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Case Report
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Prostate Cancer in a 63-year Old Man: A Case Report

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Abstract

Prostate cancer is the emergence of our century's scourge -cancer- in the prostate, which is a gland of man's reproductive system. Prostate is an of the essence organ in the spectrum of sexual reproduction and urinary system's normal function.

Worldwide, prostate cancer is the second most usual type of cancer in men (after skin cancer) [1] and meanwhile the eighth most lethal cancer type, thus appears to be the eighth most frequent cancer-related death (with a pool of both sexes) [2]. Additionally it has a strong tendency to emerge in men who count over 50 years of life and as a result it is unusual for a man to present with cancer in his thirties or forties.

This type of cancer has the life-threatening potential to spread to other tissues (via metastasis), to be more specific in bone, causing unique osteoblastic tumors (bone forming) [3].

We can stress 3 factors that increase the probability of developing prostate cancer. These are: being over 65 years old, having a specific family history (of developing prostate cancer) and finally being African-American [4].

Some primary symptoms of this terminal disease are problems during urination (pain or difficulty to stop the urine stream), lower back pain (due to metastasis in vertebrae) and painful ejaculation (because prostate gland secretes fluid for semen) [4].

This article aims at an extended presentation of this specific disease by highlighting some fundamental aspects of it. This goal will be facilitated by a study of 63 years-old man suffering from prostate cancer.

Keywords: Prostate Cancer; Prostate Gland; Metastasis; Prostate Specific Antigen Carcinosarcoma

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Introduction

Epidemiology

A fact worth mentioning is that when it comes to American men prostate cancer is the second leading cause of cancer-related death. To elaborate on the situation, within 18 years (between 1976 and 1994) prostate cancer incidents doubled and in addition the disease seems to have become more fatal as the increase in mortality rates by 20% indicates. The increase of incidents seems to be a result of increasing efforts to detect the disease. The development of the prostate-specific antigen (PSA) testing (through blood count) has contributed significantly to an easier detection of the malignancy. For younger than 70 years men, incidence's increase is more noticeable as the percentage has soared from 38% to 47% in only 7 years, between 1986 and 1993 [5].

Despite the large percentage and the high incidence of this malignancy, only 10% of patients die from the disease. Globally, researches indicate that among men of 70 to 79 years old 39% of them suffer from prostate cancer and this percentage increases to 43% in men of 80 years old. In the US during 2006, there were 234,460 prostate cancer cases and 27,350 deaths. Generally, incidence and mortality of prostate cancer present with a high rate of variability between different countries. In the United Kingdom every year 35,000 new patients are detected and 10,000 die from prostate cancer. American cancer society in 2012 reported 241,740 new cases of prostate cancer. Some other studies show that in Asia mortality rate has increased significantly in last years [6].

Furthermore, China has one of the lowest incident levels and additionally prostate cancer prevalence is lower in men of Mediterranean origin. Many researchers attribute this fact to the

consumption of many foods which dominate Mediterranean diet and contain antioxidants (which keep cancer risk at bay) such as olive oil, cereals and vegetables. The highest incidence of the disease is found in American men of African and Caucasian origin [7].

Genetics

Cancers result from the accumulation of genetic mutations in critical genes, especially those responsible for cell growth as well as division or repair of damaged DNA. Those specific alterations in the genes allow the uncontrollable growth/division of cells and thus the formation of tumors. In a plethora of cases of prostate cancer these changes are acquired during a man's lifespan and primarily present in specific cells of the prostatic gland. It needs to be stressed out that these so called somatic mutations are not inherited [8].

On the other hand, with an inherited risk of approximately 60%, prostate cancer is construed as a highly heritable disease. A percentage of 5% to 10% cases are thought to be mainly caused by certain hereditary genetic factors. The conclusion of large-scale studies reveals a linkage between family history and the appearance of prostate cancer in a patient. A family history of a first degree relative with prostate cancer is a sign of risk for this certain disease. The age of the affected relative is inversely related to the risk [9].

Inherited mutations in certain genes, such as *BRCA1*, *BRCA2*, and *HOXB13*, are responsible for some cases of prostate cancer. Patients presenting with these genes are at higher risk of having prostate cancer and possibly other types of cancer. Furthermore men with *BRCA2* or *HOXB13* gene mutations are potentially more prone to developing life-threatening forms of prostate cancer.

