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Case Report

CEA As a Diagnostic and Monitoring Biomarker in PSA-Negative Sarcomatoid Prostate Carcinoma: A Case Report

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Abstract

Prostatic sarcomatoid carcinoma (PSC) is a rare and aggressive form of prostate cancer. Its diagnosis is often complicated by the absence of prostate-specific antigen (PSA) expression, a key biomarker commonly used for prostate cancer detection and monitoring. Even more unusual is the elevation of carcinoembryonic antigen (CEA), a biomarker typically associated with gastrointestinal cancers, which can contribute to delayed diagnosis.

We report the case of a 54-year-old man who presented with an isolated elevation in CEA (161 µg/L) during routine follow-up for ulcerative colitis. Initial gastrointestinal evaluations were unremarkable. Further urological assessment was conducted due to urinary complaints, revealing a prostate lesion despite normal PSA levels. Radical prostatectomy was subsequently performed, and final pathology identified a sarcomatoid carcinoma. Throughout follow-up, rising CEA levels consistently revealed multiple local recurrences and, eventually, lung metastases, whereas PSA remained within normal limits. Treatment with salvage radiotherapy, androgen deprivation therapy (ADT), and multiple chemotherapy regimens failed to control disease progression. A partial biochemical response was only achieved after the administration of carboplatin and cabazitaxel, with a significant decrease in CEA (from 303 to 158 µg/L) observed 54 months after diagnosis.

This case highlights the diagnostic and therapeutic challenges associated with PSC, particularly in the setting of normal PSA and elevated CEA levels. It also underscores the potential utility of alternative biomarkers such as CEA in disease monitoring, and emphasizes the need for further research to establish effective treatment strategies for this rare and aggressive malignancy.

Key Words: prostate cancer; carcinoembryonic antigen (CEA); prostate-specific antigen (PSA); sarcomatoid carcinoma; tumor marker; biomarker monitoring; case report

Introduction

Prostatic sarcomatoid carcinomas are extremely rare, accounting for less than 0.1% of all prostate malignancies [1,2]. PSC typically presents as a high-grade, biphasic tumor characterized by both epithelial and mesenchymal components, contributing to its aggressive clinical behavior and poor prognosis. Most patients are diagnosed at an advanced stage, usually following the onset of obstructive urinary symptoms [1,3]. About 25% of patients have distant metastases at the time of diagnosis, frequently involving the lungs, bones, and lymph nodes [4-6]. In case of localized disease, radical prostatectomy is the only therapeutic option shown to improve overall survival [7]. The levels of PSA, the most widely used prostatic tumor marker, are often normal in patients with PSC, complicating both diagnosis and follow-up [6,8,9]. This lack of PSA elevation is believed to be related to the sarcomatoid differentiation and loss of typical prostatic epithelial features [10]. In contrast, CEA, a glycoprotein commonly used as a tumor marker in colorectal and other adenocarcinomas, has been reported to be elevated in certain cases of prostate cancer, particularly in advanced, poorly differentiated, or androgen-independent tumors [11]. However, the clinical utility of CEA in PSC remains largely unexplored. Due to its rarity, PSC is poorly understood, with limited epidemiological data and no established clinical guidelines for diagnosis, treatment or monitoring. Current knowledge is largely based on small retrospective case series and individual case reports. Only a few cases of PSC with elevated CEA have

been reported in the literature, all at a metastatic stage [9,12–18]. We describe a case of localized PSC diagnosed by elevated CEA despite normal PSA levels.

Case Description

A 54-year-old Caucasian male with a history of ulcerative colitis, managed with mesalazine, was found to have an isolated CEA elevation to 161 µg/L during routine follow-up. He had no family history of malignancy, and systemic examination was unremarkable. A recent colonoscopy, performed three months earlier, and a gastroscopy carried out at the time of CEA elevation were both normal. Additional investigations, including a thyroid ultrasound and a thoracoabdominal CT scan, revealed no abnormalities. Due to the persistent CEA elevation, an FDG-PET-CT was performed, which showed focal hypermetabolism in the left hemiprostate. Notably, there were no corresponding abnormalities on the CT component and no other lesions were identified. The patient had been experiencing urinary symptoms, such as pollakiuria and nocturia, for over a year and was under follow-up for presumed overactive bladder. Given this context, a prostate MRI was conducted. PSA levels had consistently remained within normal range (most recently 0.38 ng/mL), and digital rectal examination was unremarkable. Multiparametric MRI revealed a hypointense T2 lesion involving approximately 300 degrees of the peripheral zone, predominantly in the left lobe. The lesion demonstrated diffusion restriction and was categorized as PIRADS 5. There was also suspicion of invasion into the anterior part of the left seminal vesicle. The prostate volume was estimated at 28 cc, and no pelvic lymphadenopathy was detected (*Figure 1*).

