

## ENTITLEMENT ELIGIBILITY GUIDELINES

### PSORIASIS

**MPC** 01210  
**ICD-9** 696.1

#### DEFINITION

Psoriasis is a chronic inflammatory skin disease involving accelerated proliferation of the epidermis layer of the skin. It generally consists of erythematous, well-demarcated papules and rounded plaques, covered by silver-colored scales.

#### DIAGNOSTIC STANDARD

Diagnosis from a qualified medical practitioner is required. The diagnosis can be made clinically; no specialized tests are required.

#### ANATOMY AND PHYSIOLOGY

The characteristic anatomy of psoriasis is hyperproliferation of the epidermal layer of skin. Epidermal cell turnover at sites affected by psoriasis is approximately 10 times the cell turnover of normal epidermal cells. All types of psoriasis have similar histological features. Psoriasis develops into characteristic plaques at some time during the disease.

There are several common variants of psoriasis:

- guttate psoriasis - where numerous small papular lesions with silvery scaling evolve suddenly over the body, often 1 to 3 weeks after streptococcal pharyngitis;
- inverse psoriasis - where plaques evolve in intertriginous areas; these plaques lack the typical silver scale because of maceration and moisture;
- pustular psoriasis - where superficial pustules may stud typical plaques, be confined to the palms and soles, or be associated with a rare generalized erythematous skin condition accompanied by fever and leukocytosis;
- erythrodermic psoriasis - where there is generalized erythema and scale covering the entire body;
- psoriasiform - where there are lesions resembling psoriasis.

## CLINICAL FEATURES

Psoriasis is a common skin disease which affects approximately 1% of the general population. It may begin at any age, although the peak age of onset is between ages 15 and 25. Persons with onset at an early age tend to have more severe disease. Those with onset before the age of 40 are likely to be HLA-Cw6 positive; however, not all people with HLA-Cw6 develop psoriasis, and psoriasis occurs in other HLA types. Genetic factors play an important role. About 44% of persons with psoriasis have an affected parent. Racial factors play a more important role than geographical location in the prevalence of psoriasis.

The lesions of psoriasis are distinctive. Psoriatic lesions begin as red, scaling papules that coalesce to form round-to-oval plaques, which can easily be demarcated from the surrounding normal skin. The scale is adherent, silvery white, and reveals bleeding points when removed (Auspitz's sign). Trauma to skin may induce new skin lesions (Koebner's phenomenon). Scaling may become extremely dense, especially on the scalp. Scaling forms, but is macerated and dispersed in intertriginous areas; therefore, the psoriatic plaques of skin folds appear only as smooth, red plaques with a macerated surface. The most common site for an intertriginous plaque is the intergluteal fold.

Nails often show small pitting depressions on the surface of the nail bed, and a characteristic red-brown discoloration of the surface that resembles an oil stain under the nail. Subungual collections of keratotic material are common, with distal separation of the nail from the nail bed. Mucous membranes are rarely involved. The extensor surfaces of the arms and legs, and the scalp are the most commonly affected sites. Skin trauma increases the risk for involvement, but any epidermal surface can become involved.

The disease may remain localized to a few areas, or may cause continuous generalized disease, occasionally resulting in total body erythema and scale, i.e. erythrodermic psoriasis.

Clinical course is characterized by chronicity and seasonal fluctuations, with a worsening in winter as dry skin leads to epidermal injury, and improvement in the summer secondary to sun exposure.

There is no curative agent for psoriasis, and treatment suppresses the condition only as long as it is administered. Sunlight has been reported as beneficial and is a recommended treatment in many cases.

## PENSION CONSIDERATIONS

### A. CAUSES AND/OR AGGRAVATION

**THE TIMELINES CITED BELOW ARE NOT BINDING. EACH CASE SHOULD BE ADJUDICATED ON THE EVIDENCE PROVIDED AND ITS OWN MERITS.**

1. Idiopathic

The precise causes of the abnormal cellular proliferation and inflammation are unknown.

2. Genetic

There is an increased prevalence in individuals with human leukocyte antigens HLA-B17, B13, and BW37 and Cw6. However, not all these individuals will develop psoriasis; thus, HLA type alone does not fully explain the etiology involved in psoriasis. It appears to be a multifactorial disease in persons who are genetically predisposed.

3. Skin trauma or other interruption to skin

Skin trauma or other interruption to skin may cause the initial manifestation of psoriasis, contribute to psoriasis, and/or aggravate existing psoriasis.

Skin trauma or other interruption to skin must interrupt the integrity of both the epidermal and dermal layers of the skin.

