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*Case Report*

# Carcinosarcoma of the prostate in a patient with previous prostatic adenocarcinoma, status post brachytherapy

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**Abstract:** Carcinosarcoma of the prostate is an exceedingly rare form of prostate cancer. Prognosis is poor, and the observed biological behavior of these tumors is to be very aggressive irrespective of histologic variants. Some authors speculate that the increasing use of external beam radiation therapy and brachytherapy in the treatment of prostatic adenocarcinomas may raise the incidence of carcinosarcoma. We present the case of a 71 year-old Caucasian male with a history of prostatic adenocarcinoma treated with brachytherapy seven years ago. The patient developed progressive genitourinary symptoms leading to impotence and severe urinary obstruction. This was followed by cystoprostatectomy and incidental pathology diagnosis of carcinosarcoma of the prostate. Fluorescent in situ hybridization study for amplification of the *c-Myc* gene, frequently seen in radiation-induced sarcomas, was negative. Carcinosarcomas of the prostate show general similarities of clinicopathologic features regardless of histologic variants or etiology. Our case, as well as the accompanying review of the literature, highlights the complexity of diagnosis of prostatic carcinosarcoma, as well as its continued poor prognosis. Due to the limited number of available cases, it is not possible to conclude at this time whether or not there is a causative association of this tumor with prior radiation therapy of the prostate.

**Keywords:** Prostate, carcinosarcoma, prostatic adenocarcinoma, sarcomatoid carcinoma, radiation, brachytherapy, pathology

## Introduction

Primary carcinosarcoma of the prostate is a rare entity, representing approximately 0.1% of all prostatic neoplasms. Nearly half of the reported patients have a history of prostatic adenocarcinoma [1, 2], with the majority of these having received external beam radiation or brachytherapy [3, 4]. Serum levels of prostate specific antigen (PSA) are most often not elevated.

The differential histopathologic diagnosis may include a wide range of spindle cell lesions of the prostate, including stromal tumors of uncertain malignant potential, stromal sarcomas, and inflammatory myofibroblastic tumors. History of previous or ongoing radiation therapy may further confound the typical histopathologic features of a carcinosarcoma. Since patients are typically diagnosed at an advanced stage, prognosis is poor, and the biologic potential of these tumors is in general very aggressive irrespective of their histologic features [3, 5].

Given the limited quantity of reported cases, therapeutic regimens have not been optimized, and surgery remains the standard of care for therapy. It is hypothesized that radiation therapy may induce the development of carcinosarcomas of the prostate [3, 4] with the prediction of an increased incidence in the coming years.

## Case presentation

The patient is a 71 year-old Caucasian male with a history of adenocarcinoma of the prostate, Gleason grade 3 + 4 (score = 7), treated with brachytherapy seven years prior to presen-