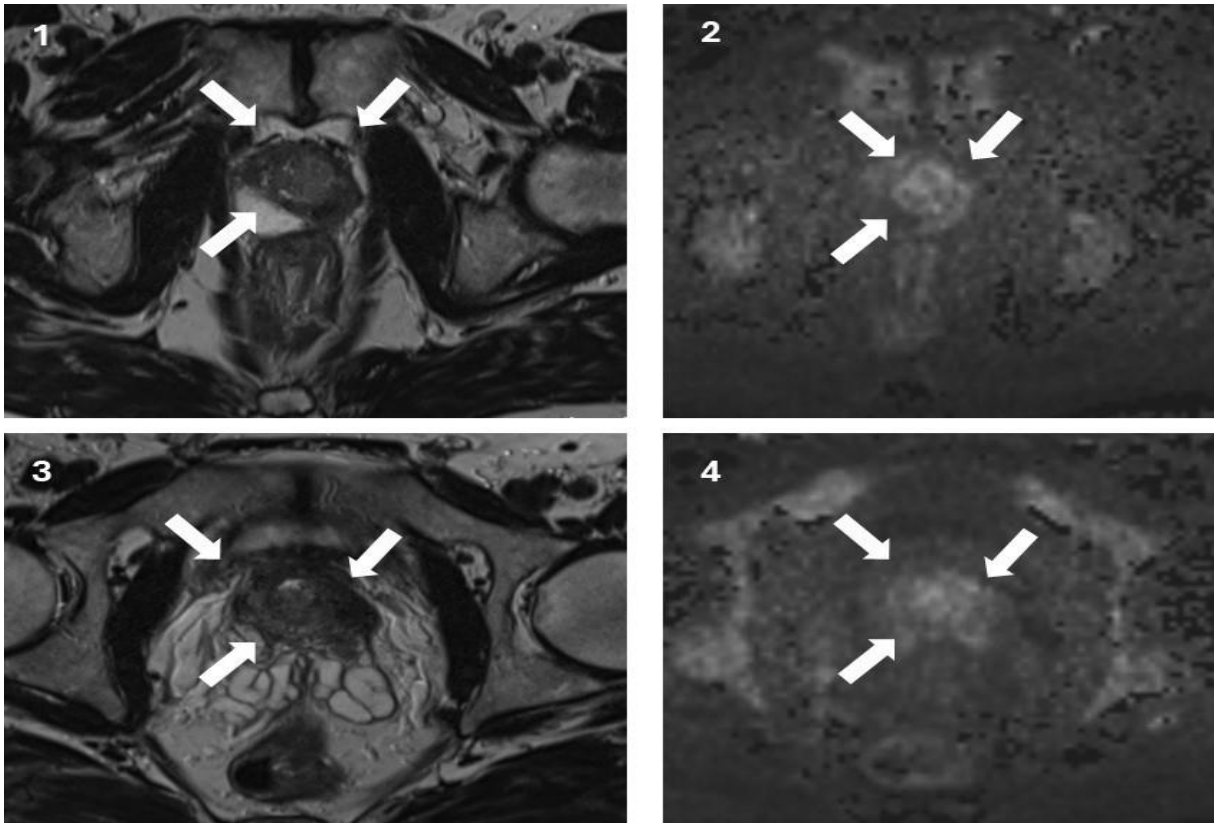

Figure 1 : Prostatic MRI image at the time of diagnosis. 1 & 3 : Axial T2-weighted sequence demonstrating a hypointense lesion (white arrows), involving most of the prostate and extending into the anterior portion of the left seminal vesicle. 2 & 4 : Axial diffusion-weighted imaging (DWI) revealing restricted diffusion within the lesion.

Targeted biopsies revealed a moderately differentiated adenocarcinoma in four out of five cores from the left lobe. In the right lobe, six cores were obtained, all showing prostatic parenchyma with atypical glands suspicious for malignancy. The immunohistochemical profile of the tumor (CK7+/CK20+) was not specific to a prostatic origin. Urothelial origin, as well as metastasis from the upper digestive tract or lungs, had to be excluded [19].

