


CASE REPORTS

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# Prostatic basal cell carcinoma treated by chemoradiation with weekly cisplatin: case report and literature review

S. Ridai<sup>1\*</sup> , C. Moustakbal<sup>1</sup>, A. Lachgar<sup>2</sup>, H. Jouhadi<sup>1</sup>, A. Benider<sup>1</sup>, M. Regragui<sup>3</sup> and F. Marnissi<sup>3</sup>

## Abstract

**Background:** Basal cell carcinoma of the prostate is a relatively rare entity. Their evolution is characterized by the frequency of local and/or distant relapses. Due to their rarity, the treatment is not consensual in the literature. We report here a case of Basal cell carcinoma of the prostate in a 40-year-old patient.

**Case presentation:** Our patient initially presented an obstructive lower urinary tract symptoms with a normal initial level of prostate specific antigen (PSA) test (3.5 ng/ml). The transurethral resection of the prostate (TURP) was in favor of a prostatic basal cell carcinoma with its specific anatomopathological and immunohistochemical characteristics. The prostatic MRI and thoraco-abdominal CT realized after the TURP revealed a tumoral lesion of the prostatic peripheral zone with extra-capsular extension combined with right seminal vesicle invasion and a suggestion of posterior bladder wall adherence. No evidence of visceral or nodal metastases at this point. Considering the tumor being locally advanced, a concurrent chemoradiotherapy with intensity modulated technique was indicated after a multidisciplinary meeting with a 70 Gy total target dose delivered in 35 fractions and weekly Cisplatin. A year and a half after, he developed a cerebellous metastases revealed by intracranial hypertension with no other visceral lesion and complete local remission with the disappearance of the lower urinary tract symptoms and the pain and the appearance of a prostatic atrophy. The PSA level was still on the upper limit of normal. He underwent metastasectomy, and the anatomopathological study was in favor of a cerebellous metastasis of the known BCC. The patient presented postoperatively paraparesis of lower limbs with balance problems for which he was placed in palliative care with indication of postoperative radiation therapy in case of improvement of his general condition. He did not recover and deceased three months later.

**Conclusions:** The prostatic basal cell carcinoma is a rare aggressive entity often non-evoked at the clinical or radiological stages because of its unspecific appearance. The diagnostic of these tumors is based on histological examination and a large immunohistochemistry panel. Given its scarcity, very few data is available for locally advanced non-metastatic stages treated by radiation therapy. We assess here a good local response with concurrent chemoradiation therapy.

**Keywords:** Prostate cancer, Basal cell carcinoma, Chemoradiation, Case report

## 1 Background

The prostatic basal cell carcinoma is a histological variant of prostatic carcinoma which was first reported and named by Frankel in 1974 [1]. Less than eighty cases were described, and the largest series included 29 cases in 2007 [2].

\*Correspondence: Ridaisara@gmail.com

<sup>1</sup> Centre Mohamed VI pour le traitement du cancer, Casablanca, Maroc  
Full list of author information is available at the end of the article

This type of prostatic carcinoma arises from the prostatic acinar cells, the transitional epithelium of the prostatic urethra and the peri-urethral tissue [3, 4].

Patients ages ranges from a low of 28 to a high of 89 with a higher rate among the elderly and very few cases among patients younger than 40 years old [3].

It generally causes an obstructive symptomatology with a high incidence of acute urinary retentions considering their frequent localization on the prostatic transitional zone [5].

At examination, the prostate is often hypertrophic and partially indurated [3].

Since it is developed from the non-secreting basal prostatic cells which does not synthesize kallikrein, it does not cause an increase in PSA levels unless associated with a glandular pattern [4–6].

While BCC borrows the basocellular skin cancer morphology and the adenoid cell carcinoma (ACC) tends to look like the accessory salivary glands ACC, multiple overlapping architectures of adenoid or basaloid pattern can be observed [2, 7]. The basaloid type is generally characterized by clusters of basaloid cells, rare cribriform architecture with a hyalinized appearance, and the adenoid morphology is represented by peripheric solid nests and cribriform structures around a loose myxoid stroma [3, 4].

