SPERMATOCYTIC SEMINOMA: A CASE REPORT

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An 81-year-old man with a painless enlargement of the right testis which developed 11 years ago was treated with right orchietomy without any combined therapy in 1981. This tumor pathologically proved to be spermatocytic seminoma. The patient has been well and has shown no evidence of recurrence for the past 5 years.

Key words: Testicular tumor, Spermatocytic seminoma

INTRODUCTION

Spermatocytic seminoma, which is clinically-pathologically distinct from classical seminoma, is relatively rare. We report herein a case of spermatocytic seminoma, which we believe is the fifth case in Japan.

CASE REPORT

An 81-year-old man with a painless enlargement in the right scrotum was seen on July 23, 1974, 3 months after its onset. A puffy right testis, the size of a goose egg, was found. We advised orchietomy but the patient then refused it. Since the size of the mass increased gradually, he returned for an operation on June 11, 1981. The whole right scrotum which was fluctuant and could be partially transilluminated was the size of a child's head. The testis was found to be replaced by a painless mass approximately 15 cm along the major axis having a hard consistency and uneven surface. The right epididymis could not be differentiated from the mass, but the spermatic cord appeared to be normal on palpation. Ultrasonography of the mass showed the so-called mixed pattern of echogenic and sonolucent areas. The existence of an echo-free area around the mass suggested the association of a hydrocele testis (Fig. 1-A, B). Laboratory data showed no significant findings except for a mild elevation in lactate dehydrogenase (497 IU/l). Tumor markers such as α-fetoprotein and human chorionic gonadotropin were within normal limits. Radical orchietomy was performed on July 3, 1981. The excised testicular tumor associated with a hydrocele testis was 14 ×11×4.5 cm, weighed 400 g, had an uneven surface, and had a firm elastic consistency. Macroscopic examinations of the cut surface revealed that the testis was replaced by gray-white to yellow-white, solid and partially cystic masses. Several nodules had areas of hemorrhage and necrosis (Fig. 2). The epididymis and spermatic cord were not remarkable. Microscopically, the testis was replaced by tumor cells which could be classified as 1) cells having round nucleoli and pale vesicular cytoplasm; 2) cells having dark stained nucleoli due to rich chromatin and acidophilic cytoplasm; and 3) giant cells apparently distinct from the previous groups. A delicate stroma with scant lymphocyte infiltration was seen (Fig. 3-A, B). Moreover, PAS staining of the tumor cells was negative. Based on these pathological findings, the patient was diagnosed as having spermatocytic seminoma. He was discharged and has been followed up without additional treatment because image diagnosis and laboratory data has shown no evidence of metastasis. For the past 5 years he has been well and has shown no evidence of recurrence.
DISCUSSION

Spermatocytic seminoma, originally described by Massoni\(^1\), is a relatively rare, locally invasive, slow-growing, malignant tumor which is morphologically and clinically distinct from classical seminoma. There are approximately 100 cases in the literature, and our case is believed to be the 5th case in Japan (Table 1). Although the incidence of spermatocytic seminoma in seminoma has been reported to be between 1.7 and 12\%, some of the spermatocytic seminoma might have been diagnosed as classical seminoma. In fact, Rosai\(^2\) reclassified as spermatocytic se-

![Fig. 1. Ultrasonograms of the testis. A, the mixed pattern of echogenic and sonoluent areas. B, the sonoluent area around the testis implying a complication of a hydrocele testis.](image)

![Fig. 2. Gross appearance of the testicular tumor showing nodularity, foci of necrosis, hemorrhage and cystic degeneration.](image)

![Fig. 3. Photomicrograph showing A, the three types of cells in spermatocytic seminomas (H & E, x400) and B, stroma with scant lymphocyte infiltration (H & E, x200).](image)