A radical prostatectomy with lymph node dissection was subsequently performed. Pathology showed a carcinoma with two distinct morphological and immunohistochemical aspects : a poorly differentiated high-grade acinar component with a Gleason score of 9 = 4+5 surrounded by a large sarcomatoid component. The immunohistochemical profile of the acinar component supported a prostatic origin, with strong NKX3.1 positivity, a marker highly specific for prostatic adenocarcinoma, and absence of GATA3 expression, which is typically seen in urothelial carcinomas, thereby ruling out transitional cell carcinoma. [20–22]. It massively infiltrated the entire prostate with its capsule, sparing the seminal vesicles. The surgical margins were positive at the body, neck and base. The five removed lymph nodes were not invaded. The final stage of the lesion was pT3a pN0 R1 (*Figure 2*).

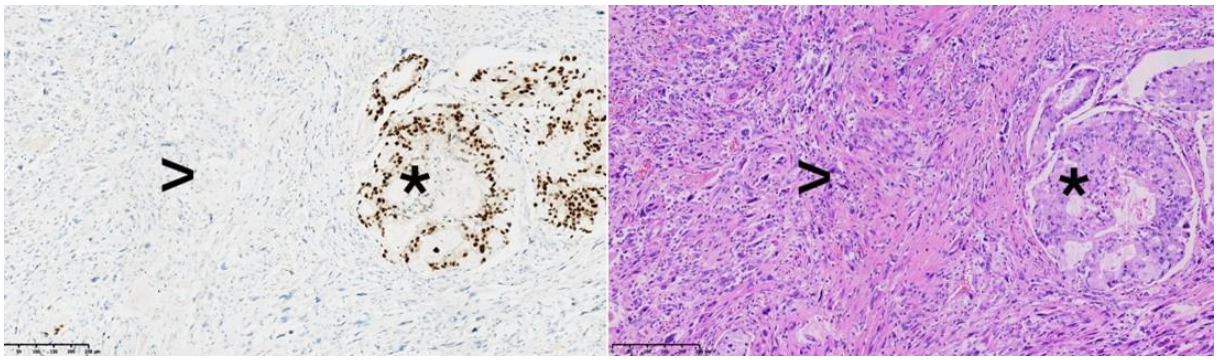

Figure 2 : Figure illustrating the two tumor components (NKX 3.1 stain on the left image, hematoxylin & eosin stain on the right image):
- The high-grade acinar tumor component (*) with fused and cribriform tumor glands, sometimes with comedonecrosis, stained with NKX 3.1.
- The sarcomatoid component (>) with pleomorphic cells, sometimes spindle-shaped or multinucleated, arranged in bundles, showing no immunostaining for NKX 3.1.

Postoperative CEA was measured at 6 µg/L, with undetectable PSA. Given the aggressive nature of the lesion, the primarily systemic rather than local risk of progression, and the patient's known ulcerative colitis, the multidisciplinary board proposed follow-up rather than adjuvant radiotherapy.

Four months after surgery, the CEA level rose to 14 µg/L, but the PSA level was still undetectable. A FDG-PET-CT revealed local recurrence at the base of the prostate bed.

We then performed salvage hypofractionated radiotherapy on the prostate bed (62.5 Gy in 25 fractions of 2.5 Gy, with a simultaneous integrated boost up to 67 Gy on the local recurrence) and pelvic lymph nodes (50 Gy in 25 fractions of 2 Gy) without ADT, given the normal serum PSA and the assumed androgen-independent nature of the primary tumor [23].

Radiotherapy was moderately tolerated, with grade 2 rectitis and anitis in the known context of ulcerative colitis, for which corticosteroid- and lidocaine-based suppositories were prescribed [24]. The CEA subsequently decreased to 7.7 µg/L.

Nine months after radiotherapy, the CEA level rose again to 19.6 µg/L, with FDG-PET-CT revealing focal hypermetabolism in the surgical bed at the level of the left seminal vesicle. PSA was still undetectable.

A rectal endoscopic ultrasound biopsy confirmed the recurrence of the prostatic sarcomatoid carcinoma, similar to the one initially described. There was no possibility of further irradiation or surgery. Therefore, tests for androgen receptors were added to the prostatectomy specimen and found to be positive, prompting treatment with bicalutamide 150mg per day [25,26].

