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Case Report
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Squamous Cell Carcinoma of the Prostate: A Case Report

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Abstract

Squamous cell carcinoma of the prostate is a rare prostatic neoplasia, representing less than 1% of all cases. It is highly aggressive with poor prognosis. Its optimal treatment remains unknown, with little survival benefits on locally advanced or metastatic diseases. We report a case of metastatic primary squamous cell carcinoma in an 83-years-old man with a history of benign prostatic hypertrophy. The diagnosis was confirmed radiologically and histologically. After a treatment of Carboplatin-paclitaxel, a significant tumoral respond was seen at three months follow-up.

Introduction

Prostate gland carcinoma is the most common cancer of the male population, 95% of which being of adenocarcinoma histological type. Squamous cell carcinoma of the prostate is a rare entity, representing 0.5% to 1% of all prostate neoplasia. Even though prostatic adenocarcinoma and PSCC clinical manifestations are similar with lower urinary tract symptoms, they differ in their histological and biological aspect, treatment, and hormonal sensitivity. It is known for being of high malignancy with locally advanced or metastatic disease, and poor prognosis. While the vast majority of prostatic carcinoma are of glandular histological features, with defined treatment guidelines [1], less than 100 cases of PSCC have been reported in literature and optimal management is unknown [2]. Treatments are ranging from radical surgery to chemotherapy, radiotherapy, or chemo-radiation mostly with little survival benefits on locally advanced or metastatic diseases.

Case Presentation

In July 2020, an 83-year-old patient with a history of mainly cardiovascular diseases was referred to our hospital for an acute urinal retention caused by a presumed benign prostatic hypertrophy. The digital rectal examination was normal at the time, with an estimated 200 ml soft and even prostate gland. PSA level was 12.04 ng/ml, correlated to the gland volume. The patient was followed up in urology with several urinal catheter ablation failures and urinary tract infections. A digital prostatic examination was performed again in October 2020, disclosing an uneven swollen gland of stone-hard consistency, with multiples suspicious nodules. PSA level was then 14.62 ng/ml. Prostate MRI revealed an important prostatic hypertrophy (340 ml), with a postero-superior voluminous and irregular mass of the peripheral zone (70 mm) invading the bladder's postero-inferior wall, both seminal vesicles and likely the mid-rectum's anterior wall, with multiples iliac and retro-peritoneal enlarged lymph nodes. A Thorax, Abdominal and Pelvis scanner (CT-TAP scan) confirmed the irregular prostate tumor, infiltrating the bladder wall, peri-prostatic and peri-bladder soft tissues, as well as multiples precaval, lateral caval and retroperitoneal enlarged lymph nodes, with a dilated left UVJ likely due to distal obstruction by the advanced prostate tumor. FDG-PET/CT scan confirmed the prostate malignancy (SUV_{max}: 21.6) and the lymph nodes involvement, with no other evidence of metastatic disease. No bone or cerebral metastases were found on bone scintigraphy and cerebral MRI. The tumor was classified T4N1M1a. Transrectal prostate biopsies were performed. The biopsies revealed a mildly to poorly differentiated non-small cell eosinophil carcinoma with squamous differentiation with no glandular nor neuroendocrine expression on the histology and immunohistochemical analysis. The tumor staining was positive for epidermoid markers (p63, CK5/6) and for urothelial markers (CK7 mainly and CK20 focally, GATA3). Prostatic markers were negative (PSA, EGR, NKX3.1), as well as keratin expression. The tumor was therefore classified as an invasive squamous cell prostate carcinoma, even though the primitive or secondary prostatic origins could not be clearly determined. However, the blood measurements of NSE, chromogranin A and SCC were within normal range, whereas CYFRA 21-1 was elevated at 53.0 ng/ml. The patient was reviewed at our institution's multidisciplinary tumor board with recommendation of exclusive chemotherapy, considering the number of lymph node metastasis and the patient's comorbidities. The chemotherapy consisted of three cycles of Carboplatin-Paclitaxel every 21 days (Carboplatin AUC 2 and Paclitaxel 80.00 mg/m^2 on day 1, 8 and 15). The treatment was well tolerated except

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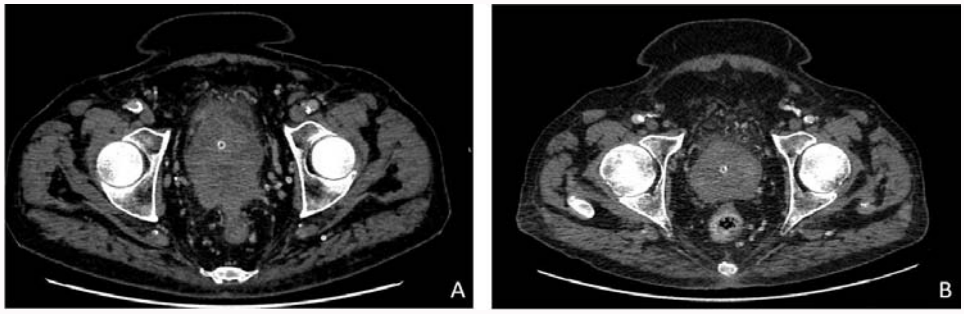

Figure 1: CT scanner of the prostate A) at diagnosis B) at three months post chemotherapy showing a reduction of the prostate tumor volume (20 mm *vs.* 70 mm) and loss of contact with the rectum's wall.

