Psoriatic Arthritis Induced by Varicella

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There has been increasing evidence in recent years to implicate viruses as etiologic factors in the pathogenesis of arthritic disorders. Such entities accompanying infection with mumps, rubella and hepatitis B are well recognized, but a variety of other viruses such as rubella, smallpox and the arboviruses may also induce articular disease. The pattern of joint disease produced by viral infection varies. Often, both small and weight-bearing joints are affected symmetrically, as in rubella arthritis.

At the University of California Medical Center in San Francisco, we recently saw a patient with psoriasis and no previous arthritis who had psoriatic arthritis involving the distal interphalangeal (DIP) joints developed after a varicella infection. With resolution of the infection, the arthritis cleared completely and the psoriasis was considerably improved.

Report of a Case

A 22-year-old woman had a three-year history of psoriasis, which had flared severely four months before the present episode. Joint disease had not previously been noted. Two weeks before admission to hospital, typical lesions of varicella developed; the patient said she had not had chickenpox in childhood. A week before admission, she noted psoriatic lesions at the site of each vesicle. At the same time, she had throbbing pain in all the DIP joints, associated with intense redness and swelling.

Physical findings were confined to the skin, which bore hundreds of guttate plaques, and to the hands. The nails showed onycholysis and Beau lines consistent with a previous episode of severe psoriasis. All DIP joints of the hands were tender, swollen and erythematous. Findings on analysis of urine, hemogram and erythrocyte sedimentation rate, liver enzyme, uric acid, VDRL, antinuclear antibody and rheumatoid factor studies were all within normal limits. HLA (histocompatibility antigen) typing was not done, and x-ray films of the hands showed no bony abnormalities.

Indomethacin, administered in a dosage of 25 mg three times a day, provided symptomatic relief; after nine days, the psoriatic arthritis had cleared completely with resolution of the varicella.

Arthritis had not recurred after 11 months, when a follow-up examination was done.

Comment

Arthropathy is a frequent manifestation of many common viral infections. Arthritis accompanying varicella is rare; in the few cases reported, monoarticular, large joint involvement occurred coincident with the development of the skin lesions. In our patient, the anatomic location and temporal sequence differed. The arthritis occurred a week after viral infection, and articular involvement was confined to the DIP joints, which is typical of psoriatic arthritis.

In psoriasis, the joints may be affected in a variety of clinical patterns that include oligoarthritis, symmetric polyarthritis, ankylosing spondylitis, arthritis mutilans and classic psoriatic arthritis with predominant or exclusive involvement of the DIP joints.

The classic form of psoriatic arthritis developed in our patient. The DIP joints, which appear to be predisposed to arthritis in some psoriatic patients, were affected following varicella. Although the psoriatic arthritis and the varicella infection might have been independent events, the temporal relationship and rapid resolution of arthritis after clearing of varicella suggest that the virus was an
of championship performance who are found to have evidence of cardiac hypertrophy and enlargement, a slow resting heart rate, systolic ejection murmur and, occasionally, a variety of arrhythmias and conduction disturbances. This condition has been thought adaptive, benign and reversible, and restrictions on physical activity were not thought to be indicated. Nevertheless, occasional reports of sudden death in marathon runners with patent coronary arteries and the knowledge that only a history of athletic prowess deters a diagnosis of cardiomyopathy in a patient with unexplained cardiac enlargement, calls into question the presumed benign nature of this condition. Moreover, the adaptive response of a normal heart to isotonic and isometric exercise differs, and it is not known under what circumstances (if any) the response may become pathologic.

Just as a patient with the athletic heart syndrome is asymptomatic at diagnosis, so too may be persons from a kindred destined to manifest familial cardiomyopathy, who often have a subsequent malignant course characterized by congestive heart failure or sudden death. If these patients were initially capable of competitive physical exercise, it is possible that a spurious diagnosis of athletic heart syndrome would be made and the person encouraged to pursue limitless physical exertion, perhaps at some hazard.

In this paper we report the case of a young national champion tennis player who carried the diagnosis of athletic heart syndrome for six years following a complete cardiac evaluation in 1972. In this woman and several members of her family, all athletically active, concentric left ventricular hypertrophy was shown by echocardiography. The woman collapsed while playing tennis and died within 24 hours. On autopsy there was evidence of acute myocardial infarction, patent coronary arteries and diffuse cardiomyopathy.

The Athletic Heart Revisited

Sudden Death of a 28-Year-Old Athlete

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IN THE PAST organic heart disease was often the diagnosis when an asymptomatic, trained athlete presented with electrocardiographic evidence of left ventricular hypertrophy and a large heart shown on x-ray studies. In recent years, however, the diagnosis is more likely to be athletic heart syndrome. This increasingly popular diagnosis is usually applied to young athletes capable

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