. Chylous ascites secondary to prostatic carcinoma has also been reported.[7] It has been suggested that the measurement of PSA may be a valuable adjunctive study for the diagnosis of malignant effusions in prostate cancer.[8]

Malignant effusions secondary to prostatic adenocarcinoma are mostly seen in relation to poorly differentiated prostatic adenocarcinoma and generally show poor response to hormonal manipulation, although Kehinde and colleagues reported a 76-year-old man who presented with palpable omental metastasis and gross ascites due to prostate cancer who responded well to surgical castration.[3] Unfortunately, in our patient the disease was much more aggressive.

In conclusion, omental metastasis and malignant ascites are potential complications of prostatic adenocarcinoma. This report serves to highlight that prostate cancer can present in a myriad of ways, including ascites. It should always be considered when evaluating elderly male patients presenting with ascites or pleural effusion and a history of prostatic adenocarcinoma.

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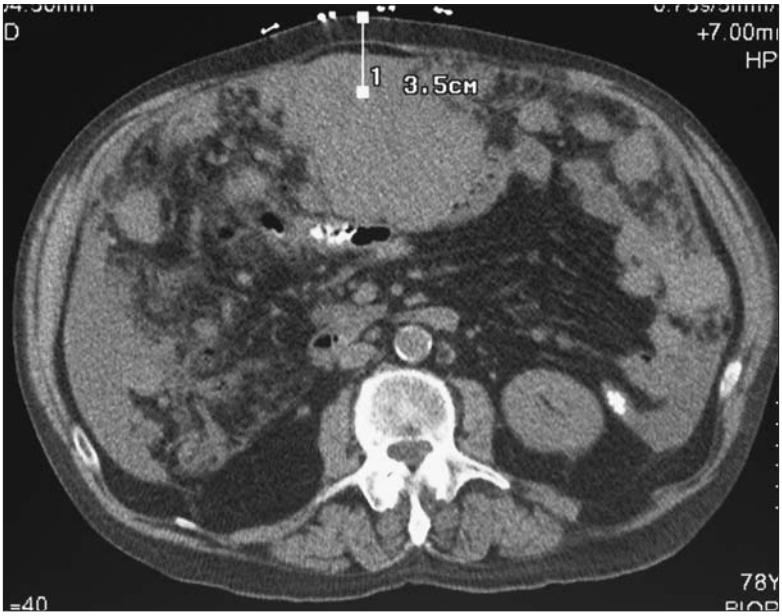

Fig. 1. CT scan showing ascites with a large nodular mass lesion within the omentum.

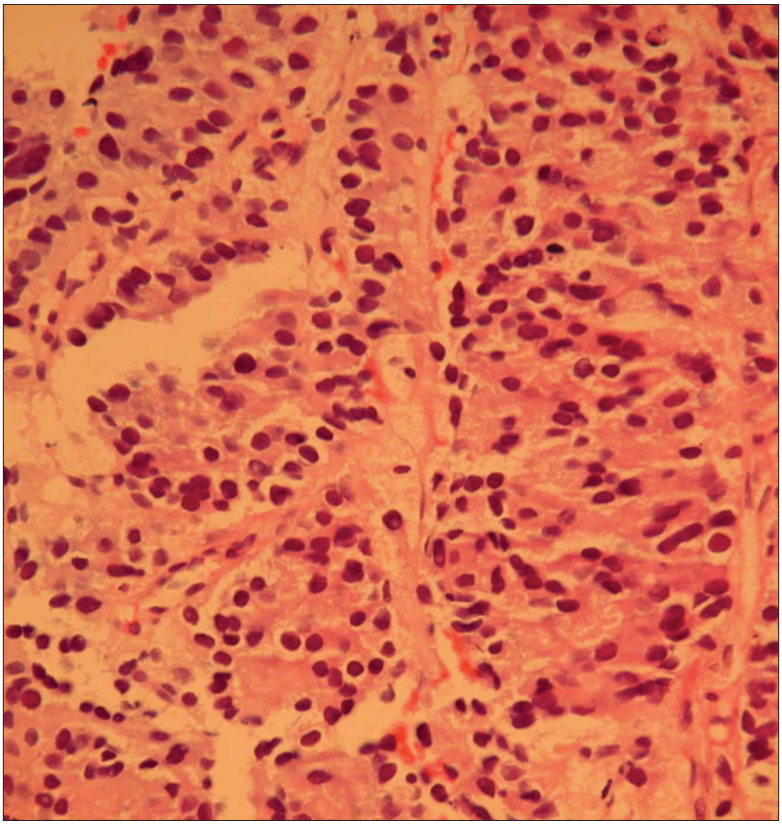

Fig. 2. Histology of the omental biopsy showing omental tissue infiltrated by metastatic cribriform carcinoma consistent with prostatic primary cancer (haematoxylin–eosin stain, original magnification 200 ×).

Table 1: Reported cases in the literature of malignant ascites in prostate cancer

Reference	Patient age, yr	Type of ascites	Primary prostate histology	Time gap between first diagnosis and ascites, yr	Other metastases	Treatment	Survival after presentation, mo
Kehinde et al[3]	76	Straw coloured	Mucin secreting, Gleason 8 adenocarcinoma	4	Omentum	Surgical castration	Alive at 18 mo
Rapoport et al[4]	76	NA	Well differentiated, mucin secreting adenocarcinoma	16	Pleural effusion, lymph nodes	Intraperitoneal 5-FU and thiotepa	3
	45	Greenish fluid	NA	1	Pleural effusion	Surgical castration	Few mo
Catton[5]	63	NA	Poorly differentiated, signet-ring variant	Initial presentation	Lymph nodes	Surgical castration	13
Saif[6]	70	NA	Gleason 9 adenocarcinoma	4	None	Maximal androgen blockade	Alive at 2 mo with progressive disease
Amin[7]	83	Chylous ascites	Adenocarcinoma (unknown grade)	5	Lymph nodes	Hormonal	4
Appalaneni[8]	60	Straw coloured	Gleason 9 adenocarcinoma	3	Bone	Palliative chemotherapy	1.5
Megalli[9]	58	Straw coloured	Adenocarcinoma (unknown grade)	Initial presentation	None	Radiotherapy, hormonal (DES)	Ascites resolved; alive at 6 mo
Beigel[10]	29	Chylous ascites	Adenocarcinoma (unknown grade)	Initial presentation	Bone	Refused	1
Tsai[11]	68	Haemorrhagic	Gleason 9 adenocarcinoma	1	Rectal wall	Hormones + interferons	4
Present case	75	Haemorrhagic	Gleason 9 adenocarcinoma	3	Omentum	Hormonal (DES)	4

NA = not available; DES = diethylstilbestrol.

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