Aseptic Necrosis of Unilateral Scaphoid Bone in Systemic Lupus Erythematosus
Shunsei Hirohata and Koji Ito

An SLE patient developed aseptic necrosis of the right scaphoid bone 4 years after an episode of aseptic necrosis of bilateral femoral heads caused by corticosteroid treatment. Since the aseptic necrosis of the right scaphoid bone was preceded by the insidious exacerbation of SLE as evidenced by facial erythema, it was considered to be a result of vasculopathy due to active SLE. It took 14 months to make a correct diagnosis of the aseptic necrosis of the scaphoid bone by a chanced roentgenogram for the routine evaluation for osteoporosis. Therefore, the importance of an awareness of this possibility and repeated radiographic examinations is emphasized for the correct diagnosis of joint manifestations in SLE.

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Introduction
Aseptic bone necrosis has been reported to occur in 4–11% of patients with systemic lupus erythematosus (1). Most cases are associated with corticosteroid administration, whereas there are several cases which are considered to be induced by small vessel vasculitis or fat emboli (1). Multiple sites are commonly affected (1). Although the large joints are frequently involved, including the femoral head, tibial plateaus, and humeral heads (1); small joint involvement has only rarely been reported in the literature (2–7). Here, we describe an SLE patient who developed aseptic necrosis of the right scaphoid bone after 4 years from an episode of aseptic necrosis of bilateral femoral heads. The aseptic necrosis of the right scaphoid bone was followed by the exacerbation of SLE. Based on the findings, the pathogenesis of the 2 episodes of aseptic bone necrosis in this patient is discussed.

Case Report
A 38-year-old Japanese woman complained of swelling and tenderness of the right wrist for 1 month in January 1990. She had also suffered from Raynaud’s phenomenon since 1983 and from erythema in the face and extremities since August 1989. In June 1983, at the age of 33 years, she developed arthralgia, skin erythema, edema and proteinuria along with high spiking fever after an episode of common cold. She was admitted to our hospital for evaluation in August 1983. Laboratory examinations revealed leukocytopenia (2,000–4,000/mm³), thrombocytopenia (9.7 × 10⁴/mm³), positive anti-nuclear antibody (ANA) test (speckled pattern at a titer of 1:160), positive LE cell preparation, proteinuria (1–5 g/day) and decreased total hemolytic complement (CH50) of <10.0 U/ml (normal: 29.3–43.9 U/ml). Renal biopsy led to the diagnosis of membranous glomerulonephritis due to SLE. After the diagnosis the treatment with oral prednisolone of 50mg/day was started. Since CH50 was decreased after the reduction of the dose of oral prednisolone, intravenous methyl prednisolone pulse therapy (1 g/day for 3 consecutive days) was repeated 2 times in March 1984. After the pulse therapy, her disease activity remained well controlled with gradual reduction of the dose of oral prednisolone. She was discharged in July 1984 with oral prednisolone 25mg/day. The dose of prednisolone was carefully decreased after her discharge, and the disease activity continued to be well controlled.

In March 1986, when she was taking prednisolone 10 mg and 12.5 mg on alternate days, she complained of pain in the right hip joint. In January 1987, she also complained of pain in the left hip joint. At this time, the disease activity of SLE remained well controlled with oral prednisolone 7.5 mg and 10 mg for alternate days.
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Radiographic examination revealed the presence of aseptic necrosis of bilateral femoral heads. She denied habitual intake of alcohol. In addition, there was no evidence for hyperlipidemia or coagulopathy. Therefore, the aseptic necrosis of the bilateral femoral heads was considered to be caused by the corticosteroid treatment. After a year of conservative treatment, hemiarthroplasty of the left femoral head and the right femoral head was performed in May and July 1988, respectively.

From the time of the hemiarthroplasty, the disease activity of SLE had been well controlled with 5mg and 7.5mg prednisolone for alternate days until August 1989, when she developed facial erythema after exposure to sunlight. The patient continued to present erythema in the face and the extremities along with the elevation of serum rheumatoid factor (RF) (Fig. 1). She complained of pain in the right wrist joint in January 1990. She denied any chances of trauma. Physical examination at this time revealed malar erythema and telangiectasias, and moderate tenderness and swelling of the right wrist. All other joints were normal. Her grip strength was 140 mmHg. Laboratory examination disclosed WBC 5,500/mm3 with 10% of lymphocytes, hemoglobin 10.6g/dl, platelets 11.9 x 10^4/mm3, Westergren ESR 62 mm/h, and CH50 18.7 U/ml. The dose of prednisolone was increased to 30 mg/day. With this treatment, she recovered from the joint manifestations with disappearance of malar rash. Her grip strength was recovered bilaterally (>260 mmHg). The WBC count and platelet count increased (6,600/mm3 and 19.6 x 10^4/mm3, respectively), CH50 was also increased (24.1 IU/ml) and serum RF was decreased (24 IU/ml) (Fig. 1).