The Gleason score cannot be applied to these specific tumors because of their basal cell's origin [3]. These tumors are recognizable by a large immunohistochemistry panel which allows to exclude a conventional prostatic adenocarcinoma.

Differential diagnoses range from benign prostatic hyperplasia to poorly differentiated adenocarcinoma or squamous cell carcinoma.

The benign hyperplasia can be differentiated by the absence of invasive characteristics: central necrosis and/or perineural invasion [3, 4, 7].

The squamous cell carcinoma even if developed in the same way as the BCC has its own characteristics of squamous differentiation (keratinization, intercellular bridges, etc.), and the poorly differentiated adenocarcinoma has a PSA immunohistochemical (IHC) staining almost or truly negative like in BCC but the cytokeratin (CK) 14 and the high molecular weight keratin negativities can make the difference in case of basaloid tumor [3].

Concerning the BCC, the luminal cells express the cytokeratin 7, while the prostatic basal cells express the high molecular weight keratin also called cytokeratin 903 (34 $\beta$ E12) and the cytokeratin 5/6 which is a component of the latter that has a higher sensitivity and is easiest to use. They also express the cytokeratin 14 [3–5, 8].

Both basaloid and adenoid forms express the anti-p63 antibody. This immunopositivity can be diffuse in

basaloid variant and compartmentalized in the adenoid type [9].

The PAP, cytokeratin 20, AMACR enzyme (p504S) and neuro-endocrine (chromogranin and synaptophysin) stainings are usually negative [10].

So is the PSA immunohistochemical staining unless there is an associated conventional carcinoma.

Some cases of BCL2 and Ki 67 high positivity have been reported. The Ki67 proliferation index allows here to differentiate between benign and malignant lesions and to correlate sometimes its value to the tumor's aggressivity [11].

Even membrane HER2neu, BRCA2 mutation and EGFR overexpression in IHC despite the absence of its gene amplification by ISH have been described [12, 13].

Being rare, there are no recommendations for treating prostatic basal cell carcinoma.

While the retropubic prostatectomy appears to be the preferred treatment option in localized disease [14], the role of surgery remains unclear in front of locally extended or metastatic forms.

In these cases, androgen deprivation therapy, radiation therapy and even chemotherapy have been discussed in several papers.

Some cases reported treatment by definitive or concomitant radiation therapy in locally advanced cases leading to a mean overall survival of 38 months [14].

Chang & al treated two of their three patients with 3D conformational radiation therapy (RT) with an important shrinkage of tumor volume but apparition osseous and liver metastases at 4 and 30 months after their treatment [15].