CEA levels never decreased. Six months later, when the CEA level was 32 µg/L, a thoracoabdominal scan revealed left-sided hydronephrosis, indicating further local progression. As a result, gonadotropin-releasing hormone antagonist treatment (degarelix) was initiated [25,26] .

The CEA concentration continued to rise slowly, reaching 102 µg/L fifteen months later. At that time, small lung metastases were discovered, justifying the combination of enzalutamide (an androgen receptor inhibitor) and degarelix [27–29]. However, local symptoms, such as pelvic pain irradiating in the left foot, progressed. According to local progression, new biopsies were performed to detect additional mutations using Next Generation Sequencing. No pathogenic variants were identified, particularly no mutations in BRCA1 or BRCA2. Chemotherapy with docetaxel was therefore initiated but stopped after 5 cycles since the CEA level continued to increase to 147 µg/L [30–32] . Local symptoms also worsened, with abdominal pain linked to a mass in the left obturator fossa and a new CEA elevation of up to 303 µg/L (*Figure 3*).

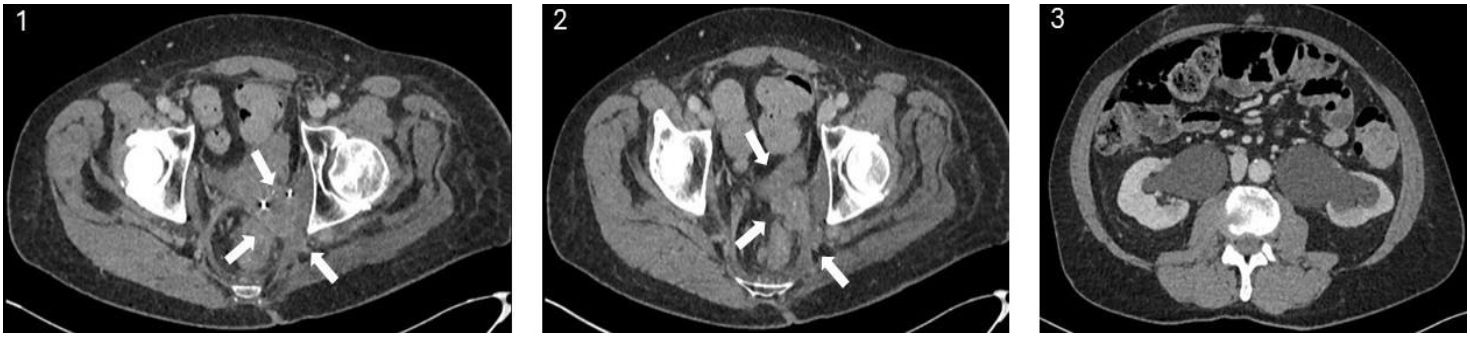

Figure 3 : CT-image showing the symptomatic mass in the left obturator fossa (lesion indicated by white arrows on images 1 & 2), causing bilateral hydronephrosis (image 3).

The patient was not eligible for treatment with lutetium-177PSMA, given the lack of PSMA expression of the tumor on PET-PSMA [33]. Chemotherapy regimens based on 5-fluorouracil combined with oxaliplatin or irinotecan, which are commonly used to treat gastrointestinal carcinomas, could be considered due to the persistent expression of CEA [34]. After discussion with the patient, treatment with carboplatin and cabazitaxel, a standard chemotherapy regimen for prostate cancer, was initiated, while the gastrointestinal chemotherapy regimen was retained for potential disease progression [35,36].

Currently, 54 months after the initial diagnosis, the patient remains alive and continues treatment with carboplatin and cabazitaxel. For the first time, the CEA level has decreased significantly, from 303 to 158 µg/L.

