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Clinical Features, Etiologic Factors, Associated Disorders, and Treatment of Palmoplantar Pustulosis: The Mayo Clinic Experience, 1996-2013

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Abstract

Objective: To further characterize clinical characteristics, etiologic factors, associated disorders, and treatment of palmoplantar pustulosis (PPP).

Patients and Methods: We conducted a retrospective review of patients with PPP at Mayo Clinic between January 1, 1996, and December 31, 2013.

Results: Of 215 patients with PPP identified, 179 (83%) were female, and the mean age at onset was 45.3 years. Most patients (n=165, 77%) were current or former smokers. At diagnosis, 15 patients (7%) had an anxiety diagnosis and 9 (4%) had an infection. Nineteen cases (9%) were drug induced. Comorbid conditions included thyroid disease in 18 patients (8%), gluten sensitivity in 3 (1%), and type 2 diabetes mellitus in 21 (10%). In all, 194 patients (90%) received topical corticosteroids, 55 (26%) received phototherapy, and 54 (25%) received systemic agents.

Conclusion: More than three-fourths of the patients in this study had a history of smoking, which is considered a triggering or aggravating factor for PPP. Regarding comorbid conditions, gluten sensitivity and thyroid disease were found less frequently than previously reported in the literature. Treatment regimens and responses in this cohort varied considerably.

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Palmoplantar pustulosis (PPP) is a chronic, relapsing, pustular eruption localized to the palms and soles.[1,2] Brown macules representing resolving pustules, erythema, scale, and fissures are additional frequent findings.[1] Although skin involvement in PPP is limited, the condition may be painful and disabling. In severe cases, discomfort associated with skin involvement hinders walking or use of the hands. The cause is unknown, and it is unclear whether it is a distinct entity or a localized pustular variant of psoriasis.[3,4] Clinical observations suggest that smoking,[1] emotional stress (eg, anxiety),[5] focal infections (eg, acute or chronic tonsillitis, dental infection, chronic sinusitis),[6] and certain drugs (eg, tumor necrosis factor [TNF]-α inhibitors)[7] contribute to the development or exacerbation of PPP. Furthermore, PPP has been linked to systemic abnormalities such as SAPHO (synovitis, acne, pustulosis, hyperostosis, and osteitis) syndrome,[8] thyroid disease,[9,10] gluten sensitivity,[11] and type 2 diabetes mellitus (DM).[12]

Many treatments have been suggested for PPP, but none have been generally accepted as being reliably effective. Topical corticosteroids used under occlusion, oral psoralen—UV-A, and acitretin have shown benefit in treating PPP.[13] The aim of this study was to retrospectively examine the features of PPP—including clinical characteristics, etiologic factors, associated disorders, and responses to treatment—in patients at Mayo Clinic in Rochester, Minnesota.

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PATIENTS AND METHODS

Data Collection

We retrospectively searched the institutional medical index and text retrieval system for the records of patients with a diagnosis of PPP who were treated at Mayo Clinic between January 1, 1996, and December 31, 2013. The

study received approval from the Mayo Clinic Institutional Review Board. Patients who denied research authorization were excluded from this study. The following information was obtained from the medical records: patient characteristics, clinical characteristics, disease duration, identifiable causes of PPP, comorbid conditions, laboratory test results, histopathologic data, patch test data, response to therapies, and follow-up information since diagnosis.

Definition of PPP

Per the methods of Brunasso et al,[1] PPP was defined as "recurrent, discrete, 1- to 10-mm sterile pustules that often coalesce and resolve with brownish discoloration, frequently associated with well-demarcated erythema, hyperkeratosis, and desquamation, located symmetrically or asymmetrically on the palms and/or soles in the absence of pustules involving other areas of the body, and/or erythroderma."[p1244] Also using the same criteria as Brunasso et al,[1] we excluded patients with more than 5% body surface area involvement outside of the palmoplantar region.

Etiologic Factors

Smoking. Patients were classified as having smoking-induced PPP if they were current or former smokers at disease onset. Following the definitions of the Centers for Disease Control and Prevention regarding cigarette smoking, we defined current smokers as those who reported having smoked at least 100 cigarettes in their lifetime and who, at the time of disease onset, smoked either every day or some days.[14] Former smokers were defined as those who reported having smoked at least 100 cigarettes in their lifetime and who, at the time of disease onset, did not smoke at all. Never smokers were defined as those who reported never having smoked 100 cigarettes.

Stress. Patients were classified as having stress-induced PPP if they experienced symptoms of anxiety in association with their skin eruption(s). Comparable with a previous study, patients needed to have anxiety diagnosed by a psychologist or psychiatrist using appropriate psychological tests.[5]

Focal Infection. Patients were classified as having infection-induced PPP if they had evidence of tonsillitis, dental infection, or chronic sinusitis associated with their skin eruption. As in a previous study,[6] patients needed to have a focal infection diagnosed through examinations such as a tonsil test, radiologic test, and blood test.

TNF-α Inhibitor. Patients were classified as having TNF-α inhibitor–induced PPP if they were receiving TNF-α inhibitor therapy at the time of disease onset.

Associated Disorders

SAPHO Syndrome. Patients were classified as having associated SAPHO syndrome if they had a personal history of SAPHO syndrome or clinical characteristics suggestive of SAPHO syndrome (ie, synovitis, acne, pustulosis, hyperostosis, or osteitis).[8]

Thyroid Disease. Patients were classified as having associated thyroid disease if they had a personal history of thyroid disease or blood test results suggestive of thyroid disease (eg, abnormal thyrotropin, abnormal thyroxine, or increased antimicrosomal or antithyroglobulin antibody levels).[9,10]

Gluten Sensitivity. Patients were classified as having associated gluten sensitivity if they had a personal history of gluten sensitivity or blood test results suggestive of gluten sensitivity (eg, increased antigliadin or tissue transglutaminase antibody levels).[11]

Type 2 DM. Patients were classified as having associated type 2 DM if they had a personal history of type 2 DM or blood test results suggestive of type 2 DM (eg, increased fasting plasma glucose or glycated hemoglobin A_{1c} levels).[12]

Response to Treatment

Patient response to therapy was assessed and classified as complete response, partial response, or no response. Resolution of old lesions with a lack of any new lesions indicated complete response. Healing of some lesions with reduced severity and frequency of flares indicated partial response. Persistence of