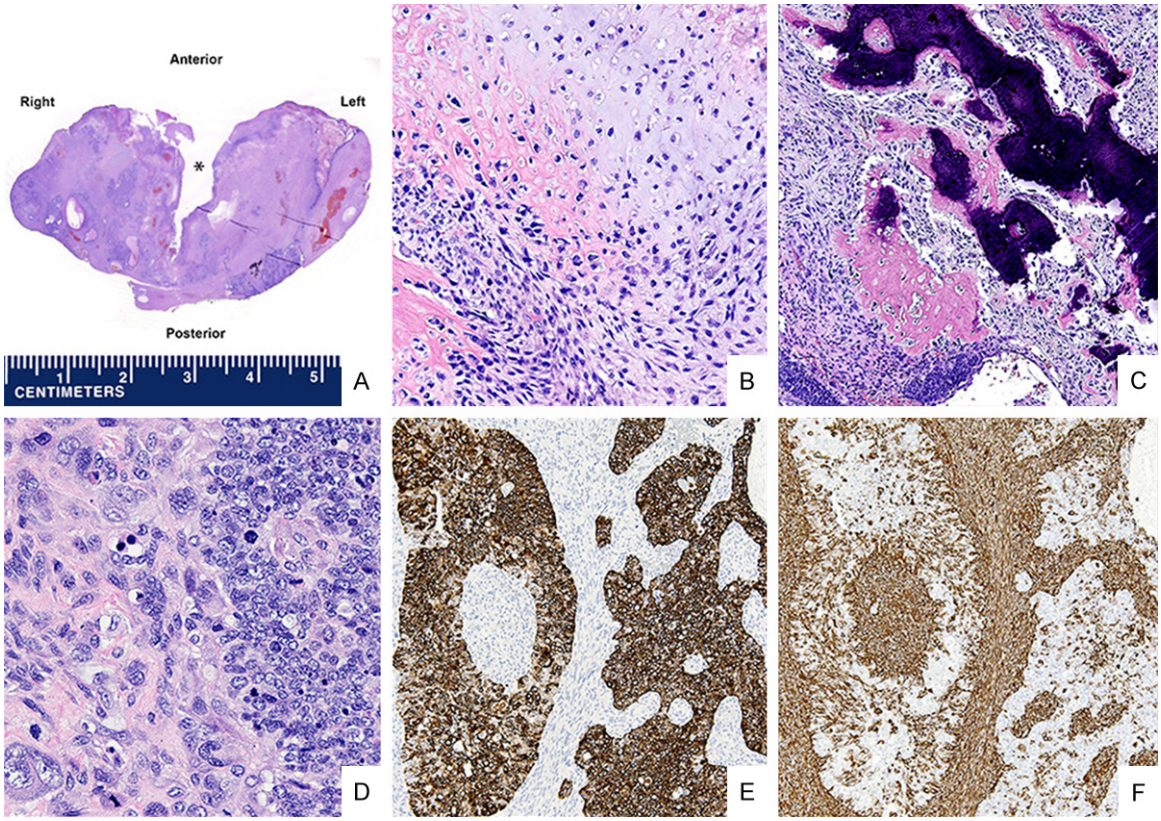


**Figure 1.** Histopathology of the carcinosarcoma of the prostate demonstrates the varying histopathologic components of this tumor. A. Hematoxylin-eosin stain of one of the whole mount transversal sections of the prostate (scanned), which has been opened at the anterior aspect, exhibiting a markedly enlarged prostate with irregular contour and a variegated appearance, and focal cystic formations and patchy necrosis; the asterisk (*) indicates the location of the prostatic urethra. Hematoxylin-eosin stain of the sarcomatous component shows: B. Chondrosarcoma tumor. C. Osteosarcoma tumor. D. Side to side, the sarcomatoid component at left and the carcinomatous component at right, show the corresponding cytological features for each tumor with marked anaplasia, increased nuclear/cytoplasmic ratio and high mitotic activity (40x objective). Special immunoperoxidase stains show: E. Positive staining for cytokeratin AE1/AE3 only in the carcinomatous component (this was similar for CK7 staining). F. In contrast, a consecutive section of the same area shown in E reveals the sarcomatoid component with positive staining for vimentin and no staining of the carcinomatous cells (B, C, E and F images are at 20x objective).

tation. Four years following initial treatment, the patient developed erectile dysfunction, and a penile prosthesis was placed. Two-and-a-half years later, the patient presented with complaints of increasing dysuria, urinary retention, fever, chills, abdominal distension, and gross hematuria. The serum PSA remained below detectable levels (< 0.01 ng/mL). A transurethral resection of the prostate was performed with no improvement in voiding. Insertion of a 16-gauge catheter was attempted, but was unsuccessful due to notable resistance at the prostatic urethra; for this reason, a permanent suprapubic catheter was implanted. A subsequent computerized tomography (CT) scan of the abdomen at an outside facility reported postoperative changes in the prostate and nonspecific bladder wall thickening, with a differential diagnosis of muscular hypertrophy from bladder outlet obstruction, cystitis, or an infiltrative process.