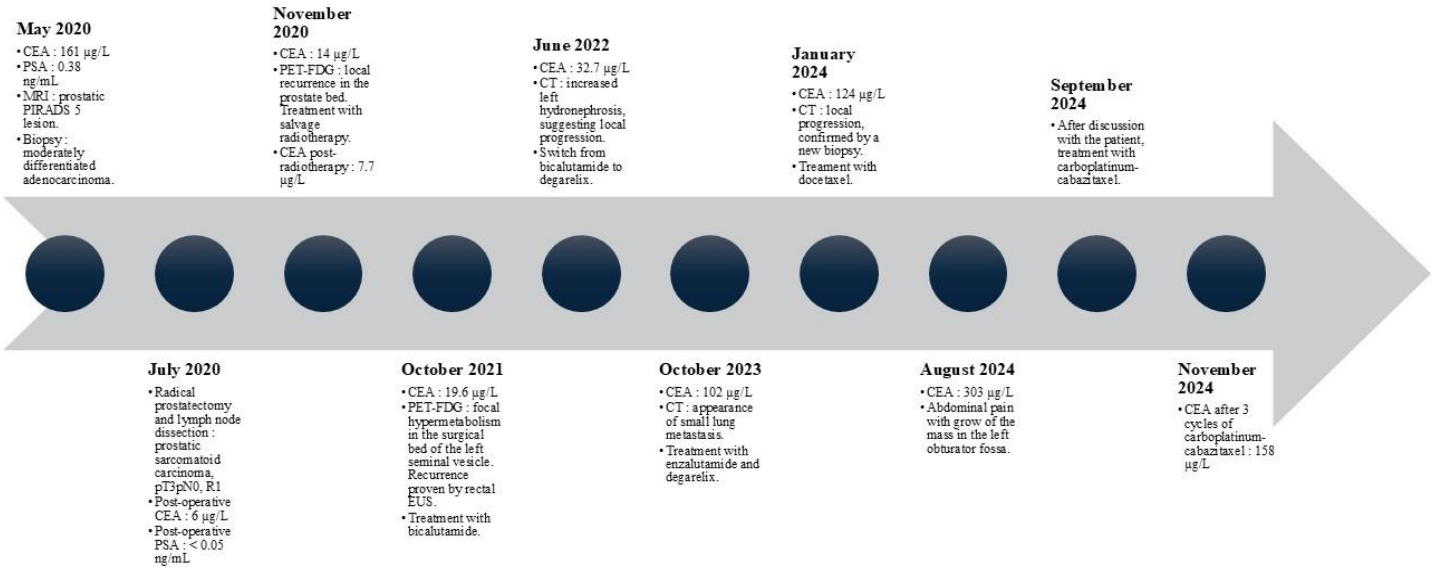

Figure 4 : Timeline illustrating the keypoints in the patient's case.

Discussion

Prostate cancer is the most common cancer in men, and the second leading cause of cancer-related mortality worldwide [12]. It is often diagnosed after a raised serum PSA screening test. The most common histological type is adenocarcinoma, which accounts for more than 90% of all prostatic malignancies[3]. On the other hand, prostate sarcomatoid carcinomas account for less than 0,1% of prostate cancers[1,2].

Clinically, PSC often manifests with lower urinary tract symptoms, particularly obstructive complaints [1,3]. It typically occurs in the sixth to seventh decade of life, making our 54-year-old patient notably younger than most reported cases [4]. These tumors are typically aggressive, often diagnosed at a locally advanced stage or already with distant metastases, and carry a poor prognosis [1,4,5], with nearly a quarter of patients presenting with metastatic disease at diagnosis [6]. However, in a retrospective case series from Johns Hopkins Hospital, among nine patients with localized disease, the median overall survival was not reached after a median follow-up of 106 months, and five patients survived beyond five years [8].

On MRI, prostate sarcomatoid carcinomas usually present as low signal masses on T1-weighted images and heterogeneous, intermediate to high signal masses on T2-weighted images, often with necrotic or cystic components. In contrast, our patient's lesion was hypointens on T2-weighted imaging. Contrast-enhanced MRI typically reveals heterogeneous or peripheral enhancement with non-enhancing necrotic or myxoid areas. Diffusion-weighted imaging commonly shows restricted diffusion [37].

Microscopically, PSC can consist of sarcomatoid cells only or display a biphasic composition, with the carcinomatous component containing an epithelial glandular structure and the sarcomatous component deriving from mesenchymal cells [3]. As observed in our patient, the epithelial component expressed NKX3.1 on immunohistochemistry, a novel and highly specific marker for prostatic adenocarcinoma. The presence of this marker confirmed the prostatic origin of the tumor. Additionally, the epithelial component may also express cytokeratins and PSA. The sarcomatoid component usually lacks some or all of these markers, making the diagnosis sometimes difficult when only the sarcomatoid component is present [10].

