CASE REPORT

A rare case of omental metastasis as first and singular site of failure from localized prostate cancer

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ABSTRACT

The most common sites of metastases from prostate cancer are the regional lymph nodes and bones followed by lung, liver, pleura and adrenals. We describe an unusual presentation of metastatic prostate cancer with omental metastases as the only site of disease. An otherwise well 72-year-old man was investigated for a rising prostate specific antigen (PSA) 12 months after curative-intent radiotherapy and androgen deprivation for high risk prostate cancer. CT scans showed omental caking with no evidence of bony or other metastases. Omental biopsy demonstrated adenocarcinoma of prostatic origin.

Key Words: Prostate cancer, Omental metastasis, Case report

1. INTRODUCTION

The most common sites of metastases from prostate cancer include regional lymph nodes and bone followed by lung, liver, pleura and adrenals. Isolated peritoneal metastasis from prostatic acinar adenocarcinoma (presenting as omental caking) is extremely rare. We present the case of a patient with prostate cancer with omental metastasis in the absence of nodal or skeletal involvement.

2. CASE PRESENTATION

2.1 Background

A fit and well 72-year-old man presented with a raised prostate specific antigen (PSA) of 23.5 µg/L. The only significant family history was his father being diagnosed with bowel cancer. On digital rectal examination he had evidence of bilateral prostate infiltration (clinical stage T2c). He proceeded to trans-rectal ultrasound-guided prostate biopsies which demonstrated extensive Gleason 4+3 adenocarcinoma involving 14 of 14 cores (see Figure 1). His prostate volume was 41 ml. Staging with CT of the abdomen and pelvis and Technetium-99m bone scan did not demonstrate any evidence of metastases.

2.2 Initial management

The patient was managed with curative intent using neoadjuvant and concurrent short term androgen deprivation therapy...
(ADT) followed by radical external beam radiation treatment (46 Gy in 23 daily fractions) and high dose rate brachytherapy boost (19 Gy in 2 fractions of 8.5 Gy) using an Iridium-192 afterloader technique. ADT was with leuprolide (Eligard) for a duration of 6 months.

Seven weeks following radiotherapy the PSA was 0.2 µg/L showing a good response to treatment. However, 6 months after treatment his PSA had risen to 3.2 µg/L. At this stage the patient was observed. Twelve months following treatment the PSA rose to 20 µg/L and he underwent re-staging with CT which revealed “oment al caking” (see Figure 2). Bone scan was negative for osseous metastases. His CA 19-9 was in the normal range (10 kU/L).

On account of the omental disease, the case was presented and discussed at the Upper Gastro-Intestinal Multidisciplinary Meeting and the consensus was that the omental caking was likely from a gastrointestinal malignancy. He underwent a laparotomy and omental biopsies. The operative findings were of diffuse omental metastases without evidence of ascites.

2.3 Histopathology
The morphology of the omental specimen revealed fibro-adipose tissue with tumour nodules. Microscopic features were consistent with metastatic acinar adenocarcinoma with single and fused complex glands lined by columnar epithelium with clear cell change within a desmoplastic stroma (see Figure 3).

The tumour was positive for PSA and ERG and negative for CK7, CK20, CDX2, TTF1 and GATTA3.

The tissue from the omental biopsy was compared to the original core biopsies by an anatomical pathologist experienced in genitourinary malignancy (GW) and deemed to be the same tumour.

2.4 Subsequent management
Following the diagnosis, the patient was discussed with a genito-urinary medical oncologist and recruited to the ENZAMET randomised trial. ENZAMET is comparing the effectiveness of enzalutamide versus a conventional non-steroidal anti-androgen, when combined with a lut einis ing hormone releasing hormone (LHRH) analog as first line ADT for newly diagnosed metastatic prostate cancer. He was randomised to the control arm and commenced on leuprolide (Eligard) 45 mg six-monthly and bicalutamide (Cosudex/Casodex) 50 mg daily.

At the time of writing, the patient remains well. His most recent PSA was 0.10 µg/L. Repeat CT of the abdomen and pelvis has shown a reduction in the volume of omental disease with no ascites or evidence of metastatic disease elsewhere. He continues on leuprolide and bicalutamide.

3. DISCUSSION
The most common sites of metastatic spread of prostate cancer are the lymph nodes and the skeleton. To the best of our knowledge, an isolated presentation of omental caking alone as first and only site of dissemination from prostate cancer, in the absence of ascites, has never previously been reported.

In a recent review, 16 cases of prostate cancer presenting with ascites were described, 7 of which did not have evidence of disease beyond the peritoneum. Brehmer et al described a case of isolated peritoneal carcinomatosis at the time of pelvic lymphadenectomy for what was thought to be a localised prostate cancer. The other cases were all associated with ascites. There has been one case presented in
2002 of mucin-producing adenocarcinoma metastasising to omentum, however this was associated with gross ascites.\textsuperscript{[6]} The patient responded well to surgical castration followed by hormonal manipulation. A second case report reported a patient with hormone refractory prostate cancer with peritoneal metastasis, again accompanied by ascites, but without bony metastases, who showed an excellent response to docetaxel-based chemotherapy.\textsuperscript{[7]} The only other case report of prostate cancer metastasising with extensive caking of the omentum, in which the disease caused a bowel obstruction, was in the setting of neuroendocrine differentiation.\textsuperscript{[8]}

This is therefore the only reported case of prostatic acinar adenocarcinoma causing omental caking as the initial and only site of metastasis.

**CONFLICTS OF INTEREST DISCLOSURE**

The authors declare no conflicts of interest.

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