Cystoscopy performed two months prior to cystoprostatectomy revealed necrosis and stricture proximal to the bulbous urethra and extending across the membranous urethra into the prostate and bladder neck. Significant dystrophic calcifications were also noted. Biopsy samples from the right and left walls of the urinary bladder showed acute and chronic inflammation, bladder neck mucosa with marked radiation effect and necrosis, and a region of

**Table 1.** Immunohistochemical staining of prostatic carcinosarcoma: Current case report

| Antibody | Reactivity |
|---|---|
| Cytokeratin AE1-3 | POSITIVE (epithelial) |
| Cytokeratin 7 | POSITIVE (epithelial) |
| Vimentin | POSITIVE (mesenchymal) |
| MA903 | POSITIVE (epithelial, focal) |
| p63 | POSITIVE (epithelial & mesenchymal, focal) |
| Uroplakin | Negative |
| GATA3 | Negative |
| PSA | Negative |
| PAP/PSAP | Negative |
| Cytokeratin 20 | Negative |
| S-100 protein | Negative |
| SMA | Negative |
| Desmin | Negative |
| CD34 | Negative |

prostatic urethra with necrosis and bacterial overgrowth. All biopsy samples were interpreted as negative for malignancy.

A cystoprostatectomy with right colon pouch urinary diversion was performed, with 80% of the prostate removed, leaving adhesions of the posterior wall and seminal vesicles to avoid risk of rectal injury. This partial resection was chosen by the surgeon based on the previous pathology results of no evidence of a malignant process. The urinary bladder was submitted in two irregular fragments measuring 10.2 × 5.8 × 2.8 cm and 11.3 × 0.4 × 3.4 cm, with a total weight of 192 gm. The mucosal surface was tan-brown, velvety, edematous and focally erythematous. No mass or lesion was grossly identified. The prostate was 91.5 g, 7.0 × 3.6 × 6.4 cm, grey-tan and partially morcellated, making the specimen challenging to orient. No seminal vesicles were identified. Serial sections demonstrated a gray-white friable parenchyma.

Histopathological examination of hematoxylin and eosin (H&E)-stained sections of the urinary bladder demonstrated urothelial mucosa with changes consistent with radiation-induced cystitis; scant infiltrating nests of poorly differentiated malignant cells were present only in the muscularis propria of the bladder neck. The prostate was submitted in toto in whole mounts using 3 × 2 glass slides (**Figure 1A**). Sections of the prostate showed a variegated prostatic parenchyma completely replaced by a biphasic malignant tumor composed of irregularly admixed epithelial (carcinomatous) and mesenchymal (sarcomatous) components. The carcinomatous cells were in some areas forming cystic formations filled with amorphous eosinophilic material. A moderate amount of tumor necrosis was also present. The sarcomatous tumor showed chondrosarcoma (**Figure 1B**) and osteosarcoma (**Figure 1C**) elements and extensive areas of spindled, round and pleomorphic giant cells, with bizarre mitotic figures (**Figure 1D**). The carcinomatous component showed malignant epithelioid cells with marked anaplasia, increased nuclear/cytoplasmic ratio, high mitotic activity, and scattered karyorrhexis (**Figure 1D**). A comprehensive panel of immunohistochemical stains was performed and is listed in **Table 1**. The carcinomatous component had positive membrane and cytoplasmic staining for pan-cytokeratin AE1/AE3 (**Figure 1E**), and negative staining for vimentin (**Figure 1F**); in contrast, the sarcomatous component was negative for pan-cytokeratin AE1/AE3 (**Figure 1E**) and positive for membrane and cytoplasmic staining for vimentin (**Figure 1F**), clearly demonstrating the biphasic nature of this tumor. Immunostains also showed positive nuclear staining for p63 antigen and positive membrane and cytoplasmic staining for cytokeratin 7 (CK7) in the carcinomatoid cells, in correlation with their epithelial origin. Immunostains for uroplakin, GATA-3 (GATA binding protein 3 to DNA sequence [A/T] GATA[A/G]), prostate specific antigen (PSA), prostatic specific acid phosphatase (PSAP), smooth muscle actin (SMA), desmin, cytokeratin 20 (CK20), CD34 and S100 were uniformly negative in different areas of the tumor. This immuno marker profile demonstrated the general identification of mesenchymal (vimentin) and epithelial (pan-cytokeratin, CK7 and p63) components. However, this profile did not reveal a more specific identification of tumor origin, such as muscle (negative SMA and desmin), urothelium (negative GATA3 and uroplakin) and prostatic glands (negative PSA and PSAP); other more ubiquitous cell markers were also negative such as CK20, CD34 and S100. Inked surgical margins from the prostatectomy specimen were diffusely positive for tumor involvement.