BRCA1 and *BRCA2* genes play a predominant role in fixing damaged DNA by producing certain proteins, which in turn aids the maintenance of cell's stability as far as its genetic information is concerned. Therefore, the *BRCA1* and *BRCA2* proteins are regarded to be tumor suppressors, meaning they inhibit cells from dividing or growing uncontrollably. Mutations in these genes deter the cell from fixing damaged DNA and as a result defects, potentially hazardous, remain. As these defects accumulate, they can trigger cells to grow and divide uncontrollably and form a tumor.

The *HOXB13* gene accounts for the production of a protein that binds itself to specific regions of DNA and regulates the activity of other genes. On the basis of this role, the protein produced from the *HOXB13* gene is called a transcription factor. *HOXB13* gene mutations are likely to result in the impairment of the protein's tumor suppressor function, resulting in the uncontrolled cell growth and division that can lead to prostate cancer. Mutations in these genes probably make only a small contribution to the overall prostate cancer risk Alas, researchers suppose that the combined influence of variations in many of these genes may increase a person's risk of developing this form of cancer [8].

Pathophysiology-Pathology

The pathophysiology of prostate cancer is poorly understood and little investigation has been focusing on it so far. Over the past decade, an increase in prostate cancer research, with a concomitant increase in funding for basic investigation has presented itself [10].

The prostate gland, part of the male reproductive system, is responsible for making and storing seminal fluid. Its typical size is 3cm long weighing 20gr. The prostate contains small glands which make 20 percent of the fluid constituting semen. Prostate cancer is classified as an adenocarcinoma, or glandular cancer, that is brought about when normal prostate gland cells mutate into cancer cells. The most common place for an adenocarcinoma to occur is the peripheral zone. Lumps of cancer cells are confined to otherwise normal prostate glands at first, a condition known as carcinoma in situ or prostate intraepithelial neoplasia (PIN). Subsequently, these cancer cells begin to multiply and spread to the surrounding prostate tissue resulting in the formation of a tumor. Eventually, the tumor may lead to metastasis meaning the invasion of nearby organs or tissues. Prostate cancer most commonly metastasizes to the bones, lymph nodes, and may invade rectum and bladder [11].

Clinical and morpholigical characteristics

Carcinosarcoma is exceedingly rare and generally presents after the age of 40 years. The mean age at diagnosis was 66 years among all published cases.Most patients are ≈70 years old (range 50–89) and present with urinary tract obstruction. In the largest published series on prostatic carcinosarcoma, Dundore *et al.* found that, among their 21 patients, 48% had a previous diagnosis of prostatic adenocarcinoma. The mean time to development of carcinosarcoma was 33 months (range 2–73 months). The presenting symptom, as stated above, was urinary obstruction in 85% of patients [12,13].

Clinically, most patients present with both filling symptoms (frequency, urgency, dysuria) and voiding symptoms (poor stream, hesitancy, terminal dribbling and incomplete voiding). However, most patients present with obstructive symptoms (poor stream, hesitancy, terminal dribbling and incomplete voiding) whereas symptoms of frequency, urgency and dysuria, are less common.The tumors produce bladder outlet obstruction and often require repeated TURs to control local symptoms. Less frequent manifestations are hematuria, hemospermia, pelvic, perineal and/or rectal pain (the pain may be localized, referred or both) and burning on ejaculation. Constipation and constitutional symptoms such as weight loss may also present. Tumor spread may occur by direct extension, hematogenous, lymphatic or perineural invasion [14-16]. Other symptoms associated with prostatic sarcomas include suprapubic or lower abdominal mass as well as resilient mass during rectal examination. Increased PSA is found in half of the patients. Nocturia, tenesmus and stranguria have also been noticed. Lastly, the patient may be presented with digestive disorders, oedema of the lower limbs and an alteration of the general status [13,17,18].

Physical examination reveals enlargement of the prostate. The tumor presents as a mass that may be rapidly enlarging. On DRE, the prostate is enlarged, nodular and hard.Elevation of prostate specific antigen (38.0ng/ml). Prostate surface can be smooth and elastic hard on digital rectal examination [12,14,19].