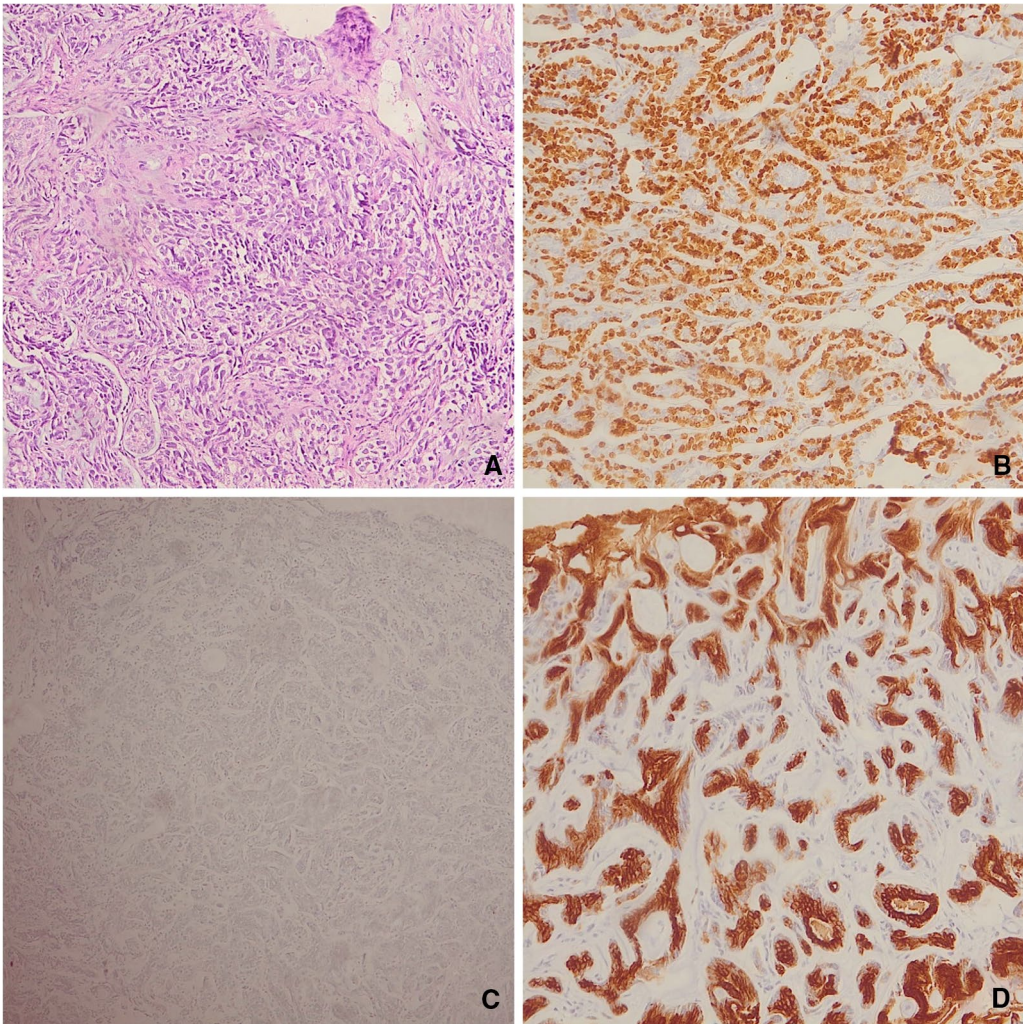
Very few complete remissions were reported but with very short follow-up times [16].

The longest reported follow-up was around nine years [17].

Even if the hormonal therapy was considered the gold standard of the first-line therapy for the advanced or metastatic disease [18], its impact remains limited as several authors reported progression and death of disease after ADT (luteinizing hormone—releasing hormone agonist and antiandrogen agents) for inoperable cases with fixed growth in the pelvis or metastatic disease [9, 15, 19].

Considering the non-androgen secretion of the zone BCC origins from and then the non-expression of the androgen receptor the impact of hormonal treatment remains poor and uncertain [12].

As for the chemotherapy, there is no standard regimens and no clinical response described but the docetaxel is the preferred drug or even the etoposide if a neuroendocrine pattern is individualized on the pathology examination.



**Fig. 1** a Cribriform pattern with large solid basaloid nests, b Expression of p63 by basal cells, c No PSA immunostaining, d Cytokeratin 7 expression by luminal cells



**Fig. 2** Prostatic MRI showing the prostatic tumoral lesion with extra-capsular extension, right seminal vesicle invasion and a posterior bladder wall adherence





**Fig. 3** Pelvic CT scan showing the prostatic hypertrophy and a seminal vesicle invasion

More recently, Beth Israel Deaconess team treated a metastatic basal cell carcinoma presenting BRCA2 mutation with PARP inhibitors and targeted therapy followed by metastasectomy 28 months after diagnosis with a quasi-complete remission under olaparib maintenance therapy [13]. These kind of personalized treatments depends on next-generation sequencing result and cannot therefore constitute a generalized recommendation.

The BCC prognosis cannot really be determined unless we identify factors predictive of metastatic potential.

This neoplasm would show, according to Grignon, an indolent course but a subclass developed local recurrences and distant metastases [20]. This could have been predicted by the initial morphology and extent because it can be predictive of an aggressive behavior [10] just like extraprostatic extension to the bladder neck or the seminal vesicle [7].

Iczkowski & co assessed the 5-year metastatic risk, and it is obviously more important for advanced disease (from 5 to 10% for T1/T2 versus 50 to 85% in T3/T4) [5].