**Table 2.** Clinical features of prostatic carcinosarcomas: Literature review Patients are clustered in the seventh and eighth decades of life. More than half presented with prior prostatic adenocarcinoma. The majority of those with a history of prior treatment received hormone therapy, external beam radiation, or brachytherapy. The mean time between diagnoses of adenocarcinoma and carcinosarcoma is 4.5 years

| Clinical Features | Data |
|---|---|
| Age (Average Range) | 68.2 y (32-91 y) |
| Carcinosarcomas with prior prostatic adenocarcinoma | 60% (63/105) |
| | pre-1996: 21/63 |
| | post-1996: 42/63 |
| *Acinar* | 96% (22/23) |
| Well differentiated (Gleason score 6) | 32% (7/22) |
| Moderately differentiated (Gleason score 7) | 5% (2/22) |
| Poorly differentiated (Gleason scores 8, 9, 10) | 59% (13/22) |
| *Ductal* | 4% (1/23) |
| *De novo* | 40% (42/105) |
| | pre-1996: 22/42 |
| | post-1996: 20/42 |
| Prior Therapy for Prostatic Adenocarcinoma (Total 55) | |
| Hormone therapy | 62% (34) |
| External beam radiotherapy | 47% (26) |
| TURP | 20% (11) |
| Orchiectomy | 13% (7) |
| Brachytherapy | 11% (6) |
| Other (Radical prostatectomy, chemotherapy, none) | 2-4% (1-2) |
| Time to Diagnosis of Carcinosarcoma (Average [Range]) | 4.5 years (2 mos.-16 y) |
| PSA (serum) at Diagnosis of Prostatic Carcinosarcoma (Total 17) | |
| Elevated | 29% (5) |
| Normal | 71% (12) |

Representative tissue sections were further evaluated for amplification of the *c-Myc* gene by fluorescent *in situ* hybridization assay performed at the Colorado Genetics Laboratory, University of Colorado, Anschutz Medical Campus. Briefly, a dual color chromosome assay for interphase cells was performed on formalin-fixed, paraffin-embedded prostate tissue that was pretreated with Proteinase K and hybridized with a Vysis (Abbott Laboratories) probe for *c-Myc* (8q24). Two areas were marked for analysis on the accompanying H&E stained section. Both areas were analyzed, with similar results. There was no evidence of high-level amplification of *c-Myc* (8q24), greater than 10 copies. Polysomy for *c-Myc* sequences was observed in both areas, with the major populations of cells having 3-4 copies of *c-Myc* (41.8%). A few cells with five (3 cells, 2.9%) and seven (one cell, 1.0%) copies of *c-Myc* were observed as well. Of note, high level *c-Myc* gene amplification has been associated with sarcomas, particularly angiosarcomas arising after radiation therapy [6].

Following cystoprostatectomy, the patient was staged by physical examination and imaging analysis without evidence of metastatic progression and was initiated on radiation therapy of the prostatic bed and weekly doses of paclitaxel. A follow-up CT scan of the thorax at six months after surgery revealed a new 6 mm sub-pleural nodule in left upper lobe and an increase in size of a fissure-based 6 mm nodule in left upper lobe, both consistent with metastatic disease. A nuclear medicine bone scan showed no evidence of bone metastasis. Since the pulmonary nodules were too small, a biopsy procedure was deferred at that time. Given that the patient was relatively asymptomatic from the pulmonary nodules, the patient was scheduled for stereotactic body radiotherapy, with

