Prostate Cancer Metastatic to the Peritoneum in a Peritoneal Dialysis Patient

Editor:

Prostate cancer is one of the leading causes of cancer death in men (1). Autopsy studies have shown that the prevalence of prostate cancer ranges from 8.2% to 34.8% in men more than 40 years of age (2). The prognosis in prostate cancer is greatly worsened by the presence of metastases, which are most commonly found in bone, lung, liver, and brain (2). Metastasis of prostate cancer to the peritoneum has been a rare finding encountered in autopsy studies (3,4). In addition, metastasis to the peritoneum in the absence of skeletal involvement is very rare in the general population; only 3 cases have been reported to date (5–7). Here, we report the first case of a patient with end-stage renal disease maintained on chronic ambulatory peritoneal dialysis (PD) with prostate cancer metastatic to the peritoneum. Almost as unique was the lack of skeletal involvement in this patient.

CASE DESCRIPTION

A 59-year-old African American man with end-stage renal disease from hypertension initiated chronic ambulatory PD (four 2.5-L exchanges of 2.5% dextrose) in February 2009. He was stable on PD until March 2010, when he developed a first episode of peritonitis from *Candida parapsilosis*. When his PD catheter was removed, asymptomatic hernias (umbilical, right inguinal) were detected. A course of oral fluconazole was administered,

and intermittent hemodialysis using a tunneled internal jugular dual-lumen catheter was begun.

In June 2010, this patient underwent elective repair of the umbilical and right inguinal hernias and placement of a new PD catheter. Routine histopathologic analysis of the resected umbilical hernia sacs demonstrated a glandular neoplasm compatible with prostatic adenocarcinoma.

A few weeks after the hernia repair surgery, the patient noted painless cloudy peritoneal effluent. Coagulase-negative *Staphylococcus* grew from the effluent, and cytology performed at the same time showed small clusters of atypical epithelioid cells with finely vacuolated cytoplasm, ovoid nuclei, and small prominent nucleoli. The cells were suspicious for prostatic adenocarcinoma. Serum concentration of prostate-specific antigen was 54.6 ng/mL.

Chronic ambulatory PD was restarted in August 2010 with a smaller dwell volume (2 L instead of 2.5 L) to prevent hernia recurrence. The patient tolerated the PD well, without impairment of small-molecule clearance or ultrafiltration, despite the peritoneal metastases. Over the course of the next 5 months, he presented three additional times with cloudy PD fluid, and the same *Staphylococcus* species was isolated. Intraperitoneal vancomycin or cefazolin was administered each time.

In October 2010, the patient underwent a 12-core transrectal ultrasound-guided prostate biopsy, which demonstrated adenocarcinoma in all cores, with 80% to 100% of the volume of each core containing cancer. Pathology staging was scored as Gleason 3+4. No lymphatic or vascular invasion was identified. Computed tomography scan of the abdomen and pelvis, performed with oral and intravenous contrast, revealed 2 lytic lesions in the iliac bones not typical of prostate metastases; they were considered to be manifestation of renal osteodystrophy. Consistently, a bone scan with ⁹⁹Tc did not reveal osseous metastases. The multidisciplinary recommendation was orchiectomy, but the patient refused treatment, and androgen deprivation therapy with leuprolide was started in April 2011. The patient died at his home in July 2011, presumably of acute coronary syndrome.

DISCUSSION

Our case is unique in two ways. First, although autopsy reports have suggested that prostate cancer metastasizes to the peritoneum in about 7% of the general population (2), the present case is the first report of prostatic metastasis to the peritoneum in a chronic PD patient. It is interesting that the diagnosis of prostate cancer was made by identification of prostate cancer tissue in umbilical hernia sacs after elective repair, and

of prostate cancer cells in peritoneal effluent during investigation of suspected bacterial peritonitis, rather than by demonstration of primary cancer in the prostate gland. Second, prostate cancer was detected in the prostate gland, in the peritoneal lining, and in the peritoneal fluid, but not in bone. In the general population, 3 cases of prostate cancer metastatic to the peritoneum, but not to the skeleton, have been reported. In each case, the diagnosis of prostate cancer in the prostate gland had been documented before discovery of the peritoneal metastasis (6,7).