<table>
<thead>
<tr>
<th>Case No</th>
<th>Author</th>
<th>Yr. publ.</th>
<th>Pt. age, yr.</th>
<th>Preop. duration</th>
<th>Size &amp; weight of tumor</th>
<th>Therapy</th>
<th>Metastases</th>
<th>Follow-up (postop.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Tomoyoshi(2)</td>
<td>1968</td>
<td>70</td>
<td>4M</td>
<td>*L 50x35x25m, 52g</td>
<td>O+C</td>
<td>(-)</td>
<td>A &amp; W 1 yr</td>
</tr>
<tr>
<td>2</td>
<td>Nishi(3)</td>
<td>1970</td>
<td>32</td>
<td>1M</td>
<td>*R 52x45x30m, 57g</td>
<td>O+R</td>
<td>(-)</td>
<td>A &amp; W 1 yr and 4 mos</td>
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<tr>
<td>3</td>
<td>Takayama(4)</td>
<td>1979</td>
<td>49</td>
<td>1M</td>
<td>*L 60x50x25m, 65g</td>
<td>O+RPLND</td>
<td>(-)</td>
<td>A &amp; W 4 yrs</td>
</tr>
<tr>
<td>4</td>
<td>Sato(5)</td>
<td>1984</td>
<td>45</td>
<td>2Y</td>
<td>*R 60x45x27m, 67g</td>
<td>O+RPLND+C</td>
<td>(-)</td>
<td>A &amp; W 3 yrs and 3 mos</td>
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<tr>
<td>5</td>
<td>Author</td>
<td>1986</td>
<td>81</td>
<td>6Y</td>
<td>R 140x110x45m, 400g</td>
<td>O</td>
<td>(-)</td>
<td>A &amp; W 5 yrs</td>
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</table>

* Synchronous, **asynchronous.
minoma 6 out of 81 cases which were diagnosed as classical seminoma. Classical seminoma mainly occurs in 20 to 30-year-old patients, whereas spermatocytic seminoma mainly develops in patients over 40 years old. There is no difference in the site of occurrence, and the incidence of bilateral occurrence is significantly higher in spermatocytic seminoma as compared to classical seminoma (6~10% vs 2~3%). Two of the 5 cases reported in Japan were bilateral (Table 1). The tumor does not arise in undescended testicles nor in organs other than the testicle. Teratomas are never associated with the tumor.

Histologically, spermatocytic seminoma has scant intervening stroma, few scattered lymphocytes around the blood vessels and no granulation or necrotic foci. An important pathological diagnostic feature of the tumor is the marked difference in size between the tumor cells (see Fig. 3). The neoplastic elements can be roughly divided into three main types in which the intracytoplasmic glycogen is absent. Masson\(^1\) suggested that these tumors originate from spermatocytes because of the microscopic similarity of the chromatin arrangement. Although Rosai\(^7\) supported this hypothesis with morphological observations under an electron microscope, Talerman and coworkers reported some findings morphologically\(^9\) and histochemically\(^9\) against it. Regarding malignant potency, only one histologically-proven metastatic case could be found in the literature\(^10\) : the patient showed bilateral axillary lymph node metastases 6 months after orchiectomy, and died 3 months later in spite of irradiation and chemotherapy. Generally speaking, the prognosis following orchiectomy is thought to be considerably good. Although spermatocytic seminoma seems to be radiosensitive\(^11\), there is no evidence indicating that post-operative radiation is effective. Postoperative radiation may increase morbidity and not appreciably lengthen survival, especially in elderly patients like our case. Patients, however, should be followed up because several cases with asynchronous bilateral occurrence have been reported\(^6,11\).

**REFERENCES**

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Spermatocytic Seminoma の 1 例

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症例は81歳の男子、1974年5月より右陰嚢内部の腫大に気付くも放置。1981年6月、同部は径約 15 cm にまで腫大したため、手術を希望して入院した。右高位精巣術を施行したところ、陰囊水腫を合併した重篤 400 g の腫瘍が摘出された。組織学的には精巣は大小不同的3群に分けられる細胞構造で置換され、これらの細胞中は、グリコーゲン（—）であった。また間質へのリンパ球浸潤もきわめて乏しいなどの所見より、

spermatocytic seminoma と診断された。1946年 Masson が報告して以来、欧米では約100例の報告をみるが、本邦では自験例が第5例目と思われる。これまで組織学的に明らかな転移を認めめた報告はわずかに 1例をみるにとどまり、一般にその予後は classical seminoma に比べて良好とされている。自験例は高齢であることを考慮して、術後に放射線療法を施行せずに、経過観察し、5年経過した現在、再発の兆候はなく健在である。

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