Basal Cell Carcinoma of the Prostate

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Basal cell carcinoma (BCC) of the prostate, a rare variant of prostate cancer, is derived from the basal cells of prostatic ducts and acini. BCC generally occurs in elderly men with obstructive voiding symptoms and levels of serum prostate-specific antigen within the normal range. In most cases, diagnosis is made through transurethral resection or simple enucleation. Most cases are indolent, but local recurrence and metastasis have been reported in a few cases. Thus, radical surgery and long-term follow-up are recommended. We report a case of a 54-year-old man who underwent radical retropubic prostatectomy after being diagnosed with BCC during a transurethral resection performed for lower urinary tract symptoms. The patient has remained free of disease for 4 months after surgery. **(Korean J Urol 2009;50:408-412)**

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Basal cell carcinoma (BCC) of the prostate is a rare variant, comprising <0.01% of all malignant tumors of the prostate. It is derived from the basal cells of the prostatic ducts and acini, and mainly arises from the transition zone.[1,2] Patients with BCC of the prostate are generally older males with lower urinary tract symptoms (LUTS). In most cases, the diagnosis is made during transurethral resection or simple enucleation of the prostate, because the serum prostate-specific antigen (PSA) level is usually normal.[1-4] Since BCC of the prostate was first reported in 1974 as adenoid cystic carcinoma (ACC), a part of the morphologic continuum of BCC,[2] approximately 50 cases have been reported worldwide.[2] However, just one case has been reported in the domestic literature.[5]

We report the case of a patient in whom BCC was diagnosed during transurethral resection for intractable LUTS after 6.5 years of medical treatment for benign prostatic hyperplasia (BPH) and who ultimately underwent radical retropubic prostatectomy.

CASE REPORT

A 54-year-old male patient visited the outpatient department for LUTS 7 years ago. He had no remarkable past medical history or familial medical history. On digital rectal examination (DRE), the prostate was found to be enlarged (50 ml) with an indurated and slightly firm consistency without tenderness. The International Prostate Symptom Score and quality of life score were 28 and 5, respectively, and the peak urinary flow rate was 8 ml/s. Urinalysis was normal, but the expressed prostatic secretion revealed many white blood cells per high power field. The serum PSA was 3.5 ng/ml and the prostate volume measured with transrectal ultrasonography was 50 ml. With the clinical impression of chronic prostatitis and BPH, an antibiotic was administered for 2 months and an α-blocker was started and used continuously.

Two years later, his PSA and prostate volume increased to 9.0 ng/ml and 60 ml, respectively. Sextant biopsy of the prostate was performed with the pathologic diagnosis of chronic prostatitis. Subsequently, PSA was followed up periodically, and ranged from 3.8 to 7.5 ng/ml. At 6 years from his first visit, his PSA increased to 10.79 ng/ml. A repeat 10-core prostate biopsy was performed, and the pathologic report turned out to be BPH. At the time of the biopsy, prostate volume was 108 ml, and no hypoechoic lesions were found in the peripheral

