Case Report

Pseudohyperplastic variant of adenocarcinoma as a component of α-methyl–CoA-racemase (AMACR negative) carcinosarcoma of the prostate

Sharmila Dudani, Ajay Malik, A. S. Sandhu[1], N. S. Mani

Departments of Pathology and [1]Urology, Army College of Medical Sciences and Base Hospital, Delhi Cantt, Delhi, India

ABSTRACT

Carcinosarcoma (CS) or Sarcomatoid carcinoma (SC) of the prostate is a very rare malignant tumor of the prostate having an aggressive clinical course and dismal prognosis. The adenocarcinomatous element is usually of the acinar type and closely admixed with a sarcomatous component. We report a case of α-methyl–CoA-racemase (AMACR)-negative pseudohyperplastic variant of adenocarcinoma in CS. To the best of our knowledge, there have been no published case reports of this variant in CS till date. An accurate diagnosis is essential as this uncommon, aggressive cancer has limited therapeutic options.

Keywords: α-methyl–CoA-racemase, carcinosarcoma prostate, pseudohyperplastic variant

INTRODUCTION

Carcinosarcoma (CS) of the prostate, also known as Sarcomatoid carcinoma (SC), is a very rare malignant biphasic tumor of the prostate associated with an aggressive clinical course and poor prognosis. Less than 100 cases have been reported in the literature.[1] It constitutes an admixture of a high-grade adenocarcinoma, usually of the acinar type, admixed with a sarcomatous element that may or may not show the presence of heterologous differentiation. CS is seen to occur after radiation or brachytherapy for carcinoma prostate, and is usually discovered at an advanced stage. We report a case of pseudohyperplastic variant of adenocarcinoma as a component of CS of the prostate in an elderly male who had not received any previous treatment. To the best of our knowledge, there have been no published case reports of this variant in CS till date. It is important to recognize this entity as this variant can mimic benign prostatic glands and patients' may not have elevated serum prostatic specific-antigen (PSA) levels. Even with complete resection, the incidence of local recurrence and metastasis is high in CS as compared with other prostatic cancers. Currently, there is no standardized treatment regimen available for CS.[1]

For correspondence: Prof. Col. Ajay Malik,
Department of Pathology, Army College of Medical Sciences and Base Hospital, Delhi Cantt, New Delhi, India.
E-mail: drajaymalik@gmail.com

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CASE REPORT

A 67-year-old male was admitted to a general hospital with acute retention of urine. A history of dysuria was present for a few days prior to this. There was no history of hematuria, hesitancy, terminal dribbling or nocturia. He was a non-smoker and non-alcoholic and reported no exposure to hazardous chemicals. There was no family history of genitourinary cancer. Rectal examination revealed a mildly enlarged prostate gland. Ultrasonography (KUB) revealed a prostate volume of 39 cc. PSA was 43. ng/mL. A TRUS-guided prostatic biopsy revealed a prostatic adenocarcinoma having a Gleason score (3 + 3) = 6. The bone scan revealed increased tracer uptake involving the left symphysio-pubic bone, suggestive of metastasis. A channel TURP and bilateral ordichectomy was performed.

Histopathological examination of the TURP specimen revealed 60% of the submitted tissue to be comprising of a malignant biphasic tumor.The epithelial component revealed numerous crowded, closely packed large atypical glands with an absent basal cell layer infiltrating the surrounding

stroma. The lining epithelium was lined by cells having even luminal borders and abundant cytoplasm. Nuclear enlargement with prominent nucleoli was seen. Corpora amylacea or crystalloids were not present. Admixed with the adenocarcinoma was a highly cellular tumor composed of spindle to polygonal cells having atypical nuclei and increased mitotic activity. Foci of necrosis along with tumor giant cells were also seen. No heterologous elements were present. The carcinomatous elements exhibited negative staining for p63 and AMACR (Clone13H4, pre-diluted, ready to use, rabbit monoclonal IgG antibody; Bio Genex, Freemont, CA, USA). The neoplastic spindle cells expressed positivity for vimentin, smooth muscle actin (SMA) and negativity for pancytokeratin [Figure 1]. A diagnosis of CS prostate with pseudohyperplastic variant of prostatic adenocarcinoma was given.