Microscopically, the carcinomatous and sarcomatous components are admixed, with blending of the two in some areas. The carcinomatous element is almost always of acinar type. Rarely ductal adenocarcinoma, squamous or adenosquamous carcinoma and mixed urothelial squamous components are present [16,20].

As far as the morphology of the sarcomatoid component is concerned, the percentage of sarcomatoid growth ranges from 5% to 99% (mean 65%). Bizarre atypia with giant cells is present in 55% of cases. A small subset of cases demonstrated foci of unusual differentiation within the prostate cancer including basaloid, foamy micropapillary or enteric-type features . The percentage of the sarcomatoid carcinoma component ranges from 5% to 99% (mean

Table 1: Relative Risk (RR) Related to Family History of Prostate Cancer[a].

RiskGroup	RR for Prostate Cancer (95% CI)
Brother(s) with prostate cancer diagnosed at any age	3.14 (2.37–4.15)
Father with prostate cancer diagnosed at any age	2.35 (2.02–2.72)
One affected FDR diagnosed at any age	2.48 (2.25–2.74)
AffectedFDRsdiagnosed<65 y	2.87 (2.21–3.74)
AffectedFDRsdiagnosed ≥65 y	1.92 (1.49–2.47)
Second-degree relatives diagnosed at any age	2.52 (0.99–6.46)
Two or more affected FDRs diagnosed at any age	4.39 (2.61–7.39)

CI: Confidence Interval; FDR: First-Degree Relative [37]

65%), with any percentage of sarcomatoid change considered as a sarcomatoid carcinoma. Histologically, the epithelial portion is adenosarcoma with a variable sarcoma component. A review of 41 cases demonstrated osteosarcoma to be the most common (50%), followed by chondrosarcoma (33%), leiomyosarcoma (17%), rhabdosarcoma (12%), malignant fibrous histiocytoma (10%), fibrosarcoma (7%), spindle cell sarcoma (7%), myosarcoma (5%), undifferentiated sarcoma (2%) and angiosarcoma (2%). Up to 41% of reported cases had more than one type of sarcoma [19,21-24]. Metastatic carcinosarcoma was described in 64% of some reports. The sites of metastases were the lung (43%), spine or bone (26%), lymph node (19%), liver (17%), brain (10%), peritoneum, pancreas, spleen, penis, kidney, retroperitoneal space, pleura and adrenal gland (each 2%) [25,26].

Case Presentation

The case report focuses on a 63-year-old man who presented with difficulty urinating and an enlarged prostate gland. The measured PSA was approximately 8ng/ml. Through CT-MRI the gland was found to be 8,5 X 7,5 X 7,2cm. A colonoscopy was performed on the patient as well.

Macroscopic view of the gland

The tissue block has a greyish colour, is solid, partially presenting cyst (+/-observation of cysts with regular internal surface and bloody, mucoid or translucent content).

Microscopic view of the gland

The epithelium is slighlty differentiated (histological level of malignancy 7-10 on the Gleason scale)

The sarcomatous elements present with the view of:

- osteosarcoma
- chondrosarcoma
- leiomyosarcoma
- fibrosarcoma
- rhabdomyosarcoma

Increased cellularity combined with diphasic model of growth can be observed on the sample. The presence of vessels is of little importance. Sites of autolysis as well as the mitotic activity of the cells are very prominent. Some of the mitoses are of atypical character.

Discussion

Diagnosis

The time from diagnosis of adenocarcinoma to sarcomatoid prostate cancer varied, ranging from 9 months to 20 years. Sarcomatoid prostate cancer can develop in the absence of PSA elevation, making it difficult to detect disease progression [17,27,28].

Trus- Biopsy-Ultrasonography-Microscopy

- Transrectal ultrasound (TRUS) can indicate the boundaries and echoic area of the prostate. A TRUS-guided prostatic biopsy reveals the Gleason score of a prostatic adenocarcinoma, a channel TURP can also be performed.
- The bone scan can be suggestive of metastasis.
- Prostate biopsy specimen discovers the components of adenocarcinoma (Gleason score) and sarcoma.
- Electron microscopy and immunohistochemical staining methods are used to classify the mesenchymal component of the tumor.
- Fine needle aspiration cytology diagnosis of metastatic sarcomatoid prostatic carcinoma.
- Ultrasonography (KUB) and digital rectal examination shows the volume of the prostate.
- Echography usually reveals a pelvic echogenic mass corresponding to a bulky prostate.
- These authors also identified focal angiosarcoma differentiation by light microscopy, and this was confirmed by immunohistochemistry. Sarcomatoid carcinoma is useful for describing pure spindle cell tumors in which epithelial diff erentiation can be shown by electron microscopy or immunohistochemistry, but may not fully reflect tumor behavior [18,20,23,24,27,29-33].

