Case Report

Sparing Effect of Hemiparesis on Palindromic Rheumatism

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Summary  
A male patient with palindromic rheumatism developed left hemiplegia. Since then, joint symptoms have not been observed in paretic limbs, although palindromic rheumatism has developed consistently in the nonparetic side. It could be speculated that neural rather than mechanical factors participate in the sparing of joint inflammation. This is the first report of the sparing effect of hemiparesis on palindromic rheumatism.

Key words  
Palindromic Rheumatism, Hemiparesis, Sparing Effect

INTRODUCTION

The sparing effects of hemiplegia on the development of various rheumatic diseases including rheumatoid arthritis (1,2,3), osteoarthritis (4,5), gout (6), polymyalgia rheumatica (7) and systemic sclerosis (8) have been reported. We report the first case of a patient with palindromic rheumatism who had not developed any articular symptoms in paralyzed extremities due to cerebral thrombosis.

CASE REPORT

A 61-year-old Japanese male patient developed painful swelling of the right wrist joint in 1958, at the age of 24. The arthritic inflammation reached its maximum within a few hours and subsided and disappeared almost within 4 days. Since then, the same attacks of painful joint swelling had repeated intermittently 3 to 4 times a month. The affected joints included bilateral wrists, elbows, shoulders, knees and ankles. Local heat and redness were sometimes found in the affected joints. In December 1991, he developed left hemiplegia due to cerebral thrombosis. A few days after the stroke, he noticed that joint pain in the left limbs did not develop, and since then, he has never experienced any pain and swelling in the joints of left hemiparetic side. In the nonparetic side, however, symptoms of palindromic rheumatism had recurred and the frequency of attack has not been reduced, although several nonsteroidal anti-inflammatory drugs and gold sodium had been administered. He was referred to our hospital in April 1994.

Physical examination revealed hemiparesis in the left upper and lower limbs. Swelling, tenderness and local heat were found in the right knee and wrist joints. No inflammatory signs were observed in the left joints of the paralyzed side. Laboratory data of urine, peripheral blood, coagulation, liver and kidney functions, lipids, electrolytes, serum protein fraction, immunoglobulins and complement hemolytic activity were within reference intervals. Rheumatoid factor and antinuclear antibodies were negative. Erythrocyte sedimentation rate was 30 mm/1h. Radiographic examination of joints did not show any abnormal findings seen in rheumatoid arthritis. Scintigraphy of bones examined during the attack (Sep. 27, 1995) showed increased uptake of radioisotope in the nonparetic right side joints including shoulder, elbow, wrist and foot as shown in Fig. 1.

Until now, he has had recurrent attacks of palindromic rheumatism in nonparetic right joints but no inflammatory symptoms in joints of paretic limbs.

DISCUSSION

In patients with hemi- or monoplegia, the development of rheumatic diseases has been observed in nonparalyzed limbs and the sparing effect of neurologic deficit on the development of rheumatic inflammations with
Sparing effect of hemiparesis on palindromic rheumatism

Palindromic rheumatism is characterized by intermittently recurring attacks of painful joints. In between attacks, neither joint pain nor other inflammatory signs are found in any joints. Since the description by Hench and Rosenberg (9), the mechanism and the cause of this peculiar joint inflammation have not yet been clarified. The patient reported here has had a 32-year history of palindromic rheumatism and the attack of arthritis has disappeared in the hemiparetic side after the cerebrovascular stroke. The sparing of joint inflammation in paralyzed limbs might be speculated to be due to some mechanical factors, neurovascular or neuromuscular factors and some unknown humoral factor. In this case, however, mechanical factors are not plausible because the sparing effect appeared soon after the stroke. A case reported by Pattrick et al. (10) and cases described by Hermann (11) give rise to the possibility that neurohumoral factor(s) rather than the mechanical factor(s) participate not only in the sparing but in the exacerbation of rheumatic inflammations. In the development of rheumatic inflammation in man, however, it appears to be difficult to clarify the mechanism of the sparing effect of neurologic deficit.

Recent investigations have disclosed the close relation of immunoneuro-endocrine network to the development of rheumatic inflammations. Neuropeptides such as substance P, neurokinin A and calcitonin-gene-related peptide were released from terminal C fibers within the joints and participate in the inflammatory processes (12). It seems plausible to speculate that the poor production of these neuropeptides due to the neurological deficit has brought to the improvement of arthritic inflammation in this case. It is the evident experiment of nature that the nervous system or neurohumoral factors participate unspecifically in the development of rheumatic inflammations.

Fig. 1: Bone scintigram of a patient with palindromic rheumatism taken during the attack.

REFERENCES