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Case report

Basal cell carcinoma of the prostate: a case report of a rare prostate cancer and review of the literature

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Abstract. Basal cell carcinoma (BCC) of the prostate is a very rare histologic variant of prostate cancer, of difficult diagnosis and uncertain prognosis. Despite the frequency of prostate cancer, only 100 cases have been described in the literature, and most of the data derived from case reports. Because of the rarity of this disease, therapeutic options for these patients are scarce and no randomized trials are available. Here, we report the case of a 63-year-old man treated for a prostatic BCC (pBCC) that was challenging in terms of both diagnosis and treatment. We also performed a review of the literature to provide an overview of the therapeutic options for this rare tumor type and to better understand the role of molecular characterization in rare prostate cancer histologies. Given the rarity of pBCC worldwide, further collection of real-world data is needed to better understand the optimal diagnostic and therapeutic strategies for this rare disease. (www.actabiomedica.it)

Key words: prostate cancer, basal cell carcinoma, next-generation sequencing, rare histology, therapy, chemotherapy

Background

Prostate cancer is one of the leading causes of mortality and morbidity due to cancer in men in Europe (1). More than 90% of prostate tumors are acinar adenocarcinomas but many other variants with different behaviors have been described (2). Rare prostate cancer histology account for 5-10% of all prostate cancers; among these, pBCC is a very rare variant with uncertain behavior.

BCC of the prostate was first described in 1974 as a distinctive variant of prostatic adenocarcinoma; its incidence is less than 0.01% and, in the literature, only 100 cases are described (3).

Because of its histological features (cribriform pattern with intraluminal eosinophilic hyalinized substance) and its resemblance to the salivary gland and breast counterparts, pBCC was initially referred as adenoid cyst carcinoma (ACC) of the prostate (4). In 2004, the World Health Organization (WHO) recognized ACC of the prostate as a spectrum of pBCC (5). The WHO 5th edition 2021 defines ACC/BCC of the prostate as a malignant neoplasm derived from prostatic basal cells with varying proportions of 1) the adenoid cystic/cribriform pattern with inspissated secretions and 2) the basaloid pattern with small solid nests of basal cells or, less commonly, cords of cells or small tubules (6,7). This tumor entity includes different subtypes with completely variable clinical courses, ranging from incidental diagnosis and indolent clinical courses to metastatic aggressive diseases (8,9).

Typical metastatic sites are liver, lung, and bowel while, unlikely conventional prostate acinar adenocarcinomas, bone is not often involved (10). Symptoms are non-specific, ranging from nocturia, urgency, or progressive/acute urinary retention (11). Moreover, since pBCC derives from the basal cell layer, its cells do not release prostate-specific antigen (PSA). In fact, PSA elevation was described in a few cases in which there was concomitant acinar adenocarcinoma of the prostate (12).

Basal tumor cells specifically express basal cell markers (e.g. p63, p40, high molecular weight cytokeratins such as CK34BetaE12 and CK5/6) (6,7) and epithelial/adhesion markers (e.g. CKAE1/AE3, CK7 only in adluminal cells, EPCAM, CDH1 and CD24) (13). Immunostains for BCL2 label pBCC more strongly and diffusely than basal cell hyperplasia; Ki67 staining is over 20% in approximately half of pBCC and PSA is expressed in a minority of cases (6,7). Compared to conventional acinar prostate acinar adenocarcinomas, in pBCC Racemase (AMACR) and androgen (AR) are typically negative (6,7).

From a molecular point of view, pBCC is often characterized by some frequent findings such as loss of PTEN expression, overexpression of EGFR and MYB translocation (14). The five putative driver genes mutated in BCC are CASC5, NUTM1, PTPRC, KMT2C, and TBX3, while the top three nucleotide substitutions are C>T, T>C, and C>A, similar to common prostate cancer (15).

In view of its rarity, therapeutic options for patients with ACC/BCC of the prostate are scarce and no randomized trials are available. Most of the patients are treated with hormone therapy, radiotherapy, radical prostatectomy, chemotherapy, or a combination of these treatments, although outcomes remain poor. Targeted therapies such as pemigatinib and vismodegib have also been used (16). An in-depth molecular analysis could allow to get a better characterization of the disease, identify prognostic and predictive factors and undercover some new therapeutic targets.

