Ductal Adenocarcinoma of the Prostate with Endometrioid Features in a 69-Year-Old Man

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Prostate adenocarcinoma often derives from the acinar epithelium of the prostate gland. Prostate adenocarcinoma arising from the large duct with histopathologic characteristics mimicking endometrioid carcinoma of the uterus is rare. In 1967, Melicow and Pachter described a case of endometrioid carcinoma of the prostate with a concentration of malignant cells in the prostatic utricle [1]. Its histopathologic characteristics resembled endometrioid carcinoma of the uterus. Melicow and Tannenbaum theorized that such tumors are derived from Müllerian duct remnants, which are supposedly embryonic female vestiges localized in the epithelium of the verumontanum [2]. Unlike the usual prostate adenocarcinoma, these tumors are centrally located and have a papillary configuration. Here, we report a case of prostatic ductal adenocarcinoma with endometrioid features.

Case Report

A 69-year-old man visited our urology outpatient clinic on December 31, 1998, due to 2 months' of intermittent, painless hematuria. He denied dysuria, frequency, or urgency. Rectodigital examination detected marked prostate posterior lobe enlargement with elastic consistency and smooth surface. The serum prostate-specific antigen (PSA) concentration in peripheral blood was 22.0 ng/mL. Transrectal ultrasonography showed enlargement of the prostate (estimated at 33.5 g)
with a hypoechoic tumor, measuring 3.0 x 3.0 cm in its largest dimension, at the right side. The seminal vesicle had a clear margin without enlargement. Cystoscopy was performed on January 13, 1999, and some papillary tumor was found within the prostatic urethra near the verumontanum. The tumor was soft and friable, and was easily removed from the prostatic urethra via cystoscopy. The mucosa of the urinary bladder was unremarkable. The biopsy specimen of the tumor had a distinctly papillary configuration with focal glandular structure (Fig. 1). Adenocarcinoma of the prostatic urethra was diagnosed. Computed tomography (CT) and magnetic resonance (MR) imaging also revealed a lobulated necrotic mass, measuring 3.0 x 3.0 cm in its largest dimension, at the right side of the prostate near the urethra. No evidence of lymph node metastasis was seen on MR imaging study. The rectal wall was preserved. The periprostatic fat plane was intact without seminal vesicle involvement. Bone scan revealed no bone metastasis.

Radical prostatectomy was performed 1 week after cystoscopy. The majority of the tumor was found in the central zone of the prostate with a necrotic area at the right side (Fig. 2). It was composed of closely packed glandular structures with minimal intervening stroma (Fig. 3). These glandular structures were lined by a single layer of high columnar cells with focal stratification. Frequent papillary projections of glandular epithelium and intraglandular bridging, similar to the histopathologic findings of endometrioid carcinoma of the uterus, were noted. These tumor cells had basally located nuclei, prominent nucleoli, and abundant eosinophilic, occasionally vacuolated, cytoplasm. Small foci of microacinar adenocarcinoma were seen peripheral to the area of the ductal adenocarcinoma (Fig. 4). The frank pattern of ductal adenocarcinoma was also noted at the large duct of the verumontanum. The tumor had invaded through the posterior capsule into the regional soft tissue along the perineural routes. Lymphatic and vascular permeation were noted. Immunohistochemical study revealed positive staining for PSA (Fig. 5). No obturator lymph node involvement was found. Androgen deprivation therapy (flutamide 25 mg tid) was given after the operation and a remarkable decrease in serum PSA was noted (0.02 ng/mL 1 month after surgery, and 0.01 ng/mL 27 months after surgery). No evidence of recurrence or metastasis was found during 27 months' follow-up.

**Discussion**

The most common prostate conditions in elderly men are benign nodular hyperplasia and microacinar adenocarcinoma of the prostate. Carcinoma can also derive from nearby structures, such as the periurethral glandularis and large duct of the prostate. Carcinoma of the prostate with a pattern of endometrioid carcinoma is a distinct clinicopathologic entity first described by Melicow and Pachter in 1967 [1]. The incidence of this condition among all prostatic carcinomas is about 0.2% to 0.8% [3, 4]. A previous study found that patients with prostate cancer with an endometrioid pattern ranged in age from 61 to 89 years old (mean, 74.2 years) [5]. Intermittent urethral bleed-
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**Fig. 3.** The tumor shows a closely packed glandular structure with minimal intervening stroma, lined by a single layer of highly columnar cells with focal stratification, similar to the histopathologic appearance of endometrioid carcinoma of the uterus. (Hematoxylin and eosin, x 100)

**Fig. 4.** Small areas of microacinar adenocarcinoma of the prostate are seen in the peripheral area of the tumor. (Hematoxylin and eosin, x100)

**Fig. 5.** Immunohistochemical staining of the tumor with anti-prostate specific antigen monoclonal antibody using avidin-biotin complex method (ABC method) shows positive staining in the cytoplasm. (x 100)

