—Report on Experiments and Clinical Cases—

Septic Arthritis of the Hip Associated with Atopic Dermatitis

A Case Report

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Abstract

We report a case of septic arthritis of the hip associated with atopic dermatitis. A 15-year female felt a pain in the right hip with unknown cause on May 11, 1998. The pain subsequently became aggravated, and she was admitted to our hospital on May 18. She has had atopic dermatitis since 4 years of age. She showed generalized dermatitis with desquamation and numerous scratch marks. A culture of both skin and joint fluid revealed Staphylococcus aureus. Physical examination revealed tenderness in Scarpa triangle and restricted range of motion. Immunological serology showed an increase in eosinophils and immunoglobulin E, and a decreased reaction of lymphocyte blastoid transformation. Computed tomography（CT）and MRI showed a joint effusion in the right hip. She was diagnosed as having septic arthritis of the hip. Intravenous drip of Cefazolin of 2 g was started on the first day of hospitalization and joint irrigation was done on the second day. CRP became negative at 4 weeks, but joint effusion was shown on CT. Additional joint irrigation with Amicamycin（200 mg）was done. As the joint fluid culture became negative, range of motion exercises were started at 6 weeks. She was discharged with a long-leg brace applied at 8 weeks. At 13 months after onset, she had complete relief of the pain and normal activities of daily living. No destructive changes in the hip were found on X-ray examination or MRI. In the present case, an abnormal immune system associated with atopic dermatitis as well as the habit of scratching eruptions may have led to hematogenous spread of skin infection, and caused septic arthritis of the hip.（J Nippon Med Sch 2000; 67: 464—467）

Key words: atopic dermatitis, arthritis, deep infection, staphylococcus aureus

Introductuion

Septic arthritis of the hip commonly occurs in infants or younger children, and often results in severe sequelae such as destructive changes in the femoral head or articular cartilage[1]. Occasionally, it occurs in older children or adults, especially in those with abnormal immune systems. Atopic dermatitis has been reported to show frequent skin infections due to abnormal immunity functions. Most patients show positive skin cultures for Staphylococcus aureus, but in rarecases have complications of deep infections such as septic arthritis or endocarditis. We report a case of septic arthritis of the hip associated with atopic dermatitis.

Case Report

A 15-year female felt a pain in the right hip with un-

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known cause, and visited a doctor on May 11, 1998. She was diagnosed as having non-septic arthritis of the hip, and was treated with nonsteroidal anti-inflammatory drugs. However the pain subsequently became aggravated, and she presented to our outpatient clinic on May 14. She has had atopic dermatitis since 4 years of age. There was past history of operations of otitis media at 2 and 6 years of age, but family history was unremarkable. On physical examinations, there was no swelling or warmth in the right hip, but tenderness was elicited over Scarpa triangle. Range of motion was markedly restricted due to pain (flexion of 30°, abduction of 15° and rotation of 5°). White blood cell count (WBC), erythrocyte sedimentation rate (ESR) and C reactive protein (CRP) were 11,700/mm^3, 68 mm/hr and 12.1 mg/dl, respectively, suggesting septic arthritis of the hip. X-ray examination showed no abnormal findings (**Fig. 1**).

She was admitted to our hospital on May 18. She had a fever of 38℃, and showed atopic dermatitis on the right hip and flexion side of the large joints with desquamation and numerous scratch marks (**Fig. 2**). A culture of skin for organisms revealed Staphylococcus aureus. Blood examinations showed a WBC of 11,300/mm^3, 7% eosinophils, and immunoglobulin E (IgE) of 4,400 U/ml (**Table 1**). Lymphocytes fraction test was normal, but lymphocyte blastoid transformation test showed low levels of phytohemagglutinin (PHA) and Concanavalin-A (Con-A). Computed tomography (CT) displayed a homogenous low-density area suggesting a joint effusion in the right hip (**Fig. 3 a**), and MRI demonstrated homogenous high-signal intensity in the corresponding area on T 2-weighted images (**Fig. 3 b**). From these results, she was diagnosed as having septic arthritis of the hip.

Intravenous drip of Cefazolin of 2 g was started on the first hospital day, and joint irrigation was performed on the second day. A culture of joint fluid revealed Staphylococcus aureus. Subsequently, CRP became negative at the 4th hospital week, but the joint effusion was shown on CT. Additional joint irrigation

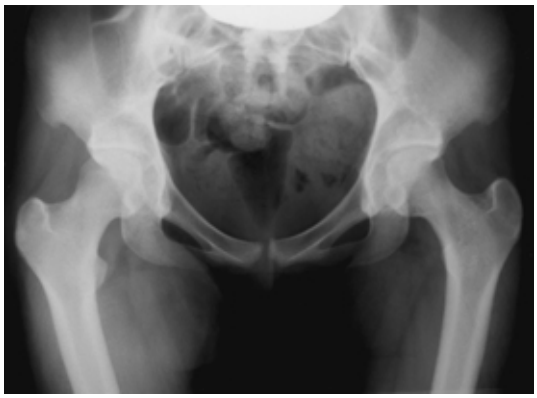

Fig. 1 X-ray findings at 3 days after onset. No abnormal findings were found in X ray examination.

