

A CASE OF EXTREMELY ADVANCED PROSTATE CANCER PRESENTING HEMOLYTIC JAUNDICE

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A 62-year-old man having prostate cancer presenting with hemolytic jaundice is reported. The hemolytic jaundice was cured by diethylstilbestrol diphosphate (DES-P) treatment, but the patient died one year and two months later from relapse of prostate cancer. An autopsy confirmed the diagnosis of prostate cancer with multiple metastases. Sudden anemia caused by bone metastasis might have caused the hemolytic jaundice. DES-P cured bone metastasis and then improved anemia when associated with transfusion. Consequently, hemolytic jaundice was cured.

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Key words: Prostate cancer, Hemolytic jaundice

INTRODUCTION

Advanced prostate cancer is manifested by many symptoms¹⁾. However, jaundice due to prostate cancer appears only rarely in the literature²⁻⁴⁾. The usual cause of jaundice due to prostate cancer is obstructive jaundice caused by a metastatic mass²⁻⁴⁾. We have reported here a case of extremely advanced prostate cancer presenting with hemolytic jaundice, which is the first case to be reported in the literature.

CASE REPORT

A 62-year-old man complaining of severe low back pain and anorexia was transferred to the Urological Clinic from the Department of Medicine. He was first seen in Orthopaedic Surgery suffering from rheumatoid arthritis in 1985, and had been taking predonin in another hospital since 1982. He had complained of low back pain on February 3, 1989. At that time laboratory findings were: red blood cell count (RBC) 449×10^4 , hemoglobin (Hb) 12 g/dl, thrombocyte 22×10^4 , erythrocyte sedimentation rate 61 mm/hour. Because he was not able to walk, and complained of severe low back pain on March 7, he was admitted to Orthopaedic Surgery. At that time,

his conjunctiva was icteric. Neurological examination showed neither sensory deficit nor motor disturbance. He was transferred to the Department of Medicine on March 12, since leukemia was suspected. He received a 600 ml transfusion, since anemia was extreme (RBC 240×10^4 , Hb 6.6 g/dl). A sternal bone marrow puncture was performed on March 13. Cytology showed poorly differentiated adenocarcinoma, suggesting prostate cancer. Therefore, he was finally transferred to our clinic on March 17. He was 157 cm tall and weighed 46 kg. The conjunctiva was anemic and icteric. There was no hepatosplenomegaly. The prostate was firm and enlarged. A prostate needle biopsy was performed on March 23, showing poorly differentiated adenocarcinoma (Fig. 1). A bone scan showed multiple metastases (Fig. 2). Prostatic specific antigen (PSA) was 69 ng/ml and prostatic acid phosphatase (PAP) was 37 ng/ml. Computed tomographic (CT) scan revealed neither liver mass nor retroperitoneal lymph node mass. HbSag was not present. Laboratory findings were as follows: RBC was 252×10^4 with 7.0 g/dl hemoglobin, the white blood cell count (WBC) was 5400. Thrombocyte was 28×10^4 . The BUN and creatinine were normal. A liver function test included total

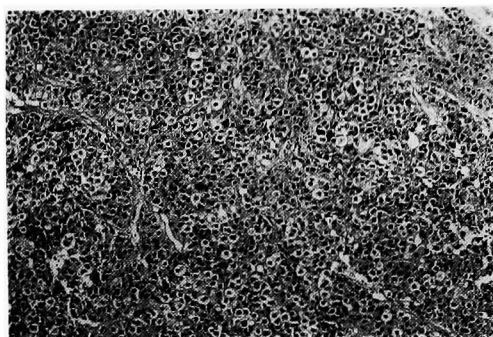


Fig. 1. Histological section of a prostate needle biopsy showed poorly differentiated adenocarcinoma. Hematoxylin & Eosin. $\times 100$.

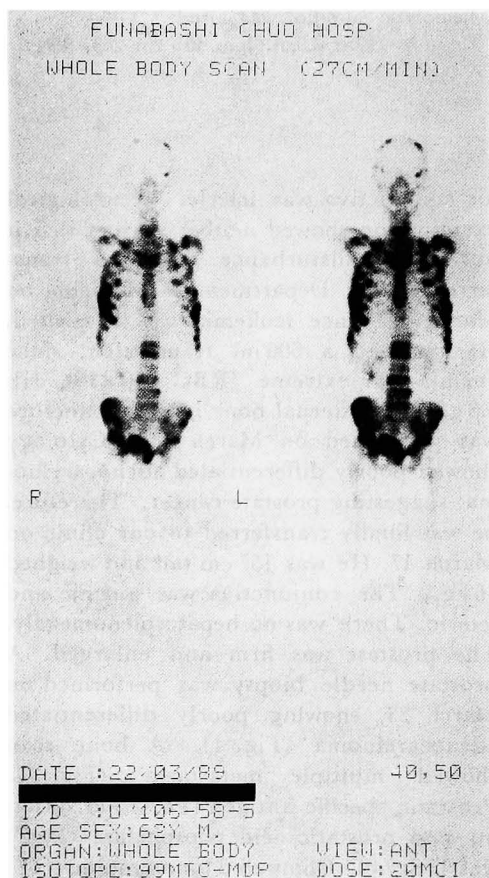


Fig. 2. Bone scan before treatment: Multiple bone metastases were observed (in skull, cervical spines, dorsal spines, lumbar spines, ribs, pelvic bones, and bilateral femur).