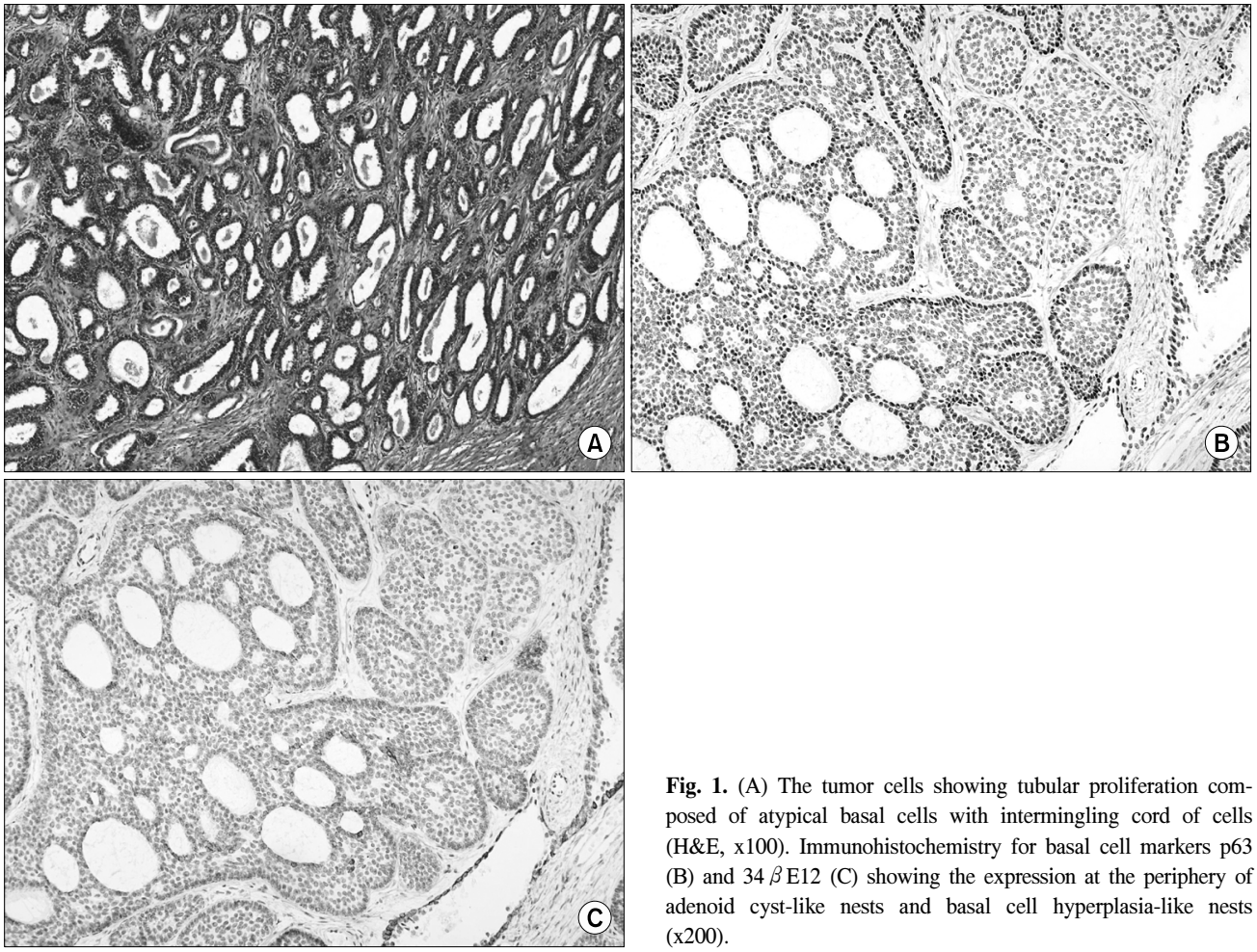

Fig. 1. (A) The tumor cells showing tubular proliferation composed of atypical basal cells with intermingling cord of cells (H&E, x100). Immunohistochemistry for basal cell markers p63 (B) and 34 β E12 (C) showing the expression at the periphery of adenoid cyst-like nests and basal cell hyperplasia-like nests (x200).

zone on transrectal ultrasonography. Persistent LUTS despite continuous administration of the α-blocker and a rapidly enlarging prostate led to the necessity for surgical treatment. At 6.5 years from the first visit, transurethral resection of prostate (TURP) was performed. Microscopic examination of the pathologic specimen showed nests and trabeculae of tumor cells punctuated by cribriform spaces forming tubules. Atypical basal cells with mitosis were present in some areas. On immunohistochemistry, positive reactions were found against basal cell markers p63 and 34 β E12, and no reaction was found against cytokeratin 7 and 20 (Fig. 1). The pathologic findings were consistent with BCC of the prostate. Magnetic resonance imaging was performed for staging, which revealed a 3x2 cm mass in the transition zone surrounding the previous TURP site. The mass showed a low signal intensity on T1-weighted image and an intermediate signal intensity on T2-weighted image. The peripheral zone was normal and no local invasion or lymphadenopathy was found (Fig. 2). A whole-body bone scan showed no evidence of bony metastasis. Because we suspected BCC confined to the prostate, a radical retropubic prostatectomy was performed 12 weeks after TURP. The operative findings were unremarkable except for adhesions around the apex and the base of the prostate and seminal vesicles. Gross examination of the bisected prostate showed a white and fleshy mass arising from the transition zone and surrounding the tissue defect created by the previous TURP. Microscopic examination revealed an ill-defined BCC confined to the prostate along with multifocal prostatic intraepithelial neoplasia (Fig. 3). No metastasis was found in 22 bilateral obturator and external iliac lymph nodes.