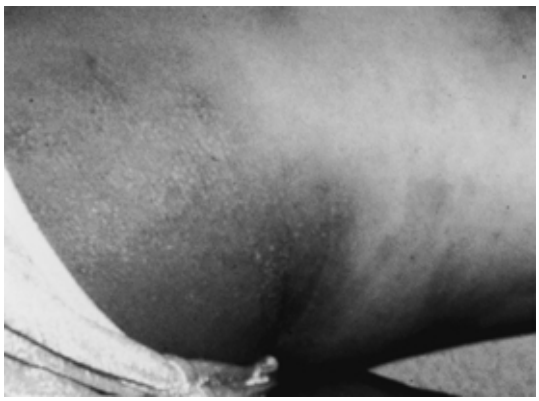

Fig. 2 Photograph of atopic dermatitis. An eruption with reddish discoloration and desquamation spread over the entire body.

Table 1 Blood examinations

Blood count		Immunological serology		
WBC	11,300 /mm^3	IgG	1,196 mg/dl	(800－1900)
RBC	398×10^4 /mm^3	IgM	230 mg/dl	(70－300)
Hb	11.4g/dl	IgA	214 mg/dl	(90－390)
Hemogram		IgD	2.7 mg/dl	(≤ 9.0)
Neutro.	72.0%	IgE	4,400 U/ml	(≤ 250)
Eosino.	7.0%			
Baso.	0.1%	PHA	11,464 CPM	(26,000－53,000)
Mono.	7.7%	Con-A	15,788 CPM	(20,000－48,000)
Lympho.	13.2%			

The figures in parenthesis show normal values.

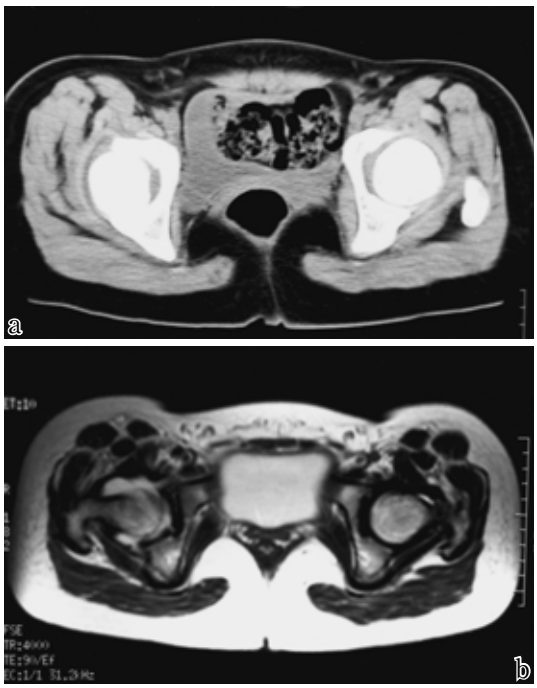

Fig. 3 CT and MRI at 8 days after onset.
a: Homogenous low density area suggestive of a joint effusion was identified in the right hip. b: T2-weighted images demonstrated homogenous high-signal intensity in the corresponding area.

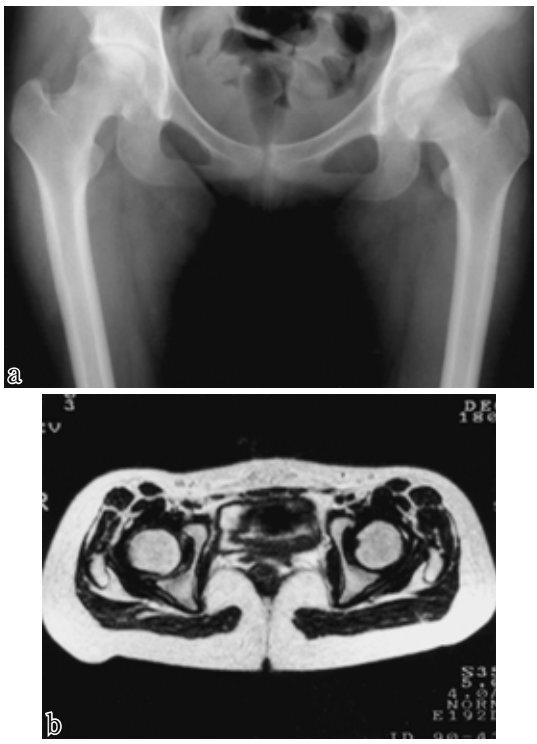

Fig. 4 X-ray and MRI at 13 months after onset.
a: No destructive change was found in the right hip.
b: No abnormal signal was visible in the right hip.

with Amicamycin（200 mg）was done. As bacteriologic cultures of the joint fluid became negative, range of motion exercises were started at 6 weeks. She was discharged with a long-leg brace applied at 8 weeks. The brace was removed 8 months after onset, and full weight-bearing gait was allowed. At 13 months after onset, she had complete relief of the pain and normal activities of daily living. Moreover no destructive changes in the hip joint were found in either X-ray or MRI（**Fig. 4 a, b**）.

