Case Report

Adenosquamous carcinoma of the prostate: Case report of a rare aggressive tumor

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Abstract

Adenosquamous carcinoma (ASCC) of the prostate is a very rare, aggressive tumor with only few cases, reported in literature. Since its initial description by Thompson in 1942, there have been fewer than 30 cases reported in the literature. Until now, it has no well-established therapeutic guideline. While the majority of these tend to arise subsequent to endocrine or radiation treatment with squamous differentiation, approximately one-third of cases have arisen in a de novo setting. This report presents an additional case of the rare adenosquamous carcinoma of the prostatearising in a 65 year old patient with no previous risk factors.

Keywords: Adenosquamous carcinoma, Prostate specific antigen (PSA).

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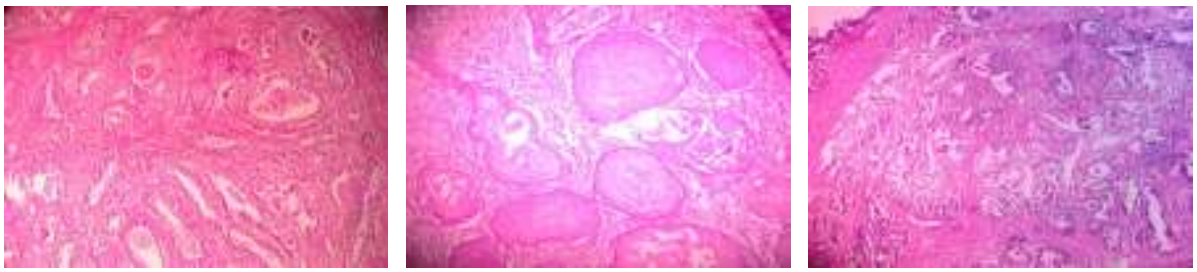
INTRODUCTION

Carcinoma of the prostate gland is the most common malignant tumor affecting adult males. Conventional adenocarcinomas represent bulk of these tumors while a few cases of mixed carcinomas comprised of two malignant epithelial components have also been described. The majority are cases of mixed adenocarcinoma and transitional carcinoma. Among the uncommon histological variants of carcinoma of the prostateadenosquamous carcinoma of the prostate is an extremely rare neoplasm associated with a poor prognosis [2]. Till now, there is no well-established guideline for the treatment of Adenosquamous carcinoma. Here we are reporting a case of this rare entity.

CASE HISTORY

A 65-year-old male patient presented with poor urinary outflow and increased urinary frequency. On examination the external genitalia were normal but the prostate was non-tender, enlarged, hard, and nodular. His urine analysis and blood biochemistry, including serum PSA were normal. Ultrasonography revealed a prostate size of 68 gm with non-homogenous texture and post-void residual urine of 110 ml. Transurethral resection of the prostate was done and the prostatic chips were sent to the pathology department for histopathological examination.



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PATHOLOGICAL FINDINGS

Gross: Multiple pale white, formalin fixed prostatic chips all together measuring 5 cms was received in a single container which was processed and stained by Hematoxylin and Eosin (H and E) stain.

Microscopy: The histological sections showed adenosquamous carcinoma mainly composed of malignant squamous elements and a small disorderly mixture of adenocarcinomatous elements. A transitional area comprising of both the malignant epithelial components was also noted.

DISCUSSION

Adenosquamous carcinoma is defined by the presence of both glandular (acinar) and squamous components[1, 2, 3]. Since the first description by Thompson, approximately 33 cases of ASCC of the prostate have been reported [3,4,5,6,7,8]. Of all ASCC cases reported in the literature, two-thirds involved patients previously treated for prostatic adenocarcinoma with hormones and/or radiation[3,4,5,67,8]. The timeframe for the appearance of squamous differentiation in the carcinoma varies from 3 months to many (up to 9) years after therapy. The remaining one-third of patients had no history of prostate cancer or hormonal therapy[4]. However, the present case lacks this history, suggesting that the 2 types of epithelia may have developed concurrently. There are several theories to explain the histogenesis of ASCC of the prostate: 1) metaplastic transformation of adenocarcinoma cells [5,6], 2) a collision-type tumor, with the squamous component developing from metaplastic foci after radiation or hormonal therapy 3) ASCC derived from pluripotent stem cells capable of multidirectional differentiation[5], or 4) a more plausible explanation would be clonal evolution/divergence of persistent carcinoma, secondary to the selective pressure of therapy, for ASCC occurring after radiation or androgen deprivation therapy[5]. Benign prostatic hyperplasia following hormonal therapy is well documented. In view of the large number of patients treated with hormonal therapy and rarity of ASCC, it is very difficult to accept any single pathogenetic mechanism Prostatic ASCC, like glandular adenocarcinomas, can spread along nerves; extend locally into periprostatic soft tissue, the bladder, and seminal vesicles; and metastasize to lymph nodes and bones[6,7,8]. In widely disseminated disease, metastatic deposits have been detected in the peritoneum, diaphragm, liver, and lungs. Clinically, these patients often present with bladder outlet obstruction and dysuria, and are quite large. The prostate-specific antigen (PSA) level may or may not be elevated depending on the squamous component. Glandular and squamous components could be distinct or could show direct transition. The Gleason score can be used for the glandular component, but not for the squamous component, of ASCC. Since squamous component is predominant in the above case, Gleason score could not be applied. The adenocarcinoma element is often high-grade, while the grade of the squamous portion is variable[5]. Most cases of ASCC of the prostate were found at the prostatic urethra and/or adjacent tissues, [5] making them more readily accessible by transurethral resection for bladder tumor (TURP) than by core needle biopsies and TURP was also performed in this case. Since there is no clinical trial specifically designed for ASCC of the prostate, the optimal treatment strategy has not been established. Radical prostatectomy, radiation therapy, or chemotherapy have been used alone or in combination. The prognosis for patients with ASCC is very poor, even in those patients with localized disease who subsequently underwent prostatectomy, suggesting this is a disease with a propensity for early microscopic dissemination.

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