At 4 months after surgery, the patient appeared to be free of cancer with a PSA level of 0.009 ng/ml. Because the PSA level is generally normal or only slightly elevated in patients with BCC, long-term follow-up should include radiographic

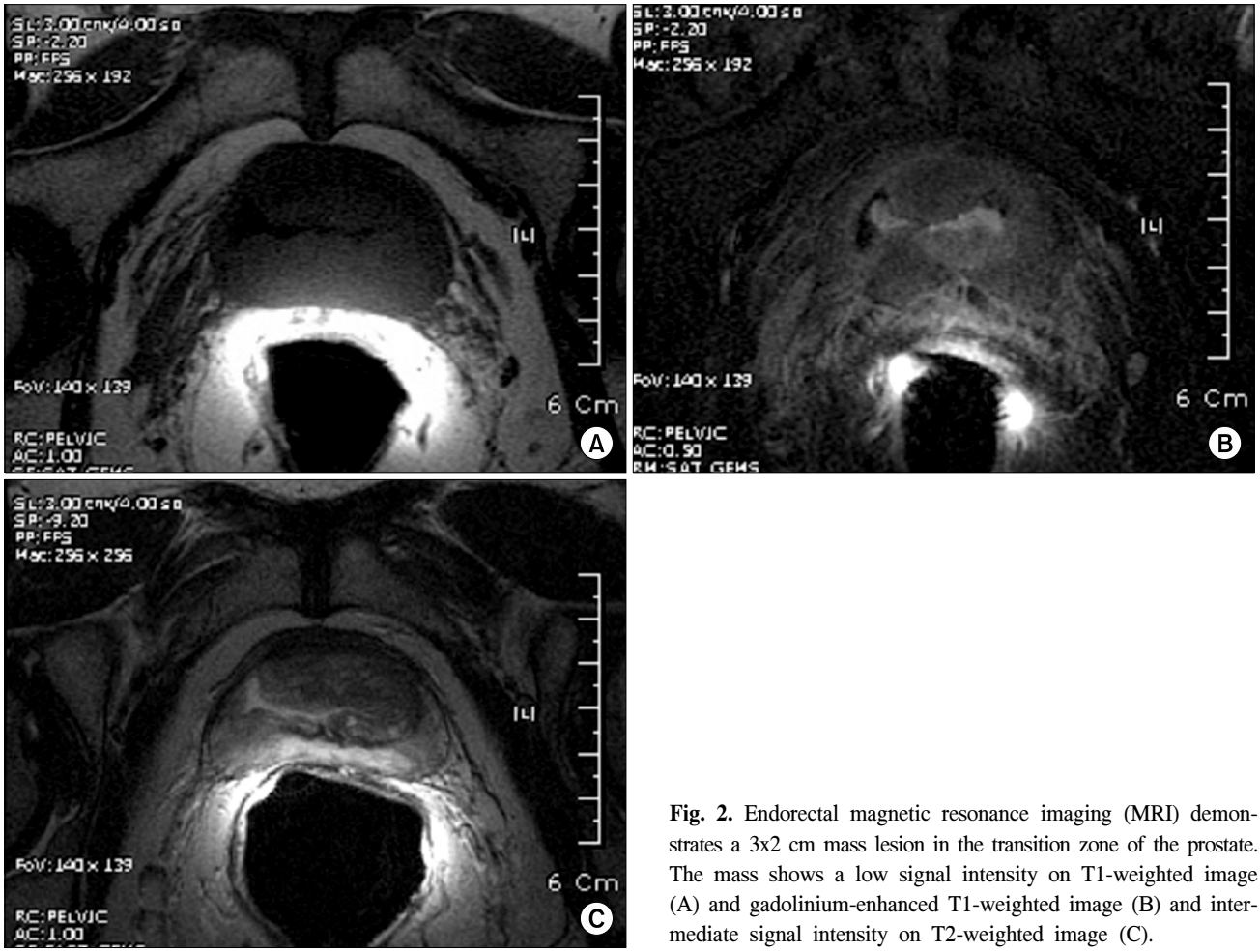

Fig. 2. Endorectal magnetic resonance imaging (MRI) demonstrates a 3x2 cm mass lesion in the transition zone of the prostate. The mass shows a low signal intensity on T1-weighted image (A) and gadolinium-enhanced T1-weighted image (B) and intermediate signal intensity on T2-weighted image (C).

