Localized Amyloidosis of Seminal Vesicle and Vas Deferens
- Report of Two Cases -

We reported localized amyloidosis involving seminal vesicles and vasa deferentia, which was found in two patients with prostatic adenocarcinoma. A 60-yr-old (Case 1) and a 59-yr-old (Case 2) man came to our hospital with elevation of serum prostate-specific antigen (PSA) and biopsy proven carcinoma, respectively. MRI revealed multiple irregular foci of low signal intensity in the prostates as well as in both seminal vesicles and vasa deferentia on T2-weighted imaging, suggesting prostatic carcinoma with extension to both seminal vesicles and vasa deferentia in both cases. Under the clinical diagnosis of stage III prostatic adenocarcinoma, a radical prostatectomy was performed in both patients. Microscopically, Gleason score 7 adenocarcinoma was observed in both patients. In addition, isolated amyloidosis of both seminal vesicles and vasa deferentia was found without carcinoma involvement. Localized amyloidosis in the seminal vesicles, which is considered as senile process, has been occasionally reported in the autopsy and in the surgical specimens. Amyloid deposition in the vas deferens has also been reported in the literature, however, the deposition mimicking extension of carcinoma has not been reported. In this report, two cases of isolated amyloidosis of the seminal vesicles and vasa deferentia are described with electron microscopic study and literature review.

Key Words: Amyloidosis, Seminal Vesicles, Vas Deferens, Prostatic Neoplasms

INTRODUCTION

Isolated amyloid deposition has been reported in almost every organ systems including the genitourinary tract (1). Localized amyloidosis in the seminal vesicle is an unusual finding and most of the cases have been described in autopsy series with its incidence being about 9-16% (1-3). The incidence of localized amyloidosis of the seminal vesicles increases with age, representing a form of senile amyloidosis (3). Some authors document its incidental detection in the surgical specimen and the incidence rate is also very low (4, 5). Amyloid deposition tends to be nodular and subepithelial. Involvement of vessel walls is characteristically not seen in localized form.

In cases associated with genitourinary carcinomas, the amyloid deposition in the seminal vesicle can be misdiagnosed as tumor invasion on magnetic resonance imaging (MRI) findings (6, 7). Our cases were also suggested extension of the prostatic adenocarcinoma both to seminal vesicles and vasa deferentia on the MRI study. However, these suspicious lesions in both seminal vesicles and vasa deferentia turned out to be localized amyloid deposition in these organs. We reported these cases because amyloid deposition in vasa deferens is extremely rare and amyloid deposition in the vas deferens simulating tumor invasion has not been reported. Electron microscopic analysis was performed on these cases and we made a review of the literature.

CASE REPORT

Case 1

A 60-yr-old male patient who has been taken medicines for treatment of hypertension and benign prostatic hyperplasia came to our hospital, because of the recent elevation of serum prostate-specific antigen (PSA) level (5.4 ng/mL). At that time, serum free PSA level was 0.76 ng/mL (14.1% of total PSA level). On digital rectal examination of the prostate, a poorly-defined firm nodule was palpable in the right side. MRI was performed at an outside hospital and revealed multiple irregular foci of low signal intensity in the prostate as well as both seminal vesicles and vasa deferentia on T2-weighted imaging, suggesting of prostatic carcinoma extension both to seminal vesicles and vasa deferentia (Fig. 1A). Based on the clinical diagnosis of T3b prostatic...
carcinoma, a radical prostatectomy was performed.

On gross examination of the radical prostatectomy specimen, the prostate was slightly enlarged (5.0 × 4.5 × 3.0 cm) and weighed 35.5 g. Two well-defined ovoid masses (1.5 × 0.9 × 0.7 cm and 1 × 0.6 × 0.3 cm) were present in the right and left sides of the prostate, abutting the prostatic capsule. The tumor occupied less than 10% of the total prostate. The cut surfaces of the masses were yellowish tan and firm. Neither hemorrhage nor necrosis was seen. On the serial sections of both seminal vesicles and vasa deferentia, they were focally thickened and firm with yellowish tan discoloration, suggesting tumor extension (Fig. 2A). Micro-

Fig. 1. Multiple irregular foci of low signal intensity are observed in the prostate as well as in the area of both seminal vesicles and vasa deferentia (arrow) on the T2-weighted MR imaging, suggesting tumor extension of prostatic carcinoma to the seminal vesicles and vasa deferentia. (A) Case 1. (B) Case 2.

