CASE REPORT

Isolated prostate cancer soft tissue recurrence 10 years after radical prostatectomy

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SUMMARY

In advanced disease, prostate cancer is well known to invade locally as well as metastasise to distant locations. Metastases occur commonly in lymph nodes and bone but have also been known to involve certain visceral organs, particularly the lungs. Involvement of soft tissue by metastases is far less common, particularly in the context of cancer recurrence. We present the case of a male aged 68 years who presented with a rising prostate-specific antigen (PSA) 10 years after radical prostatectomy (RP). The PSA increased despite salvage radiotherapy and was ultimately found to be caused by a PSA secreting prostate cancer soft tissue mass in the suprapubic region. Surgical resection of the mass caused a sharp decline in the PSA to negligible levels. This case highlights the need for ongoing surveillance post-RP and the potential for prostate cancer recurrence in the soft tissue that is refractory to routine salvage radiotherapy.

BACKGROUND

Radical prostatectomy (RP) is a common operation in urology that has a good treatment outcome for prostate cancer (CaP) regardless of the surgical approach. Postoperatively, the serum prostate-specific antigen (PSA) level is monitored closely as a biochemical indicator for recurrence and is used to guide salvage therapy. We present an unusual aetiology of a rising PSA in a patient post-RP and despite salvage radiotherapy.

CASE PRESENTATION

Ten years earlier, a male aged 58 years with a preoperative PSA of 9.0 ng/mL underwent an RP with pelvic lymph node dissection for a Gleason grade group 2 (Gleason score 3+4=7, with elements of tertiary pattern 5) pT3a N0 mixed acinar and ductal prostate cancer. There was a 2 mm positive apical surgical margin. The postoperative PSA levels remained undetectable (PSA <0.2 ng/mL) for 4 years before biochemical recurrence (PSA 0.24 ng/mL). The PSA doubled over 19 months (PSA 0.49 ng/mL), and there was no evidence of metastatic disease on staging scans. At 6 years postprostatectomy, he underwent salvage external beam radiation treatment to the prostatic bed. No adjuvant hormone therapy was used. Ongoing PSA surveillance postradiotherapy demonstrated initial biochemical response with a nadir of 0.22 ng/mL followed by a gradual increase over a period of 2 years. Peak PSA was 3.15 ng/mL.

INVESTIGATIONS

Restaging CT and MRI scans across a 12-month interval demonstrated a gradually enlarging soft tissue mass deep to the left inferior rectus muscle and superior to the pubic bone (figure 1). Bone scintigraphy was negative. The soft tissue mass was biopsied under radiological guidance and histology confirmed CaP recurrence with positive PSA immunohistochemistry.

TREATMENT

The patient underwent open surgical excision of the mass. Intraoperatively, the mass dissected easily off the pubic bone and adjacent rectus muscle was excised with the specimen. The mass did not involve the bladder. Mesh was placed to close the rectus sheath defect. Compared with the original specimen (figure 2, A-B), histology demonstrated poorly differentiated prostatic adenocarcinoma with ductal morphology, frequently associated with comedo necrosis, clear of margins (figure 2, C-D).

OUTCOME AND FOLLOW-UP

The patient recovered quickly from surgery and was discharged home the following day. The 4-month postoperative PSA and 12-month postoperative PSA were 0.07 and 0.11 ng/mL, respectively (figure 3). It should be noted that these results were performed with the ultrasensitive PSA test (which is capable of detecting PSA to the level of 0.03) and are likely comparable with the undetectable PSA following initial RP (lower limit of detection 0.2 ng/mL).