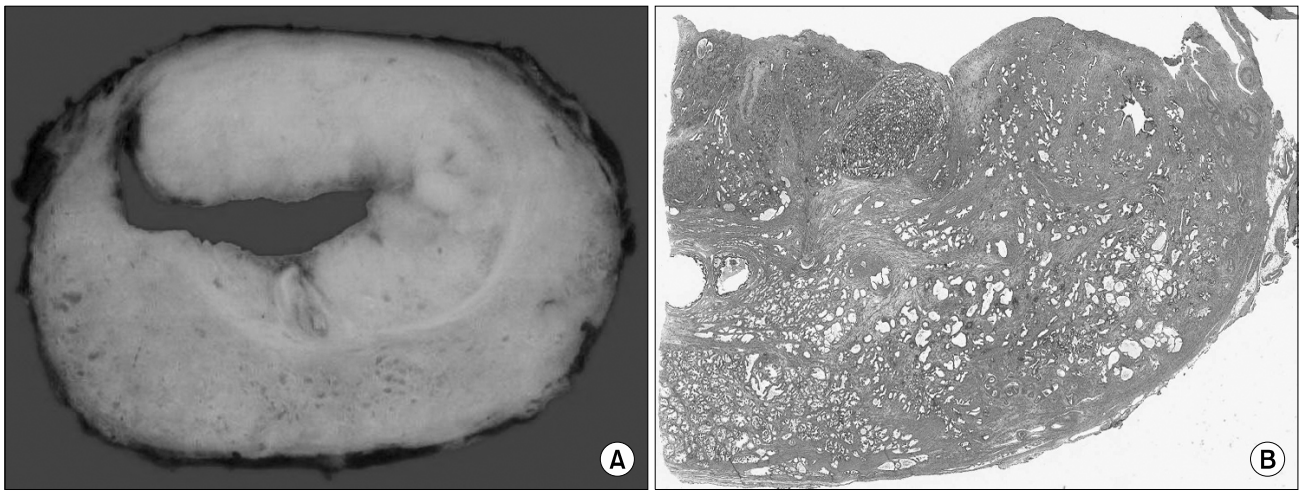

Fig. 3. (A) Bisected prostate demonstrates an ill-defined, whitish gray, solid, nodular growth of tumor located at the transition zone around the tissue defect caused by the previous transurethral resection. (B) Microscopic finding of the prostate showing the tumor composed of increased tubular structures that are confined to the transition zone (H&E, x1).

tests as well as PSA assessment.

DISCUSSION

The prostatic epithelium is composed of secretory, neuroendocrine, and basal cells. Basal cells may act as stem cells of the prostate gland with the potential to differentiate along divergent pathways and keep the secretory cells under hormonal regulation.[6,7] Lesions of basal cells in the prostate gland span a wide range from benign basal cell hyperplasia through various ranges of atypia to BCC, which includes the types termed prostate basaloid carcinoma (BC) and ACC.[2,3,7-9]

Unlike adenocarcinoma, which is usually grossly yellow, BCC is white and fleshy. It is accompanied by microcysts and a poorly defined infiltrative edge.[1,4,10] Although BCC usually involves the transition zone, some develop in the peripheral zone.[2-4,10] Microscopically, BCC can have either a predominant basaloid pattern like that of skin or cystically dilated acini and cells arranged in cribriform spaces surrounding eosinophilic-hyaline basement membrane-like material or basophilic mucinous secretion. Occasional glandular, trabecular, and solid areas can be found.[1] Histologic criteria for malignancy that distinguish it from basal cell hyperplasia include an infiltrative pattern, extraprostatic extension, perineural invasion, necrosis, and stromal desmoplasia.[8,9] Immunoreactivity of the present tumor for high molecular weight cytokeratin (34βE12) and p63, which are indicators of basal cell origin, coupled with the absence of immunoreactivity for cytokeratin 7 and 20, which are typically expressed in urothelial carcinoma, strongly favor a diagnosis of BCC.[1,4,7,10]

BCC generally occurs in elderly men,[2] but may involve patients in a wide age range (28 to 78 years) with a mean age of 50 years.[1,4] Patients usually present with LUTS including nocturia, urgency, bladder outlet obstruction symptoms, and acute urinary retention. DRE shows an enlarged and indurated prostate gland.[1,4] The serum PSA is usually normal or slightly increased,[1] but an increase in serum PSA in patients with BCC usually indicates an accompanying conventional acinar adenocarcinoma.[4] No preoperative imaging technique has sufficiently provided findings specific to detect this type of prostate tumor.[1] In most instances, the diagnosis is made after TURP or simple enucleation performed for obstructive symptoms.[4,9]

Although BCC shows mostly an indolent course, a small subset behaves aggressively with local recurrences and distant metastases. Interestingly, metastases involve liver, lung, and bowel but not bone, as is commonly observed in conventional prostate acinar adenocarcinomas.[1,4] Ayyathurai et al[2] reported that in 7 patients who developed distant metastases, 6 were ACC and 1 was BC. Also, 4 patients with ACC and 1 with a mixed pattern tumor developed local recurrence. None of those with BC developed local recurrence. But Segawa et al[7] reported that BC shows more aggressive features than AC. Also, Ali and Epstein[3] observed that central necrosis, higher expression of Ki67, and lower expression of basal cell markers are indicators of aggressive behavior.

Although an optimal management algorithm is difficult to formulate because the number of reported cases is small, radical surgery is the preferred first-line management option. Current evidence suggests close and long-term follow-up due to the possibility of local recurrences and distant metastases.[1,2,10] Radiation and chemotherapy may be helpful, but results are inconsistent.[1]

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