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Isolated non-ascitic peritoneal carcinomatosis after robotic radical prostatectomy for prostate cancer: A case report

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ABSTRACT

Oncologic recurrence can occur after Robot-Assisted Radical Prostatectomy. Prostate cancer metastasizes often in bones, however the peritoneum is infrequently targeted. Even more, peritoneal dissemination without any other organ involved especially the bones is very rare, only few cases are reported. Through the available literature we discuss about the presumed seeding theory leading to this atypical location for prostatic metastases. Here we report a case of isolated non-ascitic prostatic peritoneal metastases most probably due to iatrogenic spillage during surgery.

Introduction

Isolated non-ascitic peritoneal metastases after radical prostatectomy are very rare.[1] Few cases in the literature report this atypical location of metastases without other locations involved.[2] Several hypotheses on the mechanisms of dissemination and treatment of this affection are discussed through literature.[1–3] We report a case of possible tumor seeding after robot-assisted radical prostatectomy.

Case report

A 66-year-old man presented with elevated prostate-specific antigen (PSA) of 12.7ng/ml in 2013. Previously his PSA was measured at 6.42ng/ml and 9.29ng/ml in 2007 and 2010 respectively without any further clinical examination or imaging. In 2013, induration of the right lobe of the prostate was palpated at rectal examination and transrectal biopsy revealed Gleason 7 (4 + 3) prostatic adenocarcinoma. Bone scan and computerized tomography scan (CT-scan) of the abdomen and pelvis were negative for metastases. A magnetic resonance imaging (MRI) of the prostate revealed 2 lesions (16*21*15mm and 21*21*12mm) without extracapsular extension or seminal vesicle invasion.

The patient underwent robotic radical prostatectomy with lymph node dissection in late September 2013. The pathology report revealed a pT3aN0 Gleason 7 [(4 + 3) + tertiary pattern of 5] prostatic adenocarcinoma with negative margins and six resected lymph nodes did not show signs of malignity. His post-operative PSA nadir was <0.03 ng/ml and remained undetectable until August 2014 when it reached 0.048ng/ml. A close follow-up showed a PSA increase to 0.073ng/ml. Thus, a salvage radiotherapy (66 Gy on the prostatic bed) was performed in December 2014 with a valid biological response, showing PSA levels decreasing to 0.042 ng/ml in April 2015.

The patient then experienced a second biochemical failure with a rapid increase of his PSA reaching 0.37ng/ml in May 2016 with a PSA doubling time (PSA-DT) of 3.5 months. In June 2016 a chest/abdomen/pelvis CT scan showed iliac lymphadenopathy and multiple peritoneal nodules in the abdomen. An 18-Fluorocholine Positron Emission Tomography (18-F-choline PET-CT) confirmed peritoneal carcinomatosis associated with right iliac lymphadenopathy. By that time PSA reached 1.72ng/ml and rised to 2.4ng/ml in January 2017. He was subsequently treated with intermittent injections of Degarelix (FIRMAGON©, Ferring), with a consequent decrease of PSA to undetectable levels and testosterone to castrate levels.

PSA remained undetectable until May 2018, when it rose to 0.15ng/ml with a PSA-DT of 4 months. Dosage of testosterone confirmed castrate levels.

In September 2018, a PET prostate specific membrane antigen (PET-PSMA) showed multiple peritoneal nodules and a right iliac lymph node

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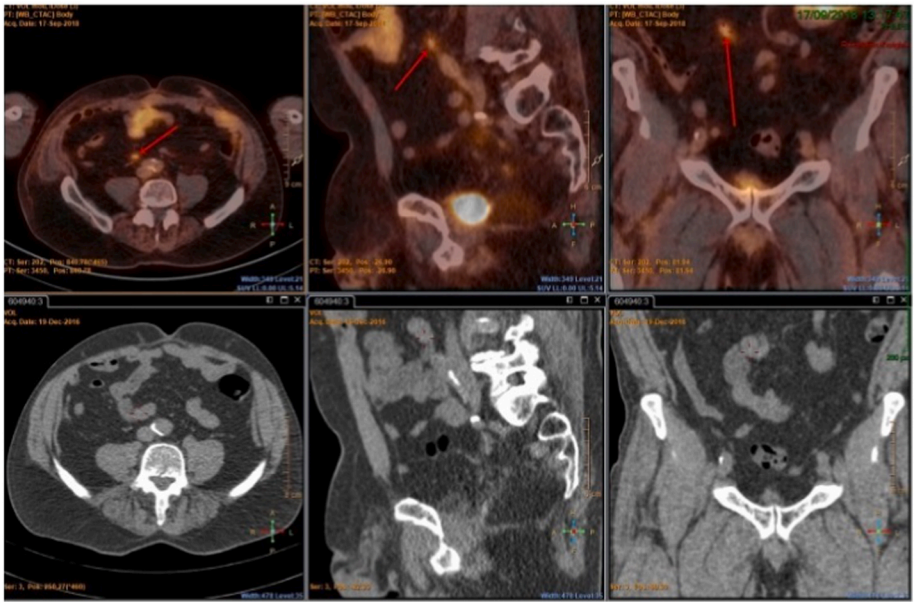

Fig. 1. PET-PSMA in November 2018: the red arrows showing peritoneal carcinomatosis nodules. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

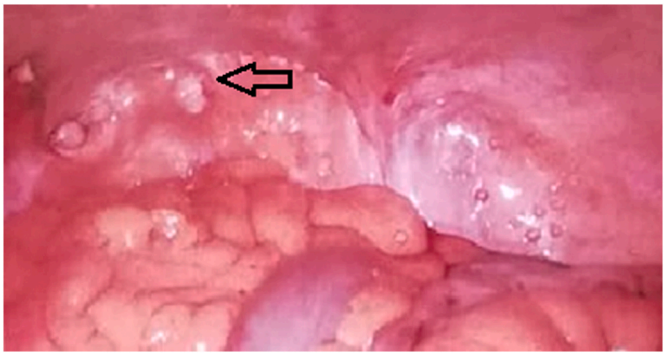

Fig. 2. Image of the laparoscopic exploration (March 2019), the black arrow showing peritoneal carcinomatosis.