Here, we present the case of a 63-year-old man diagnosed with pBCC treated in our department.

The patient has provided written consent for the treatment of his dates and their utilization for scientific purposes.

Case presentation

In February 2021, a 63-year-old man was referred after the occurrence of palpable clustering of inguinal lymph nodes. He presented with a past medical history of rectal adenocarcinoma treated with chemotherapy and radiotherapy in 2009, diabetes mellitus and visual impairment of the right eye. The patient presented in good clinical condition with an Eastern Cooperative Oncology Group Performance Status (ECOG PS) of 0. In consideration of the clinical presentation, a CT scan of the chest and the abdomen-pelvis was performed and showed pathological lymph nodes in the left inguinal region, in the para-rectal adipose tissue and along the iliac and para-aortic vessels, confirmed also with an FDG-PET-scan.

To obtain a histological diagnosis, the patient underwent an excisional biopsy of the inguinal lymph node and the histological result was suggestive of a metastatic deposit from an undifferentiated carcinoma of unknown origin (metastasis of unknown origin - MUO).

In view of this diagnosis, in April 2021 the patient started a standard first-line chemotherapy with carboplatin area under the concentration–time curve (AUC) 5 mg per millilitre per minute (AUC5) plus paclitaxel 175mg/mq every 21 days. The chemotherapy was continued until July 2021 (3 cycles), when a grade 3 neuropathy occurred, leading to the interruption of the treatment. The restaging PET-scan showed an extraordinary complete metabolic response.

Unfortunately, 4 months after the discontinuation of the therapy the patient presented haematuria and an abdominal MRI was performed showing a severe structural alteration of the prostatic gland and multiple pathological abdominal lymph nodes. Another PET-scan was performed showing pathological glucose uptake on mediastinal and abdominal (para-aortic, obturator, and iliac) lymph nodes (SUV max 8), as well as on the prostatic gland (SUV max 10). At that time, PSA was 0.33 ng/ml.

Since the strong suspect of a primary prostatic tumour, in January 2022 the patient underwent a prostate needle biopsy with a histological diagnosis of basal cell carcinoma (pBCC) consisting of cells with slightly enlarged rounded hyperchromic cores and arranged in

solid basaloid nested, both in cords of cells and with cribriform pattern (Figure 1a-c); presence of focal tumor necrosis in solid basaloid nested was also observed (Figure 1d).

A positive expression for CK34BetaE12, p63, Bcl2, Ki67 (45%), CKAE1/AE3 was observed, while no PSA expression was recorded (Figure 2).

All these characteristics led to the histological diagnosis of pBCC infiltrating the seminal vesicles (Figure 1e), with morphological and immunohistochemical features superimposable to those observed in the previous histological report of the inguinal lymph node metastasis.

Before starting a new treatment, imaging restaging was performed with total body CT and bone scan, which do not show any new metastatic sites.

Therefore, in March 2022 the patient started androgen-deprivation therapy (ADT) with triptorelin acetate 11.25 mg subcutaneously every three months in addition to docetaxel 75 mg/mq every 21 days for three cycles. A restaging CT scan (3 May 2022) showed stable lymph node disease and a slight increase of the prostate gland. PSA dropped to 0 from 0.14 ng/ml. In consideration of the overall stable disease and the good tolerance to the chemotherapy, three more cycles of docetaxel were prescribed.

In the meanwhile, due to the young age and the rare histology of prostatic cancer that limited therapeutic chances, FoundationOne®CDx and FoundationOne®Liquid CDx analyses were performed.

After one cycle of chemotherapy (27 May 2022) the patient experienced bowel obstruction and a new CT scan of the abdomen showed a severe progression of the prostatic lesion, which infiltrated and involved the entire bladder and the rectum with the development of an abscess extending to the sacrum (Figure 3a-g). Moreover, progressive disease of the pathological lymph nodes and the appearance of bone metastases was reported (Figure 3h). There was no indication for urological or radiotherapeutic interventions.

