Isolated peritoneal carcinomatosis from metastatic castration-resistant prostate cancer and associated biliary obstruction: A case report

Félix Couture, Joshua Chin, Jaron Chong, Simon Tanguay

Introduction

Prostate cancer is known to metastasize more frequently to sites such as bone, lymph nodes, and lung, with peritoneal involvement being found in up to 7.0% of cases in large autopsy series. The discovery of peritoneal carcinomatosis in patients living with prostate cancer remains rare, especially in the absence of any other solid organ or bone metastases, with malignant ascites being present in almost all reported cases. We describe what is, to our knowledge, the first reported case of non-ascitic peritoneal carcinomatosis from metastatic castration-resistant prostate cancer (MCRPC) with associated biliary obstruction.

Case presentation

The patient is a 74 year-old male who underwent laparoscopic radical prostatectomy 14 years ago for a Gleason 5 (2 + 3) prostate cancer on biopsy, with a pre-operative PSA of 4.0 ng/mL. The pathology revealed a pT2a Gleason 7 (3 + 4) adenocarcinoma with negative margins. His post-operative nadir PSA was < 0.1 ng/mL.

He experienced a biochemical failure and his PSA increased slowly, until it reached 0.55 ng/mL eight years after surgery, at which point he received salvage radiotherapy. Bone scan and CT scan of the abdomen and pelvis were negative for metastases. His PSA post radiation therapy decreased to 0.02 ng/mL, but later increased again.

He was followed closely, until his PSA reached 7.21 ng/mL two years after radiotherapy, with repeat imaging being again free of metastases. He was subsequently treated with intermittent hormonal ablation with GnRH antagonist injections for 10 months, which caused his PSA to drop to 0.14 ng/mL. Termination of treatment caused his PSA to rise to 12.03 ng/mL. It decreased again with a second 10-month course of Docetaxel injections every 3 weeks, as well as regular oral prednisone.

A year later, while still on androgen-deprivation therapy (ADT), his PSA spiked to 40.59 ng/mL. Bone scan was again negative for metastases, but CT scan of the abdomen revealed new peritoneal nodules along the perisplenic, subdiaphragmatic surface, with significant omental caking, consistent with carcinomatosis (Fig. 1). The abdomen was free of any ascites. Other structures, including the liver, all solid organs, and bones, were free of suspicious lesions. The prostatic bed did not show any local recurrence. Ultrasound-guided biopsy of the omental caking revealed poorly differentiated prostate adenocarcinoma, consistent with metastatic disease (Fig. 2).

One month later, the patient presented to the emergency room with weakness and icterus, and was found to have a bilirubin of 408.3 μmol/L. MRI revealed biliary obstruction at the hepatic hilum caused by a 2-centimeter mass from the omental caking representing metastatic involvement of the porta hepatis (Fig. 3). The liver was still free of any parenchymal lesion. He underwent ERCP stenting, which led to an immediate decrease of his bilirubin.

 Upon normalization of his liver function, he was started on Docetaxel injections every 3 weeks, as well as regular oral prednisone. His PSA responded immediately, decreasing from 102.9 ng/mL to 48.9 ng/mL. A new CT scan, done 5 months after initial imaging, revealed a minimal increase of the carcinomatosis.

Discussion

Spreading of prostatic metastases within the peritoneum and omentum in the absence of metastases to the bones or solid organs has been reported a few times through the available literature. All castration-resistant cases presented with malignant ascites, with all non-ascitic cases being responsive to castration.

In our case, the patient underwent laparoscopic radical prostatectomy 14 years ago. Iatrogenic surgical spillage has been proposed as a potential etiology of peritoneal dissemination in those cases, given the lack of involvement of other organs, which challenges the traditional lympho-vascular route of metastatic disease. This hypothesis has led...
some surgeons to stress the importance of meticulous procedures to prevent iatrogenic seeding. Excessive specimen manipulations, poor surgical technique, and carbon dioxide insufflation have all been thought to promote peritoneal and port-site seeding.2–4

Management of isolated peritoneal dissemination of prostate cancer remains poorly understood. In hormone-dependent cases, initiation of ADT or surgical castration has led to a decrease in the extent of carcinomatosis and in PSA. In reviewed MCRPC cases, chemotherapeutic agents (such as Docetaxel) and corticosteroids had some impact on the extent of peritoneal disease. Also, Sheng et al.5 reported the case of a 60 year-old male who underwent total omentectomy, with a subsequent drop in PSA from 11.8 to 1.8 ng/ml. This patient then had a rise in PSA and recurrence of peritoneal nodules, which responded well to ADT.

Our patient also presented with obstructive jaundice secondary to compression from a soft tissue metastatic mass near the hepatic hilum. Biliary obstruction from metastatic prostate cancer has been reported in the literature, primarily caused by retroperitoneal lymphadenopathy or metastasis to the pancreatic head. Biliary stenting or mass reduction through medical treatment resulted in resolution of obstructive jaundice in all reported cases. No other case of obstructive jaundice secondary to castration-resistant disease was reported in the literature, or any other example of obstruction from prostatic metastases to the omentum.

This case is therefore unique in that peritoneal carcinomatosis from MCRPC presented without any malignant ascites, suggesting an early stage of this advanced disease state. Moreover, the associated biliary obstruction was a first in the literature for castration-resistant disease, as well as in the context of isolated metastatic implants to the peritoneum and omentum. Our patient will continue to be followed to monitor his response to treatment. Possible further treatment options will include other chemotherapeutic agents, secondary hormonal manipulation, or radiation therapy, all of which have a fairly unpredictable chance of success, given the lack of data.

Conclusion

This case represents the first reported instance of non-ascitic peritoneal carcinomatosis from MCRPC with associated biliary obstruction. We reviewed and discussed the current state of the literature on isolated peritoneal involvement of prostatic metastases and its management. We also discussed biliary obstruction secondary to metastatic disease, another uncommon occurrence within the available literature. Our report contributes to the knowledge on these two rare manifestations and should guide further work on the prognosis and management of such advanced disease.

Conflicts of interest

The authors have no conflicts of interest to declare.

References

1. Bubendorf L, Schopfer A, Wagner U, et al. Metastatic patterns of prostate cancer: an autopsy study of 1,589 patients. Hum Pathol. 2000;31(5):578–583.
2. Tsivian A, Sidi AA. Port site metastases in urological laparoscopic surgery. J Urol. 2003;169(4):1213–1218.
3. Lee SW, Gleason NR, Bessler M, Whelan RL. Port site tumor recurrence rates in a murine model of laparoscopic spleenectomy decreased with increased experience. Surg Endosc. 2000;14(9):805–811.
4. Kunze C, Wunsch A, Bodeker C, et al. Effect of pressure and gas type on intraabdominal, subcutaneous, and blood pH in laparoscopy. Surg Endosc. 2000;14(4):367–371.
5. Sheng J, Findley TW, Saleghi-Nejad H. Isolated non-ascitic peritoneal carcinomatosis from metastatic prostate cancer. Urol Case Rep. 2017;10:14–15.