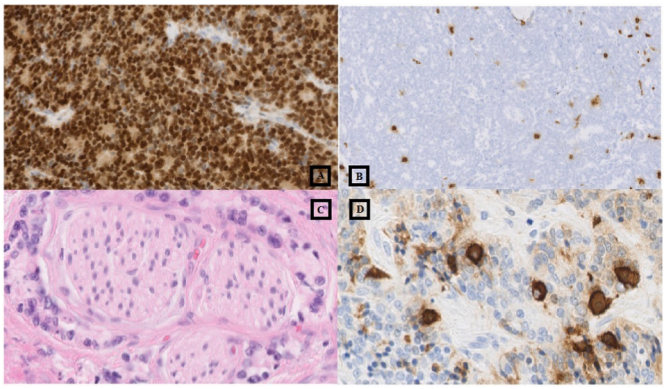

Fig. 3. Anatomopathological pictures of the peritoneal nodules (A: NKX3.1 marker; B: Chromogranine marker) and of the primitive prostatic adenocarcinoma (C: hematoxylin and eosin stain; D: Chromogranine marker).

hyperexpressing PSMA, without a significant modification compared to anterior imaging (Fig. 1).

In February 2019, Bone scan, CT scan of the abdomen/pelvis and rectal examination were negative. Back then, PSA reached 2.1ng/ml and laparoscopic exploration was performed (Fig. 2) confirming multiple metastatic peritoneal implants. The pathology report revealed a poorly differentiated prostatic adenocarcinoma positive for NKX3.1 (confirming prostatic origin) and in favor of a neuroendocrine differentiation (*anti*-chromogranin antigen, *anti*-synaptophysin antigen and CD56 were positive locally). At that time, Neuron specific enolase was dosed at 21.8 ng/ml and we performed new immunohistochemistry on the primitive neoplasm which was locally positive for neuroendocrine differentiation (5% of tumoral tissue) (Fig. 3).

Treatment with Abiraterone Acetate was initiated, and the patient exhibited an early biological response and is currently in complete biological response.

Discussion

Prostate adenocarcinoma metastasizes in 35% of cases after radical prostatectomy, the bones being the most targeted organ (90%).[4] It is known that it can metastasizes elsewhere as in the lung, the liver or even in the peritoneum.[4] Nonetheless, peritoneal dissemination without any other organ involved especially the bone is very rare. In fact, only 7 cases of histologically-proven peritoneal metastases following laparoscopic surgery, whether robot-assisted or not, are reported in the literature in the absence of other metastatic lesions.[1] Even more, only 5 cases are reported without ascites being present at the time of diagnosis.[1] Our case is thereby singular, as the diagnostic of peritoneal metastases 36 months post-operative are not associated to ascites or bone lesions.

Different mechanisms have been described: the iatrogenic and the known hematogenous and lymphatic pathways. The first one convicts the mini-invasive surgical approach and the assumed seeding of tumoral cells during surgery by different ways (pneumoperitoneum; gross spilling; positive margin etc …).[1]Pneumoperitoneum is often considered as a potential factor in spreading tumoral cells. A lot of its aspects are discussed: “the chimney effect” of insuflation-dessuflation; the modification of peritoneal cavity with the decrease in peritoneal pH due to carbon dioxide; the microenvironment of high cellular proliferation created locally by trocar insertion and the immunosuppressive effect of CO_2. All these factors could promote the theory by which cells adheres to omentum or port site and then disseminates across peritoneum.[4,5]

It is also important to note that 9 cases of isolated peritoneal carcinomatosis without any surgery have been described in the available

literature. They presented generally with high PSA, high Gleason score (≥8) and poorly differentiated adenocarcinoma associated with neuroendocrine differentiation.[1] Based on these findings, isolated peritoneal metastases can occur in some aggressive prostate cancer.

In the present case, surgical margins were negative, there was no evidence of macroscopic spillage and no involvement of dissected lymphatic nodes. In the light of all these elements, one could speculate that in the presence of only peritoneal metastases, an iatrogenic seeding theory must be taken in account.[2]

Our patient responded to ADT in 2017 with an undetectable PSA for 1 year and a slight regression of peritoneal nodules confirmed on imaging. This is in favor that in hormone-dependent cases of solitary prostatic peritoneal metastases, ADT could be privileged.[3] One can note that there is a trend to relative resistance to ADT in isolated peritoneal carcinomatosis compared to only « port metastasis ».[1]

In castrate-resistant prostate cancer with peritoneal metastases, several treatments have been tested: chemotherapeutic agents (Docetaxel/Cabazitaxel), corticosteroids, Arbiraterone acetate or even Enzalutamide.[2,3] In our patient even with a neuroendocrine differentiation, the combination of Abiraterone with corticosteroids has shown earlier good results.

Conclusion

We herein report a case of isolated non-ascitic prostatic peritoneal metastases. An iatrogenic seeding theory seems to be the most probable although its mechanism is still not understood.

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Declaration of competing interest

None.

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