DISCUSSION

The nomenclature and histogenesis of SC is controversial. Some workers consider them to be a "collision" of malignant epithelial and mesenchymal elements developing separately in the prostate. However, the recent World Health Organization classification does not distinguish between CS and SC and uses the term SC (Sarcomatoid carcinoma) to characterize these lesions as they share similar clinicopathological features, including poor prognosis.[2] It has been demonstrated that both the malignant epithelial and the spindle cell components are clonally related.[1,2] SC may be seen in the initial pathological material (synchronous presentation) or there may be a previous history of adenocarcinoma treated by radiation or hormonal therapy.[1-3]

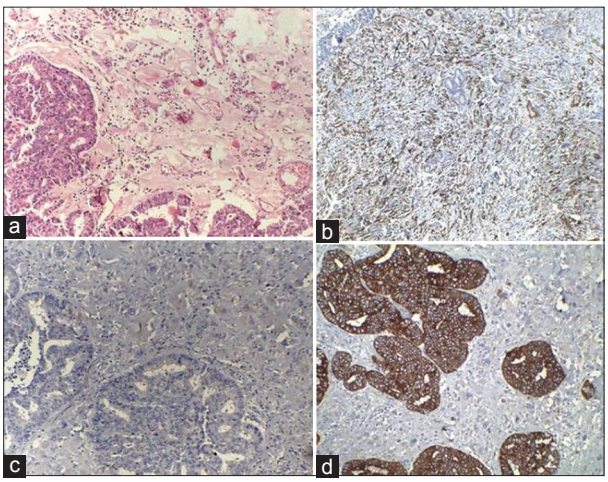

Figure 1: (a) Photomicrograph reveals numerous crowded, back-to-back glands infiltrating the surrounding stroma. Lining epithelium shows nuclear enlargement, prominent nucleoli and abundant cytoplasm. Stroma shows tumor composed of spindle to polygonal cells having atypical nuclei and increased mitotic activity. Occasional tumor giant cells are also seen. (b, d) Sarcomatous element was positive for vimentin and negative for pancytokeratin. (c) AMACR-negative prostatic adenocarcinoma

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.[4] It is almost always of the acinar type.[1] Rarely, ductal, squamous and adenosquamous carcinomas may be seen.[1] To the best of our knowledge, this is the first case to describe the pseudohyperplastic variant of adenocarcinoma as a component of SC. As this variant can have a deceptively benign appearance at low magnification, a 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. Unlike other prostatic adenocarcinomas that are AMACR positive, the pseudohyperplastic variant may show AMACR positivity in only 60–80% of cases.[2] 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.

The sarcomatous component in a CS may vary from 5% to 99% and, usually, exhibits hypercellularity, enlarged hyperchromatic nuclei, frequent mitoses and occasional bizarre tumor giant cells with necrosis. Rarely, the atypia may be mild. About 30% of cases may show the presence of heterologous elements of osteosarcoma, chondrosarcoma or rhabdomyosarcoma.[1,2]

Inflammatory myofibroblastic tumor (IMT) or post-operative spindle cell nodules need to be distinguished from SCs. IMT can appear as a result of exuberant stromal reaction weeks or months after a TUR procedure. Histologically, it is a very cellular tumor composed of intersecting fascicles of uniform spindle cells with high mitotic activity, simulating a leiomyosarcoma. The background shows variable inflammation, prominent vascularity with occasional extravasated red blood cells. However, the proliferating cells are of myofibroblastic origin and show a strong positivity for pancytokeratin, ALK, actin and p53 and negativity for S100. IMT usually follows a benign course.

Most patients present with obstructive symptoms like poor urinary stream, hesitancy, incomplete voiding and terminal dribbling.[4] Urgency and dysuria are less common. The tumor may produce bladder outlet obstruction. Serum PSA may be normal but nodal and distant metastasis are common.[2] There are no standard treatment recommendations for CS as there have been very few cases reported in the literature. Surgery is the treatment of choice in operable tumors. Non-surgical therapy (androgen ablation and chemotherapy) seems to be ineffective and 55.5% of patients are seen to be unresponsive to chemotherapy.[5] Prognosis for these patients is poor.

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