CT -MRI- PET

Clinical staging includes an MRI pelvis or CT chest, abdomen and pelvis in all patients:

- CT scan of the abdomen and the pelvis can show the heterogenity and boundaries of the mass as well as its invasion in the bladder and the adjacent muscles (muscles obturateurs internes). Abdomino-pelvic CT scans have demonstrated locally aggressive prostatic tumors in the literature and can reveal metastasis (as well as X rays).The carcinomatous element in a CS is usually of a high grade, with a Gleason score ranging from 7 to 10 and with a mean score of 9.
- MRI could better demonstrate the presence of invasion of the pelvic organs and internal architecture of the tumor. Goto et al reported that there were heterogeneous hypointense masses with cystic changes within the tumor on T2-weighted magnetic resonance images and marked enhancement with necrotic areas on contrast-enhanced T1-weighted MRI for carcinosarcoma of the prostate. Metastasis can also be found with this method of imaging.
- CT, PET, and fused PET/CTimaging can be used in carcinosarcomas taging. In a case, local disease was defined as prostate-confined, in the absence of radiographic evidence of metastatic disease and bladder invasion. Malignant invasion into the bladder was determined by radiographic imaging and/or direct visualization during cystoscopyshowing large necrotic mass indenting the base of the bladder.

- Skeletal scintigraphy shows metastatic changes in spine, ribs, both femurs and skull [15,16,18,28,34].

Morphology-histology

- Carcinosarcoma is almost always of the acinar type. Rarely, ductal, squamous and adenosquamous carcinomas may be seen. Careful search for stromal infiltration and examination of individual nuclear features including prominent nucleoli should be performed. A search for adjacent usual small acinar adenocarcinoma, intraluminal crystalloids and blue wispy mucin can be helpful diagnostic aids.
- Cytologically, nuclear pleomorphism is moderate to marked, with numerous mitotic figures, including atypical ones, readily identified.
- Histopathological excretory pyelography reveals hydronephrosis [12,15,25,29,35].

Markers

Immunohistochemical stains show mesenchymal or epithelial differentiation in the sarcomatoid component. Epithelial markers (cytokeratins, PSA, PSAP) and muscle markers can be detected by immunohistochemistry in the malignant spindle cells. Sarcomatoid elements react with vimentin or specific markers corresponding to the mesenchymal differentiation, if present Expression of cytokeratin by the spindle cell component of sarcomatoid carcinoma suggests a common origin rather than a collision tumour composed of sarcoma and carcinoma.

- Vimentin immunostains are uniformly positive, and S-100 protein is consistently found in chondrosarcoma tous regions. Skeletal muscle and vascular differentiation is substantiated by positivity for myoglobin and CD31 or CD34 respectively.
- Immunohistochemistry revealed over-expression of c-erb B2 in the papillary epithelial component of both cases, whereas the solid undifferentiated epithelial areas in the second patient expressed c-kit, CD10, CD31 and synaptophysin.
- A positive PSA and prostatic acid phosphatase (PAP) and a negative p63 or high molecular weight (HMW) keratin staining may be useful to diagnose difficult cases.
- Needle biopsy specimens containing lesions with spindle cell morphology should raise the differential of sarcomatoid carcinoma, and immunostains for cytokeratin or PSAP may be of use in these instances.
- Hematoxylin and Eosin (H&E) stain showing spindle cells and adenocarcinoma.
- Generally in an immunohistochemical investigation the exams performed use paraffin-embedded sections and antibodies against cytokeratin (AE1 and AE3), PSA, α-smooth muscle actin, desmin, CD34, and S-100 protein. The chondrosarcoma tous area showed a positive staining for S-100 protein. All areas were negative for α-smooth muscle actin [12,15,16,19,21,22,25,36,37].