Inevitably results in hematuria and/or urinary obstruction [6]. This pattern was seen in four of 15 cases reported by Christensen et al [6]. Our patient also had this tumor pattern. The polypoid lesions found cystoscopically in tumors of this pattern could be transitional cell carcinoma of the prostatic urethra, ectopic prostatic tissue, benign polyp, nephrogenic adenoma, proliferative papillary urethritis, inverted papilloma, or accentuated mucosal folds. Histopathologic studies of the intraurethral tumor are needed to differentiate these conditions. Polypoid lesions are more easily detected because their central location results in earlier onset of hematuria or obstructive symptoms. The incidence of cases with capsular invasion (95%) and positive margins (47%) [6], which were also found in our patient, are much higher than those for microacinar adenocarcinoma at the same clinical stage. These tumors have more aggressive behavior than microacinar adenocarcinoma at the same stage. The serum PSA concentrations in patients with these tumors are usually less elevated than those in patients with microacinar adenocarcinoma [4].

The intraurethral component of these tumors usually reveals an exuberant papillary pattern with fibrovascular core fronds lined by single-layer or pseudostratified tall columnar epithelium. This pattern is different from benign prostate acini lined by prostatic glandular epithelium and urothelium in prostatic urethral polyps [7]. The intraprostatic component is usually underneath an intact or partially denuded urothelium near the prostatic verumontanum, and has more intraductal papillary infolding and cribriform features than the intraurethral components. The pres-
ence of a well-established fibrovascular core, stromal fibrosis, hemosiderin deposition, large or back-to-back glands, and perineural invasion of ductal adenocarcinoma could differentiate these lesions from high-grade prostatic intraepithelial neoplasm. These intraurethral and intraprostatic components have been described by Ro et al as type A (obviously exuberant papillary endometrioid pattern with focal intraductal component) and type B (less papillary pattern and more intraductal components) [8]. Both types of components coexisted in approximately half of cases and tended to merge into each other. In addition, both types usually merged with regular microacinar carcinoma. In our patient, only 2% of tumor volume in sections was of microacinar type. Some cases of ductal adenocarcinoma of the prostate only involve the peripheral zone of the prostate [6]. Clinical and pathologic evidence of involvement of large periurethral prostatic ducts or urethra are required for definite diagnosis of ductal adenocarcinoma [9].

The presumption of a close histogenetic relationship between endometrioid prostatic carcinoma and endometrioid carcinoma of the uterus is based not only on the histologic resemblance, but also on embryologic evidence [1]. Melicow and Pachter suggested that the utricle, a site of origin for some endometrioid tumors, might be a Müllerian duct remnant [1]. Because of its resemblance to endometrioid carcinoma of the uterus and position near the verumontanum, the histogenesis of the tumor was considered to originate from the Müllerian duct remnant, the utriculus masculinus, which should involute in adults. However, the histochemistry, immunohistochemistry, and electronmicroscopy studies favor a prostatic ductal origin for these tumors [10]. Ultrastructural studies found that the glandular structures of the tumor contain lighter and darker cell types [10]. Lighter cells have lipid droplets, lysosomes, and well-developed Golgi apparatus, resembling the secretory cells of the acinus and the tumor cells of microacinar adenocarcinoma. Darker cells with less-developed Golgi apparatus, smooth or rough endoplasmic reticulum, are seldom vacuolated and rarely contain lipid droplets. These darker cells resemble the ductal cells of the normal prostate. The absence of ciliated endometrial features does not support a histogenesis arising from the Müllerian duct.

Histochemical, ultrastructural, and immunohistochemical findings support a prostatic but not an endometrial origin for endometrioid carcinoma of the prostate. This type of prostatic tumor is closely related to prostatic duct adenocarcinoma [11–14]. Prostatic ductal adenocarcinoma is heterogeneous and has a spectrum of differentiation from the duct to the acinus in most cases [4]. Endometrioid carcinoma of the prostate could be a morphologic variant of prostatic ductal adenocarcinoma [15]. This term may be a misnomer that does not indicate the precise histogenesis. Walker et al [13] and Bostwick et al [3] suggested that the term endometrioid should be abandoned to avoid ambiguity of histogenesis and therapy strategy.

Hormone manipulation is used to treat microacinar adenocarcinoma. However, hormone manipulation of endometrioid carcinoma of the prostate is con-traversial, because of the proposed hypothesis of a Müllerian origin and resemblance of the tumors to endometrioid carcinoma of the uterus. Estrogen therapy and orchietomy are contraindicated due to the well-known estrogen dependence of endometrioid adenocarcinoma of the uterus [16]. Previous studies have suggested a poor prognosis with conservative management of prostatic ductal adenocarcinoma [3, 15, 17]. Greene et al found a 5-year survival rate of 29% [17], and Bostwick et al found a 5-year survival rate of 15% [3]. Christensen et al found a marked increase in short-term failure rate after radical prostatectomy in ductal adenocarcinoma, 47% in stage B cases in a series of 15 cases at a mean of 17 months’ follow-up [6]. However, both our patient and a case of endometrioid adenocarcinoma of the prostate reported by Young and Lagios showed objective tumor regression and marked clinical improvement under conservative therapy [14].

References


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