Due to the severe clinical and radiological progression, the aggressive disease and the great response obtained with the first chemotherapeutic regimen, a rechallenge with carboplatin AUC 4 mg/ml/min plus cabazitaxel 20 mg/mq every 21 days was prescribed in June 2022. In August 2022, after two cycles of chemotherapy, the patient was admitted for

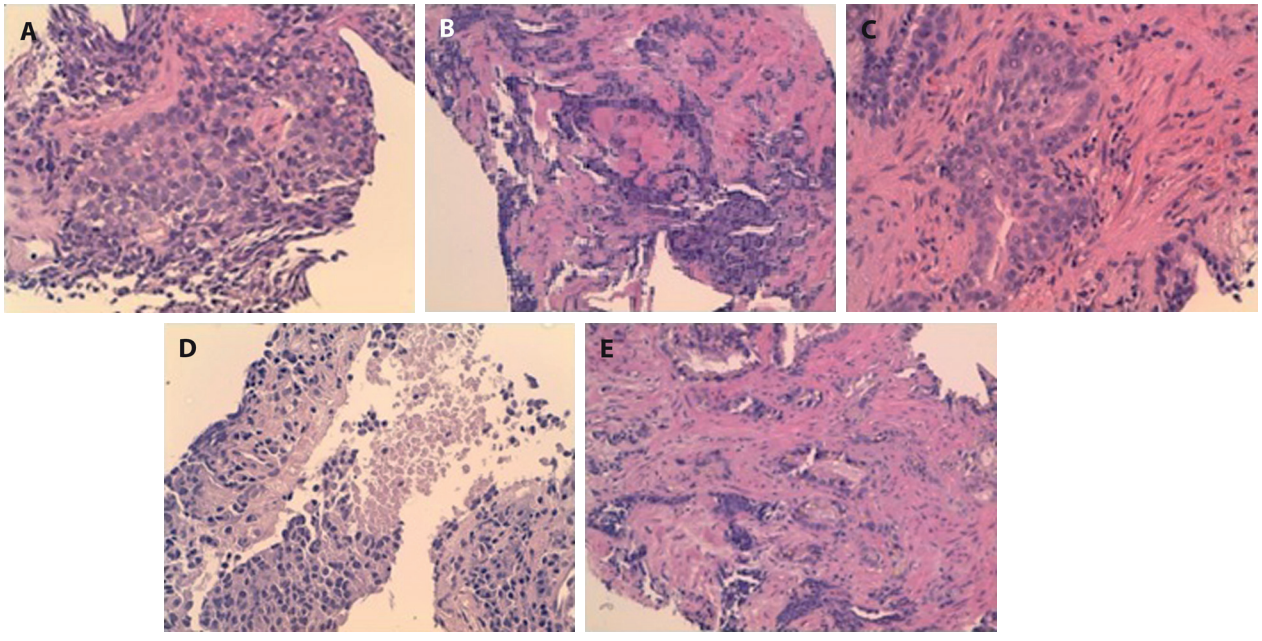

Figure 1. Histological images of pBCC. Solid basaloid nested (haematoxylin and eosin staining – H&E 40x) (a); small basaloid nested and cords of cells (H&E 40x) (b); cribriform pattern (H&E 40x) (c); tumor necrosis in solid basaloid nested (H&E 40x) (d); infiltration of seminal vesicles (H&E 40x) (e).

