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ADENOMYOSIS OF THE SEMINAL VESICLE WITH HEMATOSPERMIA

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A 62-year-old man presented in 1987 with hematospermia. No abnormal findings were observed by cystourethroscopy. Ultrasound showed the enlargement of the right seminal vesicle. The right seminal vesicle could not be visualized by seminal vesiculography. Computed tomographic scan revealed a homogeneous mass at the right dorsolateral aspect of the prostate. Surgical exploration was performed. The prostate and bilateral seminal vesicle were resected. Pathological diagnosis was adenomyosis of the right seminal vesicle.

Key words: Hematospermia, Seminal vesicle, Adenomyosis

INTRODUCTION

Adenomyosis is an infrequent urological disease. It is characterized by many glandular structures surrounded by smooth muscle hypertrophy. Herein we report a case of adenomyosis of the seminal vesicle that caused hematospermia.

CASE REPORT

A 62-year-old man was admitted to the Kobe Steel Hospital because of hematospermia in October, 1987. Physical examination showed normal male habitus and external genitalia. On digital examination, the prostate was walnut sized and felt elastically hard. Urinalysis and urinary culture results were negative and the routine hematology and chemistry analysis results were normal. Cytology of semen did not reveal any malignant findings. He had been treated with antibiotics and anti-inflammatory drugs under the diagnosis of seminal vesiculitis with improvement. In June, 1988, hematospermia recurred. Cystourethroscopy revealed no abnormal findings. Seminal vesiculography was performed and the right seminal vesicle could not be visualized (Fig. 1). CT scan of the pelvis revealed a homogeneous mass at the right dorsolateral aspect of the prostate (Fig. 2). Its margin was well circumscribed. The prostate and left seminal vesicle appeared to be within normal limits. Transrectal longitudinal ultrasonography showed the enlargement of the right seminal vesicle with a distinct margin (Fig. 3). The ultrasonographically guided transrectal needle biopsy of the seminal vesicle showed no evidence of malignancy. The patient underwent surgical exploration. The prostate and bilateral seminal vesicle were resected. The right seminal vesicle was enlarged and firm. Microscopically, many glandular structures with hyperplastic cuboidal epithelium were present in the hypertrophic muscular bundles. No spermatozoa were seen within the lumina of these glands. Serial sectioning revealed no communication between the proper lumen of the seminal vesicle and tubules of glandular network (Fig. 4). The final pathological diagnosis was adenomyosis of the right seminal vesicle.

The postoperative course was unremarkable.

DISCUSSION

Adenomyosis itself consists of a glandular structure surrounded by hypertrophic smooth muscle bundles. It is a very rare disease in urological aspect. Gal, R. et
al reported adenomatous hamartoma of the small intestine. Also Hellen et al. described mesonephric hamartoma of the seminal vesicle similar to adenomatous change. In the spermatic cord, only two cases similar to this change have been reported. To the best of our knowledge, we believe that this is the first report of adenomyosis of the seminal vesicle. Hatcher et al. reported fibromuscular hyperplasia of the seminal vesicle. In our case, glandular hyperplasia was recognized but the hyperplasia of fibrous tissue was not observed. Therefore, our case was not of fibromuscular hyperplasia.

Unfortunately, we could not make an exact preoperative diagnosis and the diagnosis of adenomyosis was made only by surgical exploration. It is important to distinguish adenomyosis from other benign diseases such as seminal vesicle cyst, fibroma, myoma, cystic adenoma, inflammation and malignant lesion, adenocarcinoma, and sarcoma.

With respect to the chief complaint of hematospermia the bleeding is mostly derived from the prostate, seminal vesicle, testis, epididymis and urethra. Rass reported as the main causes of hematospermia, seminal vesiculitis, urethral stenosis, hypertension, purpura, tuberculosis, and malignant tumor. However, the definite cause can be rarely found and most of it is idiopathic. Adenomyosis also should be taken into consideration.

Finally, the pathogenesis of adenomyosis of the seminal vesicle may be related to some changes occurring in the developing mesonephric duct, since the seminal vesicle develops from the diverticula from the caudal portion of the mesonephric duct.
Fig. 4. A, The right seminal vesicle was markedly enlarged, measuring 7.7x2.2x2.8 cm.
B, Many glandular structures were scattered within the hypertrophic muscles (×100).
C, The surrounding hypertrophic muscles (×400).

However, it remains still unknown whether this tumor is congenital or acquired.

REFERENCES


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血精液を主訴とした精巣腺筋症の1例

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62歳，男性。血精液を主訴として来院。精巣炎を疑い抗菌剤投与を行い軽快した。その後再発したため，尿道膀胱鏡を行ったが異常を認めなかった。精巣造影では右精巣は露出されず，CTにて前立腺右後側に内容均一な腫瘤を認めた。経直腸超音波検査にて右精巣の腫大を認め，超音波ガイド下生検を行ったところ，明らかな異常所見を認めなかった。診断目的で手術を行い前立腺および精巣を摘出した。病理学的診断は精巣腺筋症であった。

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