Chronic Psoriasis May Lead to Diabetes, CVD

BY BETSY BATES
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PARIS — Chronic cutaneous inflammation associated with psoriasis may trigger a cascade of metabolic events leading to serious systemic diseases, including diabetes and coronary heart disease, Enno Christophers, M.D., asserted at the European Congress on Psoriasis 2004.

A complex interaction of genetics, environmental factors, and age contributes to development of psoriasis and psoriatic flares, said Dr. Christophers, professor of dermatology at the University of Kiel (Germany).

Similarly, multifactorial causes are responsible for diseases found more commonly in people with psoriasis than those with other skin conditions or other adults. Obesity and alcoholism, both elevated in patients with psoriasis, may be environmental contributors.

But in some cases, the inflammatory connection is clear. For example, a recent study found that 30% of patients with psoriasis have evidence of arthritis.

"This is an extremely high figure that we need to memorize," Dr. Christophers said.

Crohn's disease is seen seven times more frequently in psoriasis patients than in patients with other skin diseases.

Evidence is building that chronic inflammation may also lead to development of insulin resistance and Syndrome X, characterized by hypertension, diabetes, adiposity, dyslipidemia, and coronary heart disease.

Patients with psoriasis suffer disproportionately from these conditions. Recent studies have shown that patients with severe psoriasis have a significantly elevated mortality rate from cardiovascular diseases.

In recently completed research, Dr. Christophers and associates at the University of Kiel found that hospitalized psoriasis patients have almost double the body mass index of other skin disease patients or of community controls.

They have sharply increased rates of hypertension and higher rates of diabetes in patients who are in their 40s (comparative *P* less than .001) and those in their 70s (*P* less than .001, with a prevalence of about 25%, compared with about 5% in patients with other skin diseases).

Longstanding systemic inflammation has an impact on endothelial function and lipids, both of which contribute to serious disease, Dr. Christophers stressed. Longstanding cutaneous inflammation may well have the same consequences.

The T-helper 1 response associated with psoriasis activates autoreactive T-cells, and may be compounded by environmental factors and diseases (streptococcal tonsillitis).

Therapies that activate a T-helper 2 response, such as biologics and fumaric acid esters, may dampen inflammation over the long term and protect patients from serious diseases, such as diabetes and heart disease. ■

S. aureus May Contribute to Psoriasis Severity

PARIS — Patients colonized with certain enterotoxic strains of *Staphylococcus aureus* had significantly worse Psoriasis Area and Severity Index scores than did patients not colonized with these bacterial strains, raising the possibility that antibiotics might have an adjunctive role in treatment, Austrian dermatologists reported at the European Congress on Psoriasis 2004.

Nordwig S. Tomi, M.D., and Elisabeth Aberer, M.D., of Karl Franzens University in Graz, Austria, took sample swabs from the lesional skin and nares of 25 patients with psoriasis for evidence of *S. aureus* colonization and identification of enterotoxins A, B, C, or D.

Samples from 15 of 25 patients grew positive cultures; these samples were from the nares alone in 1 patient, skin only in 4 patients, and skin and nares in 10. Sixty percent of the strains produced *S. aureus* enterotoxins.

Four patients had enterotoxin B, two had enterotoxin C, one had D, and combinations of A plus D and B plus C were found in one patient each. The Psoriasis Area and Severity Index score was significantly higher ($P = .001$) in patients with enterotoxin-producing staphylococcal strains, the investigators reported in a poster presentation at the meeting.

—Betsy Bates