DISCUSSION

Prostate carcinoma is one of the most common cancers to affect men. RP remains an effective operation for primary CaP, but approximately 15%–40% of patients will experience cancer recurrence within 5 years.^{1 2} At biochemical recurrence, a search for the site of disease involves imaging of the prostatic bed and local lymph nodes with CT or MRI of the pelvis and abdomen, along with whole body bone scan. These areas are the most common sites of recurrence but often no site is found and the cancer is assumed to be too small to detect with modern imaging techniques.³ Even with the latest PSMA-positron emission tomography CT scan, the detection rate of cancers when the PSA is <0.5 ng/mL is approximately 50%,⁴ which goes some way towards explaining why our patient did not have a radiologically detectable recurrence until there was sufficient PSA rise.

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Figure 1 T2-weighted MRI scan (axial and sagittal views; mass demonstrated by white arrow).

Unusual sites of recurrence in CaP may reflect the metastatic potential of the underlying tumour. A peculiarity worth noting in this case is that, while the original tumour histology showed mixed acinar and ductal adenocarcinoma, the recurrence consisted entirely of ductal adenocarcinoma. Additionally, the recurrence showed a predominantly Gleason grade 5 pattern due to the associated comedo necrosis,⁵ a feature seen in <1% of the original tumour. This is not a surprising finding given that ductal adenocarcinoma is generally considered to be an aggressive variant of prostate cancer.⁶ The slow progression of disease in our patient suggests the cancer did not develop the complex mechanisms required for metastasis⁷ and although haematogenous spread to the suprapubic site remains a possibility, we hypothesise that this recurrence may represent wound site seeding, which would have occurred at the time of initial surgery.

Wound site recurrence following RP is an uncommon clinical finding. The reported rate of port site involvement among laparoscopic urology operations in the literature is 0.09%–0.73%, with only a small portion contributed by prostate surgery.⁸ Unfortunately, similar data for open procedures are not available in the literature. Although wound site recurrence has been previously documented following RP, the mechanism underlying this remains poorly understood. Various theories

Figure 2 (A, B) Original radical prostatectomy specimen; (A) acinar adenocarcinoma (H&E 40x), (B) ductal adenocarcinoma (H&E 40x). (C, D) Suprapubic mass; (C) malignant glands showing Gleason grade 5 pattern of ductal morphology with a cribriform architecture and comedo necrosis (H&E 100x), (D) diffusely positive staining with prostate-specific antigen immunohistochemistry (100x).

PSA level over time

Figure 3 Prostate-specific antigen (PSA) trend over time. Note peak PSA prior to salvage radiotherapy and excision of suprapubic mass (0.49 and 3.15, respectively) in comparison to nadir PSA postsalvage radiotherapy and excision of suprapubic mass (0.22 and 0.07, respectively).

have been put forward including haematogenous spread, prostatic secretion during surgery, tumour contamination of closure devices and manipulation or inadvertent sectioning through the tumour while removing the specimen from the body.^{9–12}

Irrespective of the mechanism, recurrences that occur at the distant surgical point of access wound are thought to reflect more aggressive tumours than those occurring locally at the direct site of excision.¹³ The slow growing behaviour of the recurrence in our patient is thus very unusual, as is the fact that the cancer did not spread elsewhere. Interestingly, our patient presented in a very similar manner to the one reported by Metzendorf and Schmidt, currently the only other known case of isolated prostate cancer pelvic recurrence following open RP.¹⁴ It remains to be seen how our patient's PSA will behave in the long term following excision of the soft tissue recurrence but in the short term it is reassuring to see a dramatic reduction in PSA level, as well as stability over a 12-month period. This, in conjunction with histologically proven clear margins, points to a successful debulking of the PSA secreting mass with the hope of at least delaying the need for androgen deprivation therapy.

This case highlights the utility of PSA surveillance post-RP, along with serial restaging imaging studies in the case of a biochemical recurrence. Where a wound site recurrence following RP is suspected, we would suggest aggressive surgical resection where possible.

Learning points

- ▶ Biochemical recurrence postradical prostatectomy (RP) should prompt a radiological search for site(s) of disease.
- ▶ Wound site seeding post-RP, although uncommon, is a possible site of cancer recurrence.
- ▶ Where possible, localised recurrence post-RP should be treated with surgical resection.

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