The roentgenogram of the wrists, taken for the routine evaluation for osteoporosis in March 1991 when she had no complaint of wrist pain, disclosed enlarged cystic rarefaction with sclerotic changes in the right scaphoid bone, indicating the presence of aseptic necrosis (Fig. 3). The bone scintigram in April 1991 confirmed the diagnosis of aseptic necrosis of the right scaphoid bone (Fig. 4).

**Discussion**

The present SLE patient developed aseptic necrosis of the right scaphoid bone after 4 years from the episode of aseptic necrosis of the bilateral femoral heads. When the patient developed aseptic necrosis of the bilateral femoral heads, the disease activity of SLE was well controlled. Since the patient had received high doses of corticosteroid, including 2 courses of intravenous methyl prednisolone pulse therapy, the first episode of aseptic necrosis of the bilateral femoral heads was considered to be caused by the corticosteroid treatment, as many investigators have reported (8, 9). In fact, the corticosteroid ingestion during the 1, 3 and 6 month periods of maximal corticosteroid therapy of the present patient (1,410mg, 3,775mg, and 11,570mg, respectively) was comparable to that of the “aseptic necrosis” patients in the study by Weiner and Abeles (9).

Aseptic necrosis in SLE patients most commonly involves large joints, including femoral heads, tibial plateaus, and humeral heads (1). Although multiple sites are most typically affected (1), small joint involvement has been reported in the literature (2–7). Only 8 patients have been described with aseptic necrosis of hand or wrist joints (2–7). The present patient lacked the involvement of tibial plateaus and humeral heads, although she developed aseptic necrosis of the bilateral femoral heads. Moreover, the aseptic necrosis of the right scaphoid bone in the patient occurred 4 years after the episode of femoral head involvement, during which period she was taking less than 10 mg/day of prednisolone. Therefore, it is unlikely that the same pathogenetic factors might result in the aseptic necrosis of bilateral femoral heads and in that of the right scaphoid bone in the present patient. On the other hand, the
The present patient had shown preceding malar erythema with the elevation of serum RF suggesting the insidious exacerbation of SLE, before she developed painful swelling of the right wrist. In addition, without the increases of corticosteroid the disease activity of SLE further exacerbated 6 months later, as evidenced by the development of generalized arthralgia, thrombocytopenia and hypocomplementemia. There was no evidence for hyperlipidemia, coagulopathy or excessive alcohol intake, which may lead to the development of aseptic necrosis. By contrast, the patient had suffered from Raynaud's phenomenon, which has been shown to be significantly correlated with aseptic necrosis presumably through vasculopathy (1, 6). Moreover, since malar erythema as well as the painful swelling of the right wrist joint subsided along with the decrease in RF and the increase in CH50 and platelet count after the increase of the dose of oral prednisolone, it is most likely that the aseptic necrosis of the right scaphoid bone in the present patient is a result of synovitis and vasculitis due to active SLE.

Urman et al described 3 SLE patients who developed aseptic necrosis in their carpal bones (7). Their patients presented with wrist pain and swelling, and an average of 11 months elapsed before aseptic necrosis was correctly diagnosed (7). In the present patient, it took 14 months to make correct diagnosis of aseptic necrosis of the scaphoid bone. If the roentgenogram had not been taken for the routine evaluation of osteoporosis, the aseptic necrosis of the scaphoid bone would have been overlooked for a longer period of the time. Therefore, repeated roentgenogram evaluation is recommended for any SLE patient who complains of wrist pain and swelling even after the improvement of the symptoms.

The present findings confirm the usefulness of bone scintigram for the evaluation of the aseptic necrosis in
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SLE patients (4). An awareness of this possibility and repeated radiographic examinations are important to facilitate an overall understanding of the pathogenesis of joint manifestations in SLE.

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References