The diagnosis of carcinosarcomais usually established by subsequent TURP after recurrence of obstructive symptoms or can be found incidentally in the subsequent radical prostatetomy specimen. In some patients who do not present with obstructive symptoms, carcinosarcomais often found incidentally in the radical prostatectomy specimen after a needle biopsy diagnosis of adenocarcinoma or at the time of pelvic recurrence after a prior radical retropubic prostatectomy for adenocarcinoma. A diagnosis may also be established clinically without biopsy confirmation due to abnormal results on digital rectal examination and an elevated serum PSA concentration. In addition to long-term surveillance for disease recurrence, there should be suspicion for the development of secondary malignancies when obstructive symptomatology occurs in the absence of biochemical recurrence, particularly in a patient with a history of pelvic radiation [13,15].

Therapy

Patients with carcinosarcoma can follow different treatments according to the stage, the general condition and the recurring metastasis of the tumor. Generally the most effective therapy is surgery and it should be known that the only curative therapy is immediate radical surgical excision. Nonsurgical therapy was ineffective in metastatic cases. Carcinosarcoma of the prostate is a very aggressive. Below are various methods of therapy that are applied globally [30,38].

Surgery: Surgical removal of the prostate seems to be the best option for treatment in the select group of patients in which the disease remains confined to the prostate. A radical cystourethroprostatectomy, retropubic prostatectomy, pelvic exenteration with bilateral lymphadenectomy, ileal conduit diversion, cutaneous ureterostomy and colostomy can be applied in more difficult cases with lymph expansion. Radical surgery can be performed together with adjuvant therapy with varying results. Despite the dismal survival associated with prostate carcinosarcoma, palliative surgical extirpation can be successful in patients with debilitating pain.Various treatment modalities have been used in patients with carcinosarcoma, including those directed against carcinoma and sarcoma. Results have been disappointing [17,19,26-28,30,36,39,40].

Radiation-chemotherapy- hormonal therapy: Radiation therapy (ranged from 40 Gy to 55.8) is the primary treatment modality in a lot of cases. It is given to pelvic organs and lower spine with a subsequent drop in PSA in many forms: external beam radiation, implants and cobalt therapy. A combination of surgery followed by radiotherapy seems to be statistically superior to resection alone.

- Different chemotherapy regimens (i.e. small cell carcinoma, sarcoma, prostate adenocarcinoma) are used to treat several patients with metastatic disease. A lot of patients receive two cycles of VP-16 and ifosfamide, while others receive chemotherapy VAC, vincristine, adriamycin, and cyclophosphomide for rhabdomyosarcoma and cisplatin, adriamycin, taxotere, estramustine, carboplatinum, ifosphamide for the other subtypes.
- Some patients have received androgen deprivation therapy after the diagnosis of carcinosarcoma, and others have undergone orchidectomy. Treatment with leuprorelin has also been applied.
- There were some efforts to combine radio chemotherapy and hormonal therapy to locally advanced prostaticcarcinoma. The patients (T3-T4) were treated with 61.7 Gy (single dose was 1.8 Gy), epirubicin 20mg/m^2 and 5-fluorouracil 800mg/m^2 on day 1 to 5 and 29 to 33 of radiotherapy. The hormone therapy began before the radio-chemotherapy. The hematological side effects were interesting: three out

of 8 patients had leucopenia grade III and 1 of 8 patients leucopenia grade IV according WHO. Generally, the concurrent radio-chemotherapy with epirubicin and 5-fluorouracil in the combination with hormone therapy was tolerated well [14,18,20,21, 25,27,34,36].

Laparoscopicrobotic-assisted radical cystoprostatectomy has become widely adopted and robotic-assisted laparoscopic cystoprostatectomy is now being developed in several centers. In 2003, Menon et al reported a series of 17 robotic-assisted radical cystoprostatectomies and urinary diversions for bladder cancer focusing on nerve preservation. They used a 3-step technique of robotic pelvic lymphadenectomy and cystoprostatectomy followed by externalization of bowel and extracorporeal neobladder reconstruction, and internalization of the neobladder with robotic urethravesical anastomosis. Balaji et al reported one case of robotic-assisted radical cystoprostatectomy and ileal conduit urinary diversion performed totally intracorporeally. Blood loss was 500mL, operative time 828 minutes-10bh and hospital stay 6-10 days. It is a new minimally invasive procedure, with which, after cystoscopic examination that demonstrates extension of the tumor into the bladder neck, a robotic-assisted laparoscopic cystoprostatectomy, bilateral pelvic lymph node dissection, and ileal conduit urinary diversion can be performed. After the surgery the patient can undergo adjuvant external beam radiation [22,41].