Even Simper & all reported 10% of deaths from disease with local recurrences among patients with pT3/pT4 disease, while the more localized tumors did not cause any recurrences [12]. Local recurrences and distant visceral metastases to the liver, lungs and even to the penis have been described [2, 3] but unlike the acinar carcinoma, bone is not the predilection metastasis site [3].

## 2 Case presentation

We report a case of prostatic BCC in a 40-year-old patient with no medical history nor comorbidities who initially presented an obstructive lower urinary tract symptoms with a normal initial level of prostate specific antigen (PSA) test (3.5 ng/mL). The digital rectal examination

showed a slightly indurated and very enlarged prostate and the ultrasound objectified an important post-void residual urine volume.

The anatomopathological study of the transurethral resection of the prostate (TURP) was in favor of a prostatic basal cell carcinoma; there was a cribriform pattern with large solid basaloid nests without perineural invasion. The luminal cells were columnar and mucus-secreting, while the basal ones were smaller and basophilic. The immunohistochemistry included markers of epithelial and neuro-endocrine differentiation and showed intense expression of p63 by basal cells and cytokeratin 7 by luminal cells. In contrast, the prostate-specific-membrane antigen, the alpha-methylacyl-CoA-racemase (AMACR) enzyme and the neuro-endocrine markers (chromogranin and synaptophysin) were negative. A focal and non-specific expression of CD56 was noted.

The prostatic MRI realized 6 weeks after the TURP revealed a 41 × 30 × 15.3-mm tumoral lesion of the prostatic right peripheral zone with extra-capsular extension combined with right seminal vesicle invasion and a suggestion of posterior bladder wall adherence. The thoraco-abdominal CT scan showed no impact on the upper urinary tract and no visceral nor nodal metastases (Figs. 1, 2, 3).

Considering the tumor being locally advanced, the multidisciplinary board indicated a concurrent chemoradiotherapy(RT). The patient was treated in a prone position with six MV X-ray beams and an eight-field intensity-modulated technique with an empty rectum and a comfortably full bladder.

The total dose prescribed was 70 Gy, delivered in 35 fractions to the prostate and involved seminal vesical, 56 Gy on entire seminal vesicles and a total dose of 46 Gy on the pelvic nodes with weekly cisplatin. Then, the PSA level decreased to 0.4 ng/mL.

Eighteen months after his treatment, the PSA level was still under the upper limit of normal, and new CT scan showed that the patient was still on complete local response with the disappearance of the lower urinary tract symptoms, as long as the pain and the appearance of a prostatic atrophy.

However, he developed an intracranial hypertension. A brain MRI objectified a 3-cm unique cerebellar lesion.

The patient underwent metastasectomy with an ECOG of 1, and the anatomopathological study was in favor of a cerebellous metastasis of the known BCC. The patient had no evidence of disease after 3 months postoperatively.

### 3 Conclusions

The prostatic basal cell carcinoma is a rare aggressive entity often non-evoked at the clinical or radiological stages because of its unspecific appearance.

The diagnostic of these tumors is based on histological examination and a large immunohistochemistry panel. Given its scarcity, very few data is available for locally advanced non-metastatic stages treated by radiation therapy. We assess here a good local response with concurrent chemoradiation therapy.

### Abbreviations

ACC: Adenoid cell carcinoma; AMACR enzyme: Alpha-methylacyl-CoA-racemase; IHC: Immunohistochemistry; PSA: Prostate specific antigen; TURP: Transurethral resection of the prostate.

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### Authors' contributions

SR, CM, AL, HJ and AB are radiation oncologists who indicated, planned and monitored the radiation therapy; MR and FM are anatomopathologists who made the diagnosis; and all authors have read and approved the manuscript.

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### Declarations

### Ethics approval and consent to participate

NA.

### Consent for publication

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### Competing interests

No conflict of interest.

### Author details

<sup>1</sup>Centre Mohamed VI pour le traitement du cancer, Casablanca, Maroc. <sup>2</sup>Institut National d'Oncologie, Rabat, Maroc. <sup>3</sup>Centre Hospitalier Universitaire Ibn Rochd, Casablanca, Maroc.

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