**Table 3.** Histologic features of prostatic carcinosarcoma: Literature review Prostatic carcinosarcomas demonstrate carcinomatous (epithelial) and sarcomatous (mesenchymal) elements, with heterologous components identified in over half of cases. Carcinomatous components are positive for conventional epithelial markers. Sarcomatous components reveal near-universal vimentin staining, with one-half to two-thirds of lesions also staining for muscle, neural, and endothelial markers. A subset of carcinosarcomas show staining of the sarcomatous component with PSAP, a finding consistent with recent studies demonstrating the monoclonal epithelial origin of a series of prostatic carcinosarcomas

| Histologic features | Data |
|---|---|
| Tumor Type | |
| *Carcinoma* (Total 73) | |
| Acinar | 77% (56) |
| Ductal | 20% (15) |
| Squamos. us | 15% (11) |
| Neuroendocrine | 5% (4) |
| Other (Basaloid, Papillary, Foamy micropapillary, Enteric, Urothelial) | 1-3% (1-2) |
| Undifferentiated | 3% (2) |
| *Sarcoma* | |
| Heterologous elements | |
| Present (Total 60) | 58% (60/104) |
| Osteosarcoma | 55% (33/60) |
| Chondrosarcoma | 40% (24/60) |
| Rhabdomyosarcoma | 13% (8/60) |
| Leiomyosarcoma | 13% (8/60) |
| Other (fibrosarcoma, “MFH-like,” angiosarcoma, liposarcoma) | 13% (8/60) |
| *Absent* | 42% (44/104) |
| Immunohistochemistry | |
| *Carcinoma* | |
| Pan-cytokeratin (CK AE1/AE3); Cam 5.2; EMA | 100% (14/14; 5/5; 8/8) |
| p53 | 100% (3/3) |
| CEA | 80% (4/5) |
| PSA | 65% (11/17) |
| PSAP/PAP | 63% (7/11) |
| Synaptophysin; Chromogranin | 50%; 33% (1/2; 2/6) |
| Vimentin | 11% (1/9) |
| Desmin; Myoglobin. SMA; MSA; CD31; CD34; S-100 protein | 0% (0/4; 0/1; 0/1; 0/2; 0/2; 0/2; 0/2) |
| *Sarcoma* | |
| p53 | 100% (5/5) |
| Vimentin | 95% (19/20) |
| MSA; SMA | 67%; 60% (4/6; 6/10) |
| S-100 protein | 54% (7/13) |
| CD31/CD34 | 50%, 0% (1/2; 0/3) |
| Pan-cytokeratin (CKAE1/AE3); Cam 5.2; EMA | 44%; 100%; 25% (8/18; 1/1; 2/8) |
| Desmin; Myoglobin; MyoD1 | 23%; 50%; 100% (3/13; 1/2; 1/1) |
| PSAP/PAP | 23% (3/13) |
| Synaptophysin; Chromogranin | 0% (0/2; 0/2) |
| PSA | 0% (0/19) |

delay of chemotherapy as long as was feasible. At twelve months after surgery, the patient is still alive, with no evidence of residual pelvic malignancy, but evident growth of the known untreated lung lesions, with no new lesions identified.

**Table 4.** Clinical outcomes for prostatic carcinosarcomas: Metastatic involvement by prostatic carcinosarcoma is seen in approximately half of cases, with overall survival averaging slightly less than two years in patients with continuous follow-up

| Clinical Outcome | Data |
|---|---|
| Metastasis (Total 106) | |
| Regional lymph nodes (only) | 5-8% (5-8) |
| Distant metastasis | 45-48% (48-51) |
| Lung | 53% (26/48-51) |
| Spine/Other bone | 51% (25/48-51) |
| Liver | 30% (15/48-51) |
| Other (Brain, adrenal, pleura, pancreas, spleen, skin) | 2-10% (1-5/48-51) |
| None | 47% (50) |
| Prostatic Carcinosarcoma Treatment (Total 80) | |
| TURP | 44% (35) |
| External beam radiotherapy | 33% (26) |
| Chemotherapy | 26% (21) |
| Radical cystectomy/cystoprostatectomy/prostatectomy | 24% (19) |
| Orchiectomy | 23% (18) |
| Hormos.ne therapy | 23% (18) |
| Total pelvic exenteration | 9% (7) |
| Brachytherapy | 5% (4) |
| Other (debulking, supportive care) | 1% (1) |
| Overall Survival | |
| (Average [Range]) | 23 mos. (2-107 mos.) |
| 6 mos. | 74% (37/50) |
| 1 y | 44% (22/50) |
| 3 y | 28% (14/50) |
| 5 y | 18% (9/50) |