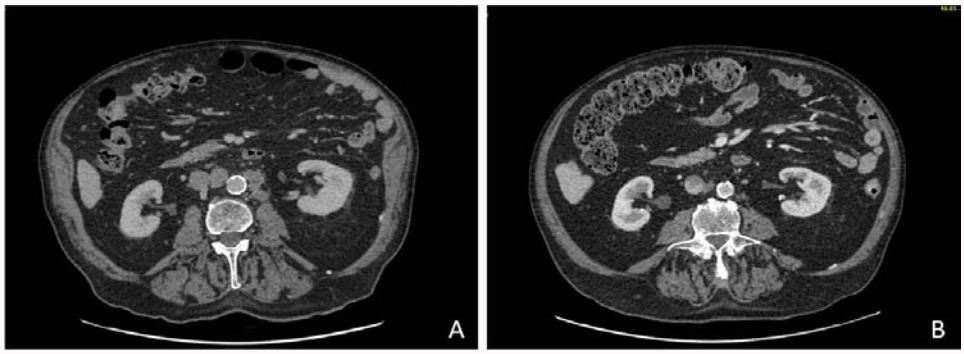

Figure 2: CT scanner of latero-caval lymph nodes A) at diagnosis B) at three months post chemotherapy showing the disappearance of the metastatic lymph nodes.

for a grade 3 sensitive neuropathy of both hands and feet (CTCAE v5), motivating the termination of the paclitaxel at day 15 of cycle 2. At the three months follow up in March 2021, the CT scan showed a significant respond with reduction of the prostate tumor volume (20 mm *vs.* 70 mm) (Figure 1), the loss of contact with the rectum's wall and the disappearance of all metastatic lymph nodes (Figure 2). No new metastasis had appeared. Considering the great treatment outcome at 3-month, chemotherapy was continued with 6 cycles of carboplatin every 21 days (AUC 5 on day 1).

Discussion

Squamous cell carcinoma of the prostate is a rare malignancy, accounting for less than 1% of all prostate tumors. Whereas the adenocarcinoma derivates from the glandular cells of the prostatic acini, PSCC origins are more controversial. Non-neoplastic squamous metaplasia is frequently seen associated with chronic inflammation such as chronic prostatitis or infarction, even though malignancy transformation remains rare. Some have postulated that its loss of PSA secretion and production of keratin reduces its likelihood of androgen blockade response [3]. Secondary squamous metaplasia of common adenocarcinoma has been reported, mainly several years post radiation treatment [4,5] or hormonotherapy [6]. Two main hypotheses arise concerning the primary PSCC origin: The basal or reserve cells of prostatic acini [7], or the transitional epithelium lining the urethra, and the major ducts Kahler and Thompson et al. [8]. It has also been supposed that it could derive from pluripotent stem cells. Mott proposed strict histological criteria for the diagnosis of primitive pure squamous cell carcinoma in 1979: 1) clearly malignant features including disorganized growth pattern, cellular anaplasia, invasion; 2) features of squamous differentiation including keratinization, presence of squamous pearls or distinct intercellular bridges; 3) lack of glandular/acinar component; 4) no prior estrogen therapy; 5) the absence of primary squamous cancer elsewhere [9]. These criteria could be improved with the use of immunohistochemistry even if no specific markers exist today. Squamous cell differentiation loses reactivity to prostatic specific antibodies such as PSA, PSAP, PSMA, P510S, AMACR [5]. SCC blood measurements can also be performed. Our case is a PSCC de novo with multiple metastatic lymph nodes, with no urologic history of prostatic adenocarcinoma, urothelial carcinoma nor hormonal or radiation treatment. The PSA level was elevated but correlated to the benign prostate hyperplasia component. The histological analysis showed no glandular or neuroendocrine component. However, it didn't show any morphological squamous differentiation, such as keratinization, due to its poorly differentiated nature. From immunohistochemistry, the tumor was marked negative for prostate differentiation (PSA, NKX3.1) and positive for squamous differentiation (p63, CK5/6) and for urothelial markers (CK7/CK20, GATA3). These results combined with an elevated CYFRA 21.1 blood measurement might indicate a possible urethra origin. Huang et al. have highlighted the interest of blood and urine cytokeratin 19 (CYFRA 21.1) measurement in bladder carcinoma in a recent meta-analysis [10]. Squamous cell carcinoma of the prostate is an aggressive disease, with a post diagnosis median survival reported of 14 months [11]. The age at diagnosis rates from 42 to 85 years, with common osteolytic bone, liver, and lung metastasis. Various therapeutic strategies have been tried throughout the years, ranging from surgery, to chemotherapy or radiation with seldom positive results on survival. Several reports have shown a possible long-term disease-free survival with low dose radiation combined with chemotherapy on local to locally advanced diseases, with a cisplatin and 5 fluorouracil combination and 5-year survival for Munoz et al. [2], and 21 months survival free for radiation

with IV bleomycin an intra arterial cisplatin for Uchibayashi et al. [12] other promising results were found in 2017 by Onoda et al. [11] with great tumoral response to DCF chemotherapy (5 FU, Docetaxel, and cisplatin) with radiotherapy. Few efficient treatments have been reported for PSCC with multiple metastatic lymph nodes, where radiation therapy cannot be performed. To our knowledge the use of combined Paclitaxel and Carboplatin for PSCC with these good response rates has not been previously reported. Carboplatin had been chosen over cisplatin due to our patient's acute obstructive kidney failure and other comorbidities. Our case suggests that this chemotherapy combination may be an effective treatment for the SCC of the prostate. Further investigations on taxane and platine in this indication should be undertaken. If our patient maintains his nodular and tumoral response at the follow up evaluation after the carboplatin chemotherapy, a closure treatment with radiotherapy of the prostate will be discussed, following Onoda et al findings. Platin and Radiation combination being a known effective treatment for squamous cell carcinoma of the head and neck [13], it could show encouraging results in another squamous cell carcinoma localization.

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