Rheumatoid arthritis associated with Henoch-Schönlein purpura

Sirs,

Henoch-Schönlein purpura (HSP), also known as anaphylactoid purpura, is defined as a syndrome with cutaneous, gastrointestinal, and joint involvements, as well as nephropathy (1). It is generally recognized that HSP appears mainly in children, rarely in adults, and that HSP in childhood is mostly self-limited, but that in adults it can continue for longer periods of time (2, 3). Although this is still controversial, the prognosis of renal damage in adult HSP is poorer than that in children (4).

Rheumatoid arthritis (RA) is an autoimmune disease of unknown etiology. Multiple organs and sites including the joints, skin, eyes, lungs, pleura, nerves, and kidneys are involved. The articular manifestations in RA include morning stiffness, joint pain, swelling, deformity, and bone destruction. Unlike RA, the arthropathy found in HSP is non-deformed and the non-erosive inflammation occurs predominantly in the knees and ankles (2). It has been reported in the literature that the association of HSP with RA is rare (5, 6). We report here the case of an RA patient and her clinical course associated with HSP.

A 73-year-old Japanese woman started to have right knee joint pain in December 1993. Three months later arthralgia had extended bilaterally to the proximal interphalangeal and metacarpophalangeal joints in the fingers. She visited a local doctor and was treated with anti-rheumatic drugs (DMARDs), including bucillamine (200 mg/day), salazosulfapyridine (1 g/day) and methotrexate (2.5 mg/week) and non-steroidal anti-inflammatory drugs (NSAID) etodolac (400 mg/day) or flurbiprofen (120 mg/day) was begun. Gradually, the joint symptoms began to subside, with values of 32 mm/hr for ESR and 0.3 mg/dl for CRP, respectively, on October 16, 1997.

However, she suddenly noted purpura bilaterally on the lower legs on March 2, 1998. A skin biopsy showed leukocytoclastic vasculitis (Fig. 1) with IgA deposits on the vascular walls by immunofluorescence. She was treated with steroid therapy (prednisolone: 40 mg/day). The skin rash disappeared after one week.

Concomitantly, proteinuria (< 500 mg/day) and microhematuria were noted by urinalysis. A renal biopsy was performed on May 20, 1998. Renal histology showed mild mesangial proliferative glomerulonephritis with IgA and fibrinogen deposits. The steroid dosage was tapered until the proteinuria became negative in a spot urinalysis at the end of June 1998. Normal renal function with creatinine clearance was sustained during the whole clinical course and no relapse of purpura was observed as of August 1999.

The characteristic tetra-symptom complexes in HSP are purpura, abdominal pain, hematuria, and arthralgia. Articular manifestations such as joint pain and swelling are commonly (67%) associated in adult HSP, although they rarely last for more than a few days (2). The most common joints involved are the knees and ankles (2). Unlike RA, joint deformity and bone destruction do not occur in HSP.

References


Mitsuhashi et al. (5) reported 3 Japanese patients with both HSP and RA. In 2 of the 3 cases HSP preceded RA in the clinical course. The articular manifestations found in all 3 cases were not caused by HSP itself, because those patients showed X-ray findings of erosive lesions, which are typical of RA. Our patient had already been diagnosed as having RA with joint destruction when the HSP appeared. Cho et al. (6) also reported a patient who had RA prior to HSP with protein-losing enteropathy.

The precipitating factors in HSP include preceding streptococcal infection, food allergy, insect bites, drugs, and chemicals (1). Our patient was taking DMARDs and an NSAID when her HSP appeared. It is not conceivable that those drugs induced the HSP because: (1) HSP has not reappeared even though she is still taking the same drugs for RA that she had been taking before the appearance of HSP symptoms; and (2) there are no reported patients in whom the DMARDs used in this patient induced HSP. Mitsuhashi et al. (5) have described the possibility that the co-existence of RA and HSP might be dependent on the HLA type of DR3. However, this is not the case in our patient because her HLA typing was A2, A24; B55, B61; Cw1; DR9, DR12. It is reported that IgA class antineutrophil cytoplasmic antibodies (ANCA) are detected in HSP patients (7) and that positivity of ANCA in RA is associated with vasculitis (8). Thus, it is possible that ANCA in RA might drive HSP. However, IgA class ANCA for two ANCA antigens, i.e., myeloperoxidase and lactoferrin, were negative in this patient (data not shown). About one-third of RA patients have high serum IgA concentrations, although our patient had normal serum IgA concentration (230 mg/dl), as did 3 of 4 other reported patients with RA and HSP (5, 6). It has been reported that IgA immune complexes may cause leukoclastic vasculitis in HSP (9), and that IgA nephropathy is often associated with Japanese RA patients (10). Taken together, it might be speculated that a common underlying IgA dysfunction brought on the association of RA and HSP in this patient.

Osteogenesis imperfecta

Sirs,

Osteogenesis Imperfecta (OI) is a genetic, connective tissue and skeletal disorder, which is characterized by bone fragility, osteopenia, a variable degree of short stature and progressive skeletal deformities (1-3). The disorder clinically manifests in a broad spectrum ranging from mild to severe forms. Additional clinical presentations include blue sclera, joint laxity and maturity onset deafness. OI appears in approximately 1 in 20,000 births and is caused by different quantitative and qualitative defects in the synthesis of colla-

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References

Osteogenesis imperfecta-inducible migratory stress fractures in a military recruit

Sirs,

Osteogenesis Imperfecta (OI) is a genetic, connective tissue and skeletal disorder, which is characterized by bone fragility, osteopenia, a variable degree of short stature and progressive skeletal deformities (1-3). The disorder clinically manifests in a broad spectrum ranging from mild to severe forms. Additional clinical presentations include blue sclera, joint laxity and maturity onset deafness. OI occurs in approximately 1 in 20,000 births and is caused by different quantitative and qualitative defects in the synthesis of collagen I. More than 150 mutations of the COL1A1 and COL1A2 genes, which encode for type I procollagen, have been identified so far (2).

A 19-year-old, previously healthy military recruit presented to our orthopedic clinic with pain in his left foot which appeared after minor physical stress (mainly walking for few days). Based on the physical examination, bone radiographs and a whole body bone scan, a diagnosis of stress fracture of the third metatarsal bone was made. Three weeks later he started to complain of pain in his right thigh, and a stress fracture of the femoral neck was noted. At this stage a systemic illness was searched for, and eventually OI disease was found. His form of the disease was very mild and had not shown any clinical symptoms until that time.

Lately, an association between transient osteoporosis and OI has been reported (4-6), and the authors considered that a stress fracture was the triggering factor. Hence, it is suggested that in certain groups which have a higher tendency to bone fractures such as gymnasts, military recruits, etc., collagen disease should be looked for prior to their undergoing vigorous training. This is especially important in the recent years due to the development of better genetic studies (2). It is also recommended to perform cost-benefit analyses of such programs.

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References