Fig. 2. (A) There are multiple foci of yellowish tan and firm nodular lesions, both in seminal vesicles. These nodular lesions are better illustrated on the right side of seminal vesicles (short arrow). In this picture, a tip of prostatic carcinoma mass is seen in the left lower corner (long arrow). The remaining prostatic parenchyma shows benign nodular hyperplasia. (B) Microscopically, prostatic masses are confirmed to be adenocarcinoma of Gleason score 7 (4+3) (H&E, ×40).
scopically, two prostatic masses were diagnosed as Gleason score 7 (4+3) adenocarcinoma (Fig. 2B). The tumor was confined within the prostate with no extraprostatic extension. In both vasa deferentia and seminal vesicles, nodular deposits of amorphous eosinophilic material were seen beneath the epithelial layer with no evidence of prostatic carcinoma involvement (Fig. 3A, B). Pelvic lymph nodes were free of tumor. The final TNM stage of the prostatic carcinoma was T2bN0M0 (stage II). He is alive and well 6 months after surgery. His most recent serum PSA is less than 0.2 ng/mL.

Case 2

A 59-yr-old male patient has been taken medicines for symptomatic treatment of right flank pain and gross hematuria originated from a known renal multicystic disease for 4 yrs. However, the patient did not have an evidence of renal failure nor receive a hemodialysis treatment. Past history was noncontributory with right ureterolithotomy 20 yrs ago and the resection of unknown brain tumor 9 yrs ago. He was admitted in an outside hospital due to the recent aggravation of the low urinary tract symptoms, including urinary...
frequency and nocturia for 2 yrs. Prostatic adenocarcinoma was detected on the prostatic needle biopsy. He was transferred to our hospital for the operation of the known prostatic carcinoma. On admission, serum PSA and free PSA level were 7.4 ng/mL and 1.4 ng/mL (18.9% of total PSA level), respectively. On digital rectal examination of the prostate, no palpable nodule was present. MRI of the prostate, both seminal vesicles and vasa deferentia showed similar findings with those of Case 1 (Fig. 1B), except a focus of high signal intensity both in T1- and T2-weighted imaging in this case, suggesting of inprostatic hemorrhage. No change of the renal cysts was observed. Like Case 1, a radical prostatectomy was performed, based on the clinical diagnosis of T3b prostatic carcinoma.

On gross examination of the radical prostatectomy specimen, the prostate was slightly enlarged (5.0 × 4.8 × 3.5 cm) and weighed 49 g. Two well-defined ovoid masses (1.2 × 1.2 × 1.1 cm and 0.7 × 0.4 × 0.3 cm) were present both in the left and right side of the prostate, abutting the prostatic capsule. The tumor occupied less than 10% of the total prostate. The cut surfaces of the prostatic masses and both seminal vesicles and vasa deferentia were similar to those of Case 1. Microscopically, two prostatic masses were diagnosed as Gleason score 7 (3+4) adenocarcinoma. The tumor was confined within the prostate with no extraprostatic extension and pelvic lymph node metastasis. In this case, nodular deposits of amorphous eosinophilic material were seen beneath the epithelial layer in the ejaculatory ducts as well as both vasa deferentia and seminal vesicles with no evidence of prostatic carcinoma involvement. The final TNM stage of the prostatic carcinoma was also T2bN0M0 (stage II) like Case 1. This patient is recently discharged from the hospital after operation with uneventful outcome.

The deposits of both Cases 1 and 2 exhibited Mahogany red color with Congo red amyloid staining and apple green birefringence under a polarized microscope. By electron microscopic examination, these deposits were composed of fine, 7.5 to 10 nm diameter, rigid, and non-branching filaments of variable length with haphazard arrangement (Fig. 3C, D), indicating amyloid fibers. This amyloid material was observed in the subepithelial connective tissue without vascular wall deposition.

**DISCUSSION**

Localized amyloidosis in the seminal vesicle has been uncommonly reported as an incidental finding in the autopsy (1-3). This process is considered as a senile change because of the increase incidence with age. Some authors rarely document the incidental amyloid deposition in the seminal vesicle in the surgical specimens (4, 5). Localized amyloidosis in the seminal vesicle occurs bilaterally and the deposits tend to be nodular and subepithelial with no vascular involvement. Most of the patients presented asymptomatically. Two symptomatic cases, one each with hematospermia and enlargement of the seminal vesicle, were reported in the literature (8). Amyloid deposition in vas deferens, however, is extremely rare and there is only one paper reporting three cases of localized amyloidosis both in seminal vesicles and vasa deferentia (2). All these cases were incidentally detected. Our cases will be the fourth and fifth reported cases of localized amyloidosis involving vasa deferens.