### Review of the literature

A review of the English language medical literature performed on PubMed including all dates, utilizing the keywords “carcinosarcoma” or “sarcomatoid carcinoma” and “prostate”, without exclusions, revealed 39 articles with case reports and case series. Including this case report, there are a total of 119 reported cases of primary prostatic carcinosarcoma. The most relevant review articles are presented in the Reference section.

Statistical analysis of unpaired clinical data sets for patients receiving radiation-based therapies versus other therapeutic interventions prior to diagnosis with prostatic carcinosarcoma was performed using the Student *t* test. Clinical outcomes were compared using Kaplan-Meier curves analyzed by the Mantel-Cox log rank and Gehan-Breslow-Wilcoxon tests (GraphPad Prism 6, LaJolla, CA).

Consistent with prior reports, patients were clustered in the seventh and eighth decades of life. More than half presented with a prior adenocarcinoma of the prostate (**Table 2**). Among those having received treatment for prostate cancer, the majority received hormone therapy, external beam radiation, or brachytherapy. The time to diagnosis of subsequent prostatic carcinosarcoma averaged 4.5 years.

Microscopically, prostatic carcinosarcomas demonstrated the definitional mix of carcinomatous (epithelial) and sarcomatous (mesenchymal) elements, with heterologous components, predominantly osteoid and chondroid, being identified in over half of cases (**Table 3**). Immunohistochemical stains showed the carcinomatous components to be positive for conventional epithelial markers such as cytokeratin AE1/AE3, cytokeratin CAM 5.2 and epithelial membrane antigen (EMA), and, in a small cohort of samples, to overexpress the p53 anti-

**Table 5.** Prostatic Carcinosarcoma with or without prior radiotherapy: Time to diagnosis is longer in radiation-associated prostatic carcinosarcomas. Other clinicopathologic parameters, including age of diagnosis, presence of metastasis, and overall survival, are not significantly different

| | Radiotherapy (N = 13) | No Radiation (N = 22) | *P* value |
|---|---|---|---|
| Age (Average [Range]) | 67.1 y (50-84) | 69.3 y (59-89) | 0.47 |
| Time to Diagnosis (Average [Range]) | 61.9 mos. (4-180 mos.) | 28.3 mos. (2-73 mos.) | 0.02 |
| Metastasis | | | 1.0 |
| Regional or Distant | 66% (6/9) | 66% (14/21) | |
| None | 33% (3/9) | 33% (7/21) | |
| Overall survival | 38.6 mos. (6-96) | 25.3 mos. (3-107) | 0.40 |
| 6 mos. | 100% (6/6) | 73% (11/15) | |
| 1 year | 50% (3/6) | 47% (7/15) | |
| 3 years | 50% (3/6) | 26% (4/15) | |
| 5 years | 33% (2/6) | 13% (2/15) | |

gen. PSA and PSAP stains were positive in approximately two-thirds of epithelial components. The carcinomatous components were typically negative for vimentin and for markers for muscular, neural, and angiogenic differentiation. By contrast, the sarcomatous components revealed near universal vimentin staining, with one-half to two-thirds of lesions also staining for muscle, neural, and endothelial markers.

Of note, a smaller subset of approximately one-quarter of carcinosarcomas showed staining of the sarcomatous component with PSAP. Of note, a recent study by Rodrigues *et al.* investigated the observation that some epithelial markers were expressed by the sarcomatous component of some carcinosarcomas, acknowledging that it was unclear whether the sarcomatous component was a second primary, potentially radiation-induced, or had evolved from the same epithelial origin as the acinar adenocarcinoma. After FISH analysis of the erythroblast-transformation-specific related gene (*ERG*), in a series of prostatic carcinosarcomas, the group demonstrated that the sarcomatous and the adenocarcinoma components shared a clonal genomic aberration that confirmed their common origin [7].