serum bilirubin 2.3 mg/dl (normal 0.2~0.8 mg/dl) with direct reacting level of 0.8 mg/dl (normal 0~0.2 mg/dl). Serum glutamic

oxaloacetic transaminase (GOT) and serum glutamic pyruvic transaminase (GPT) were normal. Alkaline phosphatase was 212 mIU (normal 23~75 mIU). Cholesterol was normal; urine was positive for urobilinogen and negative for bilirubin. On March 24, 1989, our patient was treated with intravenous DES-P (500 mg/day) for 40 days. Castration was not performed. From March 20 to April 11, blood transfusion was performed, resulting in a total of 2,000 ml. On the fourth day of treatment, jaundice began to decrease. On the 27th day, he was able to walk without assistance. Tumor marker (PSA and PAP) became normal on the 32nd day. The total serum bilirubin level became normal on the 47th day. A bone scan, three months later, showed marked improvement. DES-P was changed to estramustine phosphate (560 mg/day) and the patient was followed up. He was discharged on August 1. While following his ambulatory treatment, a relapse of prostate cancer was diagnosed on December 14, 1989. His condition became worse, and he died on May 25, 1990. Jaundice was not present from April 10, 1989 to the time of death.

Autopsy findings (courtesy of Dr. Haruo Ohkubo): The heart weighed 300 g. The right lung weighed 400 g and the left lung 400 g. There were pulmonary edema and congestion. The right pleural fluid was 750 ml and the left pleural fluid 550 ml. The liver weighed 1,200 g. The spleen weighed 140 g. The right kidney weighed 160 g, and the left kidney 170 g. There were many crystals in the renal tubules. The prostate was small and measured 3 cm $\times$ 3 cm. The lymph nodes in the porta hepatis were uninvolved. Histologically, the metastatic organs were lung, liver, left adrenal gland, bone (sternum, lumbar vertebra and ilium), and lymph nodes (Virchow, mediastinal and pelvis). In the liver section, hemosiderin particles were well stained, indicating that the patient was not icteric. The diagnosis was adenocarcinoma of the prostate with multiple metastases.

DISCUSSION

A liver function test showed that total

serum bilirubin was elevated (2.3 mg/dl). GOT and GPT were normal. Alkaline phosphatase was high (212 mIU). A urinalysis included positive urobilinogen and negative bilirubin. A CT scan demonstrated neither liver metastasis nor retroperitoneal lymph nodes. These findings are compatible with hemolytic jaundice.

Why did hemolytic jaundice occur in our case? Within one month, sudden anemia occurred (RBC 449×10^4 , Hb 12 g/dl, thrombocyte 22×10^4 on February 3, 1989; RBC 261×10^4 , Hb 7.1 g/dl, thrombocyte 4.9×10^4 on March 8). He was not icteric until March 7. During the one month, bleeding such as gastrointestinal and urinary was not noticed. The patient's history showed no exposure to toxic drugs. These findings suggested that sudden bone metastasis progressed simultaneously with anemia and that the balance between red cell production and destruction would be disturbed possibly followed by hemolysis.

Why was DES-P effective for hemolytic jaundice? The bone metastasis was mark-

edly improved with DES-P treatment. We thought that since the bone marrow was repaired by DES-P, red cell production would increase and then the balance of red cell production and destruction would be better maintained, and that the jaundice would therefore be cured.

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和文抄録

溶血性黄疸を呈した進行性前立腺癌の1例

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症例は62歳, 男性. 主訴は黄疸, 腰痛, 食思不良. 原因不明の貧血にて当院内科で, 胸骨骨髓穿刺施行. 細胞診にて低分化型腺癌の所見で, 前立腺癌が疑われ, 1989年3月17日当泌尿器科へ紹介される. 前立腺生検にて, 低分化型腺癌であった. 腫瘍マーカー (P-AP, PSA) は高値で, 骨スキャンにて多発性骨転移を認めた. Diethylstilbestrol diphosphate 投与によ

り, 黄疸は消失し, 全身状態も改善し一時退院となったが, 前立腺癌の全身再燃にて1990年5月20日死亡した. この黄疸のタイプは, 骨転移による急速な貧血により惹起された溶血性黄疸と診断した. 溶血性黄疸を伴う進行性前立腺癌は本例が第1例目である.

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