In our case, an episode of fungal peritonitis occurred 3 months before the prostate adenocarcinoma in the peritoneum was discovered. Fungal peritonitis is responsible for 2% – 15% of cases of infectious peritonitis in chronic PD patients. Usually, fungal peritonitis occurs after the end of treatment with antibiotics for bacterial peritonitis, suggesting that alteration of the colonic flora by antibacterial treatment allows fungi to proliferate and consequently migrate across the bowel wall into the peritoneal space. In our case, antibiotic treatment did not precede the fungal peritonitis. We wonder if asymptomatic disruption of the peritoneal lining by prostate cancer metastases (undetected at the time) allowed fungi in their usual colonizing density to transmigrate from the colonic lumen to the peritoneal space. Lowered immunity from metastatic disease might also have predisposed our patient to *Candida* peritonitis. More intriguing is the possibility that fungi migrated with the cancer cells from the prostate tissue to the peritoneum. Fungal prostatitis can coexist with prostate cancer and is a risk factor for systemic fungal infection (8).

Prostate cancer is the second most commonly diagnosed form of cancer and the sixth leading cause of cancer-related death among men worldwide (1). The prevalence of prostate cancer in men with end-stage renal disease might be higher than that in men without renal disease (9). Because of reduced urine output, men undergoing renal replacement therapy are less likely to manifest symptoms of prostatism, and the diagnosis of prostate cancer depends more on screening for elevated serum levels of prostate-specific antigen. In our patient, serum prostate-specific antigen was not assayed until after peritoneal prostate cancer had been detected.

Our case emphasizes the importance of serum biomarker screening for prostate cancer in the dialysis population. Because the number of end-stage renal disease patients is increasing steadily, as is their average age, and because home dialysis, including PD, is becoming a more popular renal replacement modality, prostate cancer metastasis to the peritoneum in chronic PD patients may be seen again.

Once peritoneal metastases were detected in our patient, we were unsure whether it was safe to continue PD. Our patient greatly preferred PD to hemodialysis, which had been used to support him transiently after removal of the PD catheter for fungal peritonitis. We were concerned about repeated infectious peritonitis episodes, increased peritoneal permeability with ultrafiltration failure, and decreased effective peritoneal surface area causing decrease in small-solute clearance. In fact, peritonitis did recur even after the peritoneal catheter was replaced, but the organism was a skin *Staphylococcus* species rather than a fungus or enteric bacterium. However, over the 13-month period from discovery of the prostate cancer in the patient's peritoneum to his death, small-molecule clearance and control of extracellular fluid volume were readily achieved.

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DISCLOSURES

The authors have no financial conflicts of interest to declare.

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REFERENCES

1. GLOBOCAN 2008. Prostate Cancer Incidence and Mortality Worldwide in 2008: Summary [web page]. Lyon, France: International Agency for Research on Cancer; 2010. [Available at: <http://globocan.iarc.fr/factsheets/cancers/prostate.asp>; accessed 9 November 2011]
2. Bubendorf L, Schöpfer A, Wagner U, Sauter G, Moch H, Willi N, et al. Metastatic patterns of prostate cancer: an autopsy study of 1,589 patients. *Hum Pathol* 2000; 31:578–83.
3. Saitoh H, Hida M, Shimbo T, Nakamura K, Yamagata J, Satoh T. Metastatic patterns of prostatic cancer—correlation between sites and number of organs involved. *Cancer* 1984; 54:3078–84.
4. de la Monte SM, Moore GW, Hutchins GM. Metastatic behavior of prostate cancer. Cluster analysis of patterns with respect to estrogen treatment. *Cancer* 1986; 58:985–93.
5. Kehinde EO, Abdeen SM, Al-Hunayan A, Ali Y. Prostate cancer metastatic to the omentum. *Scand J Urol Nephrol* 2002; 36:225–7.
6. Zagouri F, Papaefthimiou M, Chalazonitis AN, Antoniou N, Dimopoulos MA, Bamias A. Prostate cancer with metastasis to the omentum and massive ascites; a rare manifestation of a common disease. *Onkologie* 2009; 32:758–61.
7. Brehmer B, Makris A, Wellmann A, Jakse G. Solitary peritoneal carcinomatosis in prostate cancer (German). *Aktuelle Urol* 2007; 38:408–9.
8. Wise GJ, Shteynshlyuger A. How to diagnose and treat fungal infections in chronic prostatitis. *Curr Urol Rep* 2006; 7:320–8.
9. Kamata T, Fushimi K. Prevalence of prostate cancer in end-stage renal disease patients. *Urol Int* 2008; 80:419–24. doi:10.3747/pdi.2011.00281