Molecular therapy: The researchers are using the small molecule Lutetium 177Lu-PSMA-617 to target prostate-specific membrane antigen (PSMA), a protein that is abundantly expressed in 85-90 percent of metastasized prostate cancers. The small molecule binds to PSMA and delivers precise radiation therapy intended to shrink the cancer—even in cases in which cells have yet to form a visible tumor on a bone or CT scan.

Because prostate cancer growth is highly susceptible to tumor-microenvironment interaction it is reasonable that control of prostate tumor growth might be optimized by co-targeting both tumor and stroma. To explore this concept, chimeric tumor models we established, consisting of human prostate cancer cells and bone stroma. By introducing a "bystander" therapeutic gene, herpes simplex thymidine kinase (hsv-TK), to stromal cells only, effective cell kill in tumor epithelium was observed *in vitro* and shrinkage of tumor size in vivo upon addition of a pro-drug, gancyclovir (GCV). By interrupting this communication, and targeting both tumor and stroma, tumor growth and survival may be adversely affected. Conceptually, co-targeting tumor and stroma in prostate cancer bone metastasis is a rational approach to the "vicious cycle" constantly operating between tumor and stroma. Directly inducing cell-kill of tumor epithelium and starving cancer cells by disrupting tumor interaction with the stromal compartment could achieve the best possible tumor regression. An adenoviral vector in which therapeutic gene expression was controlled by a tissue-specific and tumor restrictive promoter, can be used, such as osteocalcin, osteonectin or bone sialoprotein. These have been shown to be highly effective in inducing long-term tumor regression, and even some cure in pre-established tumor in the skeleton with administration of the adenovirus through the intravenous route (Hsieh and Chung, 2001; Matsubara et al., 2001; Hsieh et al., 2002) [42,43].

Other therapies include palliative transurethral electro vaporization of the prostate (TUVP) brachy therapy and endocrine therapy [15,20,33].

As with other prostatic sarcomas with uniformly poor outcomes, multimodality therapy may be applied. Surgery generally involves anterior exenteration with urinary diversion. For patients with bulky disease who may be at higher risk for positive surgical margins, small series have demonstrated that neoadjuvant doxorubicin and cisplatin can produce significant tumor necrosis. Adjuvant therapies include doxorubicin-based chemotherapy and radiation therapy. Patients with post-irradiation malignancies, however, may have already received their maximum dose of pelvic irradiation. In patients who present with metastatic disease, palliative surgery to relieve urinary or bowel obstruction may be appropriate. Nevertheless, non-surgical therapy (androgen ablation treatment and chemotherapy) seems to be ineffective and 55.5% of patients are unresponsive to chemotherapy (taxotere, estramustine, car-boplatinum, or cisplatinum [13,16,44,45].

Prognosis

Carcinosarcoma is an aggressive, high-grade tumor. Recurrence, distant metastasis (54%), and angiolymphatic invasion are frequently encountered. The lungs are the most common site of metastasis. Metastasis may also occur to the liver, bones, and brain. Twenty-five percent of patients have metastatic disease at diagnosis, and an additional 39% subsequently develop metastasis. Involvement of cervical lymph nodes with metastasis is uncommon.The median period of survival after diagnosis was 10 months, ranging from 34 days to 5 years. The 3-year survival rate is approximately 10%.17; in 63% of cases, there was no evidence of recurrence after a median period of 22.4 months. The outcome of patients with prostatic SC is poor, with a median survival of 3 years and 7-year survival rate of 14%. The disease can be locally aggressive, with local recurrences and formation of large pelvic masses. No parameter, including the age, sarcoma's components, condition of the metastatic lesion, therapy, history of radiation, or androgen deprivation therapy, histologic subtype, tumor size, percentage of necrosis, percentage of sarcoma, sarcoma grade, or Gleason grade of adenocarcinoma, has been found to be predictive of outcome. The use of hormonal treatment, chemotherapy, adjuvant or neoadjuvant therapy and radiotherapy after surgical resection for carcinosarcoma is not a standard approach, and it does not provide apparent survival [12,14,19,25,26,27].