Serum PSA values are typically normal in PSC, as the undifferentiated tumor cells often lose glandular differentation and no longer produce PSA [6,38]. This lack of expression can complicate both diagnosis and follow-up [16]. CEA is a glycoprotein primarily found in gastrointestinal epithelium, but it is also present in other epithelial tissues, including the prostate [12]. Elevated serum CEA levels can occur in various malignancies—such as colorectal, breast, thyroid, and prostate cancers—as well as in benign conditions like liver disease and inflammatory bowel disorders [12,39]. Normal prostatic cells may produce CEA, and elevated levels are seen in 25–59% of prostate cancer cases, particularly in androgen-independent and poorly differentiated tumors [9,11,12,16,40,41]. Few case reports describe metastatic prostate cancers with high CEA but normal PSA levels [12–14]. In our case, the patient's elevated CEA was initially attributed to ulcerative colitis, delaying the diagnosis of PSC.

Therapeutically, there is no established consensus on optimal management of PSC. Radical surgery appears to be the only modality to improve overall survival in localized disease [7,42]. In our patient, radical prostatectomy was performed but with positive margins (R1), and local recurrence occurred despite salvage radiotherapy. However, a more extensive surgical approach might have prevented local recurrence, as illustrated in a case report by Fukawa et al., where pelvic exenteration was performed with no subsequent local recurrence or metastatic spread [42]. ADT is generally of limited efficacy in PSC, especially in tumors lacking PSA expression [5,8]. This aligns with our case, where the tumor expressed androgen receptors but showed no respons to bicalutamide, degarelix, or enzalutamide. Similarly,

Markowski et al. observed resistance to ADT in their PSC patient series, reinforcing its limited therapeutic value [8]. Moreover, some reports suggest that PSC may arise in adenocarcinomas previously treated with radiation or ADT [5,38,43,44]. Chemotherapy options for PSC have not been proven effective yet [4]. Regimens used for small cell carcinomas, sarcomas or classical prostate adenocarcinomas —such as etoposide, adriamycin and docetaxel —have shown only transient responses in the Johns Hopkins Hospital retrospective case series [8]. In our case, docetaxel was ineffective. However, partial biochemical response was observed following administration of carboplatin combined with cabazitaxel, the only platinum-containing chemotherapy regimen that is advised as a treatment option for metastatic castration-resistant prostate cancer (mCRPC)[35,36,45]. This supports further exploration of platinum-based chemotherapy in PSC, especially in tumors with atypical marker profiles. Given the persistent CEA elevation and partial response to platinum-based treatment, chemotherapy regimens typically used for gastrointestinal cancers, such as 5-fluorouracil combined with oxaliplatin or irinotecan, may warrant further consideration. Phase II trials evaluating oxaliplatin in combination with 5-fluorouracil or docetaxel have shown promising results in mCRPC [46,47]. Although irinotecan did not demonstrate benefit in a phase II trial [48], oxaliplatin-based combinations may still hold potential, especially in biomarker-selected populations.

This case highlights the potential value of CEA as a diagnostic and monitoring biomarker in PSA-negative prostate cancers such as PSC. Due to the rarity of these tumors, further research is challenging, making individual case reports important for improving clinical understanding and guiding management.

Conclusions

PSA testing is often ineffective for the diagnosis and monitoring of prostatic sarcomatoid carcinoma, as patients frequently have normal PSA levels. In such cases, CEA, commonly used to monitor colorectal cancer, should be considered as an alternative biomarker, as it can be elevated in certain prostate cancers, including PSC. Given the rarity of this tumor, further research and clinical reporting are essential to assess the value of CEA and guide treatment options. Radical surgery remains essential for achieving an accurate diagnosis and reducing the risk of recurrence, given that conventional systemic therapies for prostate cancer have shown limited efficacy. Additionally, gastrointestinal chemotherapy regimens may warrant investigation in PSC cases expressing CEA. Finally, to enhance the management of unusual prostate cancers such as PSC, the establishment of rare cancer registries or dedicated databases should be considered.

Abbreviations

ADT : androgen deprivation therapy

CEA : carcinoembryonic antigen

CT : computed tomography

FDG-PET-CT : 18-Fluorodeoxyglucose positron emission tomography

mCRPC : metastatic castration-resistant prostate cancer

PSA : prostate-specific antigen

PSC : prostatic sarcomatoid carcinoma

PSMA : prostate-specific membrane antigen

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Author's contributions :

All authors have contributed to the manuscript in significant ways, have reviewed and agreed upon the manuscript content.

Concept and initial idea: Tollet Aline, Van Ooteghem Geneviève

Writing and review of the article: Tollet Aline, Van Ooteghem Geneviève, Dano Hélène,

Revisions and final edits: Tollet Aline, Van Ooteghem Geneviève

Conflict of interest

The authors have no conflict of interest to report.

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