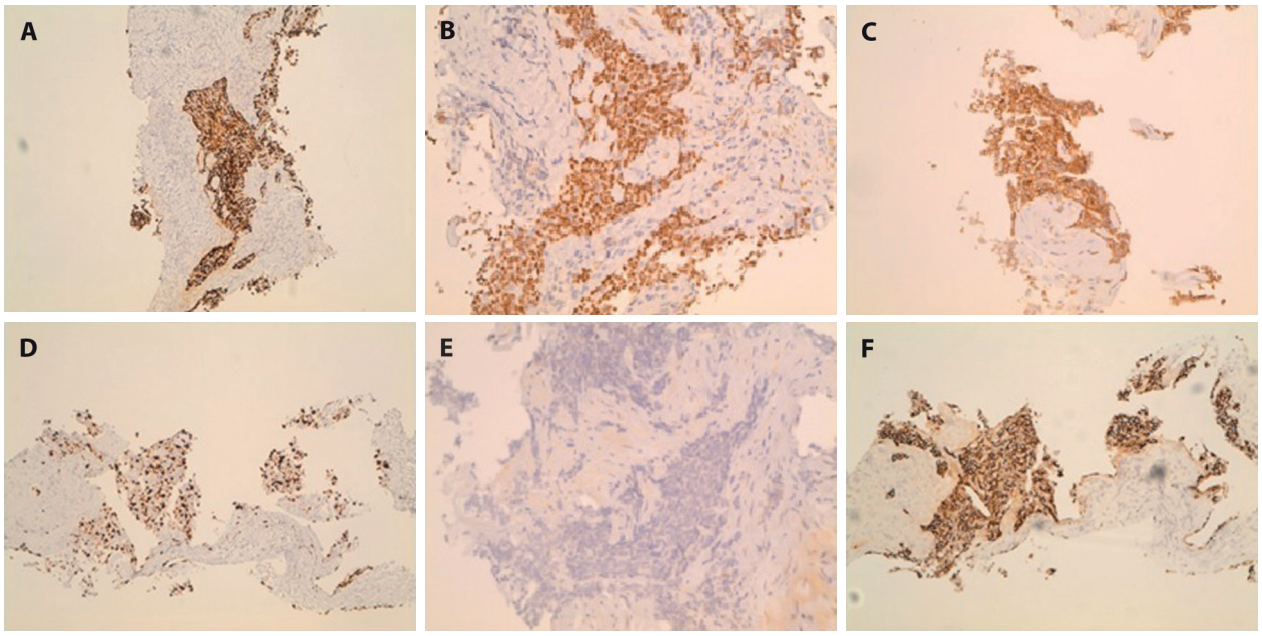

Figure 2. Immunohistochemical expression images of pBCC. CK34BetaE12 expression (30x) (a); p63 expression (20x) (b); Bcl2 expression (20x) (c); Ki67(MIB1) (d); PSA negativity (e); CKAE1/AE3 expression (30x) (f).

acute renal failure and bilateral hydronephrosis due to the local progressive disease, further complicated by sepsis sustained by Klebsiella pneumoniae. In the meanwhile, the sequencing of the circulating tumour DNA revealed an ATM alteration (R1875) with a Variant Allele Frequency of 0.10% that, unfortunately, was not confirmed on the tumoral tissue. For this reason, the patient was not considered eligible to start a PARP inhibitor. No other molecular alterations, including MMR, TMB and BRCA, were detected. During the hospitalisation, another total body CT scan (August 2022) showed further significant progressive disease and the occurrence of peritoneal carcinomatosis.

In consideration of the poor performance status, the prior treatments received, the severe disease progression despite chemotherapy and the lack of targeting mutations, the patient was considered for best supportive care only. The patient died in October 2022.

Discussion

BCC of the prostate is a very rare and aggressive prostate cancer type, and no standard systemic treatments are available. Several case reports and case series on pBCC are available in the literature and very few case reports described metastatic pBCC patients.

For patients with localized disease, radical surgical resection is the preferred therapeutic option, with the aim of ensuring free margins and clear histological characterization of ACC/BCC. However, ACC/BCC is usually characterized by an extensive local infiltration pattern and perineural spread, which makes it difficult to achieve radical surgery (18). Negative prognostic factors included young age, involvement of the peripheral zone of the prostate, extra-prostatic spread, perineural and peri-glandular invasion (11,14). Aggressive surgical treatments, such as pelvic exenteration, were initially described in some case reports (11) but were subsequently abandoned to give way to more conservative treatments, often characterized by the combination of two or more therapeutic approaches (12). When primary surgery is performed, the addition of postoperative radiotherapy is considered when negative prognostic factors are present (e.g. advanced tumors, positive resection margins and perineural infiltration) (18). In some other cases, radical radiotherapy associated with hormone therapy or chemotherapy was administered (19).