Case reports and series available in the literature uniformly demonstrate dismal outcomes, but recent studies have postulated an improvement of prognosis after radical surgery in locally advanced cases that lack distant metastases, thus suggesting that such initial treatment should be elected in these cases whenever it is possible. The favorable outcomes in the local disease group highlight the importance of early diagnosis. In sarcomatoid prostate cancer, the lack of PSA expression by the sarcomatoid component may make it difficult to diagnosis localized disease Immunohistochemistry and RNA in situ hybridization studies looking at AR expression may help to better understand the lack of response observed in this small subset of tumors [13,20,36].

As opposed to what stated before, in some cases, in terms of tumor-related factors, the histologic subtype of prostate sarcoma appears to have prognostic significance. It has been found that the overall survival for adults with non-RMS histologies is poor with a median survival of only 2 years. Pediatric patients with RMS faired much better with a median survival of over 10 years. The presence of metastatic disease at diagnosis, however, is a poor predictor of outcome. In terms of treatment-related factors, surgery alone is inadequate treatment. The median survival for the rhabdomyosarcoma subgroup and

non rhabdomyosarcoma subgroup is 142 months and 24 months, respectively. The OS hazard ratio in the local disease with bladder invasion (median OS: 9 months) and metastatic disease groups (median OS: 7.1 months) are 20.46 (95% CI: 2.43, 172; p=0.005) and 43.34 (95% CI: 4.39, 427.4; p=0.001) respectively. In one case after a median follow-up of 21 months, the four patients receiving gonadotropin-releasing hormone agonists and/or anti-androgens survived 7, 8, 8, and 10 months, while those treated without hormonal therapy survived from 1 to 21 months, with a median of 9 months. Thus, the poor outcomes of patients with local invasion into the bladder may highlight the propensity of these sarcomatoid tumors to spread systemically, limiting curative options [13,20,39,46].

Approximately half of all patients developed metastatic disease either at time of presentation or subsequently. Of patients with meaningful follow-up, 6/7 died within 1 year of the diagnosis of sarcomatoid carcinoma; Kaplan-Meier analysis revealed that the actuarial risk of death at 1 year after diagnosis of sarcomatoid carcinoma was 20%. The sarcomatoid component that demonstrates variable degrees of atypia, has fatable outcome in 85, 8% within 1 year of the diagnosis of sarcomatoid carcinoma. Kaplan-Meier analysis revealed that the actuarial risk of death at 1 year after the diagnosis of sarcomatoid carcinoma was 20%, despite clinical intervention [16,21].

Conclusion

All in all, what can be observed easily is the fact that prostate cancer's incidence varies among different countries and continents and additionally this disease characterizes men who are over 60 years-old. When it comes to the etiology of this malignancy, mutations in 3 genes (*BRCA1*, *BRCA2*, *HOXB13*) are contributory factors in the development of prostate cancer. Elaborating on our case the 63 years-old man had most of the primary symptoms (dysuria, increased gland's size and high concentration of PSA-prostate specific-antigen) and the diagnosis was confirmed by a transurethral biopsy. Several structure changes are triggered in the prostate gland by the disease, such as increase in size, colour and solidity differences and other histological differentiations. In most cases the diagnosis is established via a TURP (Transurethral resection of the prostate) and also taking into account the frequency of the symptoms. Although there are countless cases where a biopsy is not needed to confirm the disease as the increased [PSA] concentration level and the abnormal results on digital rectal screening indicate that the patient suffers from prostate cancer. There are numerous means of therapy, these are surgery, chemotherapy, radiation therapy, hormonal therapy, laparoscopic cystoprostatectomy. As in other types of cancer multimodality therapy can be applied in prostate cancer as well. A new way to encounter the disease is molecular therapy which is not applied yet because it's under thorough examination. Unfortunately, non-invasive therapy seems to not have hopeful results as more than half of patients undergoing chemotherapy have not improved. Almost half of patients develop a metastatic tumor that can be localized in lungs, bones, brain and liver. Despite the disease's often poor survival rate, recent studies reveal a rise in survival rate in cases (with absence of metastases) where drastic surgery was considered to be the best therapy. This is without any doubt a very hopeful message for years to come.

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