It has been suggested that this type of amyloid is derived from a secretory exocrine product of the normal seminal vesicle epithelium (9). Unger et al. (5) demonstrated that the prior luteinizing hormone-releasing hormone (LHRH) treatment may act as a seminal vesicle epithelial stimulant for elaboration of amyloid. One of our patients (Case 1) has been taken unknown medicines for benign prostatic hyperplasia during the last 3 yrs at an outside hospital and this medication may be related with the amyloid deposition.

Various organs, including lung, larynx, skin, tongue, eye and genitourinary tracts have been reported to be the sites of localized amyloidosis which amyloid involves subepithelial connective tissue without involving vascular wall. Unlike systemic amyloid deposition, which is mostly composed of AL (lambda chain) type, the amyloid of the seminal vesicle was negative for the known amyloidogenic substances, such as AA protein, AL (kappa and lambda chains), transthyretin (prealbumin), β2-microglobulin, β-amyloid protein, cystatin C, calcitonin, and amylin by immunohistochemical stainings (10-12). Tsutsumi et al. (10) proposed that the lactoferrin was the major constituent in localized senile amyloidosis of seminal vesicle.

Some authors (6, 7) reported that the localized amyloidosis of the seminal vesicle was clinically and radiologically similar to direct tumor invasion from bladder or prostate cancer, because these deposited areas also displayed low signal intensity on T2-weighted imaging of MRI. To differentiate amyloid deposition from tumor invasion of seminal vesicle, an administration of the gadopentetate dimeglumine may be useful since amyloid deposition shows no enhancement unlike tumor involvement (6).

We report very unusual cases of the localized amyloidosis in both vasa deferentia as well as seminal vesicles, which were found in the radical prostatectomy specimens. Our cases were originally interpreted as the extension of prostatic adenocarcinoma to seminal vesicles and vasa deferentia based on the preoperative radiologic studies. Therefore, a possible seminal vesicle and/or vasa deferens involvement of prostatic or bladder cancer by image analysis is not necessarily true involvement of tumor on pathologic examination. Therefore, a possibility of localized amyloidosis of the seminal vesicle and/or vasa deferens should be considered in prostatic cancer cases, which showed abnormal MR imagings in the seminal vesicles and vasa deferentia.
REFERENCES

1. Coyne JD, Kealy WF. Seminal vesicle amyloidosis: morphological, histochemical and immunohistochemical observations. Histopathology 1993; 22: 173-6.

2. Goldman H. Amyloidosis of seminal vesicles and vas deferens. Primary localized cases. Arch Pathol 1963; 75: 94-8.

3. Pitkanen P, Westermark P, Cornwell GG, Murdoch W. Amyloid of the seminal vesicles. Am J Pathol 1983; 110: 64-9.

4. Seidman JD, Shmookler BM, Connolly B, Lack EE. Localized amyloidosis of seminal vesicles: report of three cases in surgically obtained material. Mod Pathol 1989; 2: 671-5.

5. Unger PD, Wang Q, Gordon RE, Stock R, Stone N. Localized amyloidosis of the seminal vesicle. Possible association with hormonally treated prostatic adenocarcinoma. Arch Pathol Lab Med 1997; 121: 1265-8.

6. Jager GJ, Ruijter ET, de la Rossette JJ, van de Kaa CA. Amyloidosis of the seminal vesicles simulating tumor invasion of prostatic carcinoma on endorectal MR images. Eur Radiol 1997; 7: 552-4.

7. Kaji Y, Sugimura K, Nagaoka S, Ishida T. Amyloid deposition in seminal vesicles mimicking tumor invasion from bladder cancer: MR findings. J Comput Assist Tomogr 1992; 16: 989-91.

8. Krane RJ, Klugo RC, Olsson CA. Seminal vesicle amyloidosis. Urology 1973; 2: 70-2.

9. Cornwell GG, Westermark GT, Pitkanen P, Westermark P. Seminal vesicle amyloid: The first example of exocrine cell origin of an amyloid fibril precursor. J Pathol 1992; 167: 297-303.

10. Tsutsumi Y, Serizawa A, Hori S. Localized amyloidosis of the seminal vesicle: identification of lactoferrin immunoreactivity in the amyloid material. Pathol Int 1996; 46: 491-7.

11. Suess K, Moch H, Epper R, Koller A, Durmuller U, Mihatsch MJ. Heterogeneity of seminal vesicle amyloid. Immunohistochemical detection of lactoferrin and amyloid of the prealbumin-transthyretin type. Pathologe 1998; 19: 115-9.

12. Esslimani M, Serre I, Granier M, Robert M, Baldet P, Costes V. Urogenital amyloidosis: clinico-pathological study of 8 cases. Ann Pathol 1999; 19: 487-91.