Discussion

In our case, an abnormal immune system may have contributed to the occurrence of septic arthritis of the hip, because the patient was older than the common onset age of septic arthritis, and lacked previous infectious foci and abnormal immunological serology, including low levels of PHA or Con-A and high levels of serum IgE. Decreased reactions of lymphocyte blastoid transformation to non-specific mytogens such as PHA or Con-A are occasionally identified in patients with acute infection. In our case, low levels of PHA or Con-A were revealed on the 24th hospital day when CRP became negative with normal WBC count, suggesting the possible existence of chronic low levels of PHA or Con-A.

An increase in IgE is prominent in hyper-IgE syndrome, but is shown in some cases of atopic dermatitis. Atopic dermatitis shows skin lesions similar to those observed in hyper-IgE syndrome, and should be different from this syndrome[2]. This rare syndrome, which was first described by Buckley[3] in 1972, is characterized by the following clinical features: generalized skin lesions, distinctively coarse features, high incidence of skin abscess due to Staphylococcus aureus, prior infection with Candida, familial tendency for allergy and the detection of specific IgE antibody. In the present case, skin lesions were relatively limited to the hip and flexion side of large joints, the facies appeared as normal and no skin abscesses were observed. There was no past history of infection with Candida and no family history of allergy. As for the results of specific IgE antibody, the antibody against Acarus, which is negative in most patients with hyper-IgE syndrome, was positive. Conversely the an-

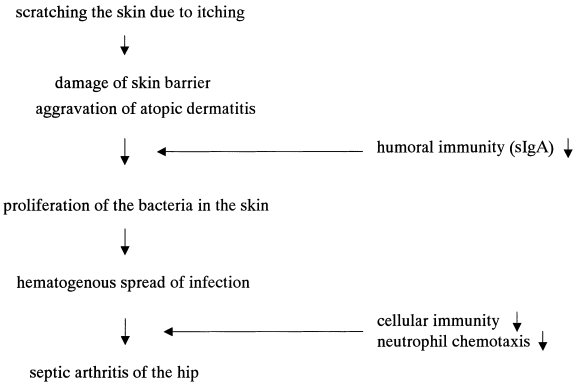

Fig. 5 The mechanism for onset of septic arthritis of the hip

tibody against Staphylococcus aureus, which is identified in all patients with hyper-IgE syndrome, was negative[4]. The possibility of hyper-IgE syndrome seems to be excluded by these results.

The main defense mechanisms of the body are neutrophil response, humoral immunity and cell-mediated immunity. As reasons for the susceptibility to infection in atopic dermatitis patients, it has been previously pointed out that neutrophil chemotaxis, which initially stops the spread of infection, is suppressed by histamine released from mast cells in inflammatory skin lesions[5,6].

As for humoral immunity, considerable attention has been focused on the importance of secretory component of immunoglobulin A (sIgA), which is discharged with sweat on to the body surface. Imayama[7] reported that the secretory amount of sIgA from the skin in atopic dermatitis patients is significantly lower than that in healthy subjects, and concluded that a decrease in sIgA might account for the high positive skin culture of Staphylococcus aureus observed in patients.

Regarding abnoramal cellular immunity, atopic dermatitis patients often show negative results in delayed hypersensitivity skin tests against Candida-antigen and Mumps virus-antigen. In the present case, lymphocytes obtained from the patient showed a decreased reaction of lymphocyte blastoid transformation to non-specific mytogen such as PHA or Con-A.

Deep infections associated with atopic dermatitis have been reported to occur not only in the hip joint[8], but also in the sacro-iliac joint[9] and the endocardium[10]. However, little is known about the onset mechanism of deep infections. Ourspeculation for the mechanism of septic arthritis of the hip is represented in **Fig. 5**. It is likely that atopic dermatitis patients customarily scratch the skin due to severe itching, resulting in aggravation of atopic dermatitis as well as in damage to the skin barrier which causes an invasion of Staphylococcus aureus. This process may lead not only to proliferation of the bacteria in the skin, but also to a skin infection entering the blood stream. Impairments of neutrophil chemotaxis and cellular immunity fail in destroying organisms, allow hematogenous spread of infection, and eventually result in septic arthritis of the hip.

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