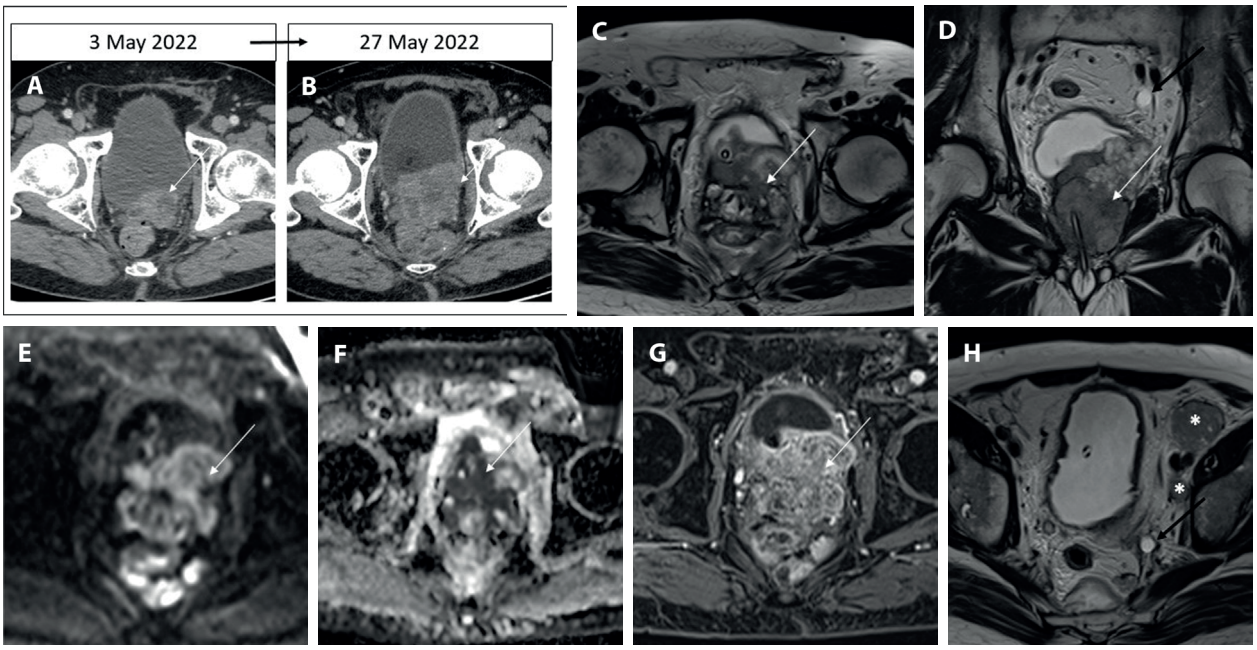

Figure 3. CT and MRI imaging of mBCC patient. CT scan images (in the rectangle) of the prostatic disease (arrow) progression during May 2022 (a,b). MRI scan images of the large prostatic mass (white arrow), which enveloped the intraprostatic urethra, infiltrated the mesorectal adipose tissue and determined left hydronephrosis (black arrow) (c-g). The pathologic tissue showed a heterogeneous high-intermediate signal intensity on T2-weighted images, which are not typical for adenocarcinoma prostate cancer (c, d) with high signal restriction on diffusion-weighted imaging (e), low signal on Apparent Coefficient diffusion map (f) and early and significant contrast enhancement on T1 weighted images (g). Large left iliac pathologic lymph nodes (asterisks) had the same MRI signal features (h).

For patients with unresectable locally advanced or metastatic disease, it is not possible to draw clear conclusions on therapeutic options since very few cases are reported in the literature (Table 1). Different therapeutic options can be used in this setting, such as chemotherapy based on platinum agents (cisplatin, carboplatin) or taxanes (docetaxel, paclitaxel), as in head and neck cancer, 5-fluorouracil plus mitomycin C and ADT (bicalutamide and/or luteinizing hormone-releasing hormone analogues/antagonists), alone or in combination strategies (19-23). Moreover, also etoposide could be considered, especially for heavily pretreated patients with small cell/neuroendocrine differentiation and aggressive histology, even if its mechanism of action in patients with pBCC should be further studied (8). Only limited data are available regarding the use of immunotherapy in this setting. The literature reports a favorable response with the use of Pembrolizumab, while the combination of Atezolizumab with PARP inhibitors has shown lower efficacy (24,25).