Clinical outcome data demonstrated metastatic involvement by prostatic carcinosarcomas in approximately half of cases, with overall survival averaging slightly less than two years in patients with continuous follow-up (**Table 4**). In these patients, one-year overall survival was less than 50%, and five-year survival was seen in fewer than one-in-five (20%), further establishing the poor prognosis of this tumor.

Since the first prostatic carcinosarcoma after radiotherapy was reported in 2006 [3], a total of 13 have been reported. In the interest of further evaluating the hypothesized etiologic connection between prior radiation therapy and subsequent prostatic carcinosarcoma, we divided data sets from the prostatic carcinosarcoma literature into (1) prior radiation therapy cases (external beam radiation and brachytherapy) and (2) any other therapy categories (**Table 5**). Time to diagnosis was longer in radiation-associated prostatic carcinosarcomas. Other clinicopathologic parameters, including age of diagnosis, presence of metastases, and overall survival, however, were not significantly different. *c-Myc* gene amplification data were not available from cases in the literature for comparison.

## Discussion

Primary carcinosarcoma of the prostate is an exceedingly rare entity. Clinical and pathologic recognition of this entity are paramount to effective early diagnosis and treatment. Because conventional chemotherapy and hormonal therapy regimens used for prostatic adenocarcinoma and other prostatic sarcomas are generally ineffective for carcinosarcomas of the prostate, aggressive surgical management remains the standard of care. Early diagnosis and treatment may offer the promise of improved outcome in these patients.

The case presented here illustrates the challenges to early diagnosis of primary carcinosarcoma of the prostate. In spite of a prior history of prostatic adenocarcinoma, serum PSA levels remained undetectable. Early symptoms were non-specific and special imaging, endoscopic, and histopathologic studies were inconclusive for cancer. CT imaging reported postoperative changes in the prostate and bladder. Cystoscopy demonstrated significant dystrophic calcification of the prostate and bladder neck, with focal necrosis. It was only when the cystoprostatectomy specimen was examined that the classical biphasic pattern of carcinosarcoma was readily identified and the diagnosis of primary carcinosarcoma of the prostate was rendered.

Our review of the current literature underscores the rarity of primary carcinosarcoma of the prostate. The prevalence of case reports appears to be gradually increasing with time, though whether this phenomenon is due to the increased utilization of therapeutic protocols hypothesized to play a causative role in the development of carcinosarcomas [3] or, instead, merely to an increased recognition and reporting of this rare diagnostic entity, is unclear from the scope of this review. Notwithstanding, from our review and evaluation of the literature, we found that all carcinosarcomas of the prostate, once diagnosed, regardless of their etiology - spontaneous, after conventional therapy or after radiation therapy-appear to behave in a similar manner, with frequent metastasis and poor outcome.

As a closing point, it bears mention that this is the first case of carcinosarcoma of the prostate following brachytherapy from which a study for *c-Myc* gene amplification is reported. Interestingly, the lack of *c-Myc* gene amplification in this case suggests a potential divergence from the literature reports for radiation-induced sarcomas arising in other organs, which have shown a significant minority of cases to harbor high level *c-Myc* gene amplifications [8, 9]. Further molecular studies of a larger cohort of prostatic carcinosarcomas would be merited to better understand the generalizability and implications of these findings.

## Conclusions

Primary carcinosarcoma of the prostate is a very rare and extremely aggressive form of prostate cancer. We present here a case of an elderly male with a prior history of prostatic adenocarcinoma and subsequent brachytherapy treatment, presenting with obstructive urinary symptoms and no elevation of serum PSA. Our study for *c-Myc* gene amplification, a feature frequently seen in radiation-induced sarcomas in other organs, was negative in this tumor. A review of the literature highlights the complexity of diagnosis of prostatic carcinosarcoma, as well as its continued poor prognosis. Data suggest that therapy-related prostatic carcinosarcomas behave in a similar manner, regardless of their radiation-associated etiologies.

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## Disclosure of conflict of interest

None.

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