In our clinical case, at the beginning of the medical history, we chose carboplatin plus paclitaxel as the main combination chemotherapy in patients with metastases of unknown primary carcinoma (26).

Second-line treatment with carboplatin AUC 4 mg/ml per minute in association with cabazitaxel 20 mg/mq was proposed due to the complete response reached with the platinum-based treatment, the indication of cabazitaxel in mCRPC patients receiving progressed o docetaxel and the indication of this combination in patients with aggressive variant metastatic CRPC (27).

Of note, since ACC/BCC has proven to not express the AR, the impact of ADT remains unclear, even if LHRH-agonists and antiandrogen drugs are still considered the treatment of choice for inoperable cases or metastatic disease (15).

In our case, we decided to perform a next-generation sequencing (NGS) assay with the aim of identifying new therapeutic options for our patient. In

Table 1. Summary of case reports on metastatic pBCC.

First author, Year [Reference]	Age	Site of mts	Molecular alterations	Treatment	Outcomes
Kumar Julka P et al., 2020 [21]	79	Liver	NA	CBDCA + Paclitaxel + ADT	PFS: 6 mo OS: 16 mo
Chang K et al., 2013 [22]	48	Bones, liver	NA	Docetaxel + local ablative therapy	OS: 3 yrs
	65	Lungs	NA	ADT	OS: 6 mo
Segawa N et al., 2008 [15]	67	Lymph nodes	NA	ADT	OS: 8 mo
Dong S et al., 2020 [8]	62	Lung	ATM mutation SMARCB1 mutation PIK3R1 mutation	1° line: docetaxel + CDDP 2° line: etoposide	Best response: PD Best response: PR PFS2: 9 mo
Rebhan K et al., 2022 [16]		Lymph nodes, liver	FGFR2-TACC2 fusion	1° line: vismodegib 2° line: pemigatinib	Best response: PD Best response: CR PFS2: NR

Abbreviations: mts: metastasis; NA: not analyzed; ND: not detected; CDBCA: carboplatin; ADT: androgen deprivation therapy; CDDP: cisplatin; mo: months; PFS: progression-free survival; OS: overall survival; PD: progressive disease; PR: partial response; CR: complete response; NR: not reached.

fact, one case of pBCC resulted in fibroblast growth factor receptor (FGFR) fusion and was treated with the FGFR inhibitor pemigatinib achieving both clinical and radiologic response (16).

As for ACC/BCC, also for intraductal carcinoma of the prostate, some gene signatures are described (28). Deeper knowledge of the fundamental molecular mechanisms and genomic characterization of prostate cancer could lead to the development of actionable targets for novel therapeutic agents (29). In this setting, unfortunately, our clinical case has no actionable target from NGS and the patient was treated with conventional chemotherapy with poor long-term outcomes.

In the end, in MUO disease where rare histologies can be diagnosed, vigilance is crucial for clinicians during the diagnostic workup and further pathologic revisions and experts' second opinions should be considered. This is fundamental to provide the optimal treatment for oncologic patients, even if it requires a change in the initial therapeutic plan (30).

Conclusions

Given the rarity of BCC/ACC worldwide, further collection of real-world data is needed to better understand the optimal diagnostic and therapeutic strategies. Especially for patients with locally advanced or metastatic pBCC, only a few data are available until now and defining the right treatment option and sequence remains an unmet clinical need. Moreover, since the role of ADT remains unclear, more studies are needed to find the most effective therapeutic strategies for this histologic subtype.

Informed Consent: the patient had signed informed consent for the treatment and collaboration with research.

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Ethics Committee: Not applicable.

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manuscript. SER, GF, MS, VE, FR: clinical management of the patient and provision of patient's data. All authors contributed to the article and approved the submitted version.

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