“Skin trauma” may result from a wound, cut, laceration, gunshot wound, or abrasion of the skin.

“Other interruption to skin” may result from disorders which produce cutaneous vesicles, including drug reactions and dermatitis, and from external forces which damage the skin, including chemical, thermal (e.g. frostbite), and electrical burns.

Skin trauma or other interruption to skin must be significant enough to result in the *Koebner phenomenon*, i.e. the development of isomorphic psoriatic lesions. The lesions must occur at the site of the trauma or other interruption to skin. The lesions should develop within weeks of the trauma or other interruption to skin.

#### 4. Medication

Certain medications may cause or aggravate psoriasis.

Treatment should be ongoing at time of clinical onset or aggravation, although drug-induced aggravation may occur up to several months after the medication is first taken.

As drug-induced psoriasis may continue, improve or disappear on discontinuation of the drug, evidence of a chronic condition is required.

Drugs may also produce temporary flare-ups or exacerbations. These temporary flare-ups or exacerbations must lead to permanent aggravation of psoriasis to be considered pensionable.

Drug-induced aggravation can be unpredictable and severe.

- Lithium

Lithium is a known cause of psoriasis in susceptible individuals, and can aggravate existing psoriasis. Pre-existing psoriasis is not, however, a general contraindication to lithium treatment, and the disease does not permanently worsen in many lithium-treated persons.

- Beta-blocker: oral or parental (by injection)

Beta-blockers have been recognized to cause psoriasiform, i.e. a psoriasis-like eruption, and to aggravate true psoriasis. These drugs may be used to treat hypertension, certain heart conditions, thyrotoxicosis, and as a preventative for migraines.

- Anti-malarial, e.g. hydroxychloroquine and chloroquine

Anti-malarials may aggravate psoriasis, although pre-existing psoriasis is not contra-indicated in persons who have psoriasis and need prophylactic treatment for malaria. Chloroquine is also used to treat other conditions, including Rheumatoid Arthritis and certain dermatological disorders.

- Systemic and topical steroids

These drugs may aggravate psoriasis. However, it is important to note that they may also cause flare-ups which may or may not represent permanent worsening. It is recognized that withdrawal

---

after prolonged use of steroids can frequently result in a severe flare-up of psoriasis, notably pustular psoriasis.

5. Anxiety and depressive disorders for aggravation only

Anxiety and depressive disorders may aggravate psoriasis. While the role of stress is felt to be acute in most cases and is not considered to lead to permanent worsening, clinical studies support the fact that anxiety or depressive disorders, when chronic or severe, may aggravate psoriasis in some individuals.

*For an anxiety or depressive disorder to aggravate psoriasis, the following should be evident:*

The anxiety or depressive disorder must attract a diagnosis under DSM-IV; *and*

Medical intervention must have been sought for the anxiety or depressive disorder; *and*

Increased signs/symptoms of psoriasis should be ongoing or recurrent for at least 6 months.

It is recommended that each case be examined on its individual merits, taking into account the chronicity and severity of the anxiety or depressive disorder.

6. Respiratory streptococcal infections in Guttate Psoriasis only

The medical literature supports an association between acute respiratory streptococcal infections and the development and aggravation of guttate psoriasis.

Streptococcal lesions in other parts of the body are excluded.

No specific strain of the streptococcal bacteria has been identified.

The guttate variant of psoriasis usually develops or is aggravated one to three weeks after the respiratory streptococcal infection.

Because guttate psoriasis may resolve spontaneously within weeks or months, it is necessary to ensure that it is chronic, i.e. in existence for more than 6 months, before a relationship can be recognized for pension purposes.

7. HIV infections for aggravation only

Psoriasis may become particularly severe in individuals who are HIV infected. It may become guttate in appearance and more refractory to treatment.

8. Inability to obtain appropriate clinical management

**Exclusions:**

Despite research efforts to date, there is a lack of sufficient evidence at this time to establish, for pension purposes, causation and /or aggravation between the following and psoriasis:

- staphylococcal infections
- yeast infections
- viruses
- cigarette smoking\*

\*While there is some medical research linking cigarette smoking to "*palmoplantar pustulosis*", this is regarded by many to be an entity separate from psoriasis.

**B. MEDICAL CONDITIONS WHICH ARE TO BE INCLUDED IN ENTITLEMENT/ASSESSMENT**

- Secondary skin infection

**C. COMMON MEDICAL CONDITIONS WHICH MAY RESULT IN WHOLE OR IN PART FROM PSORIASIS AND/OR ITS TREATMENT**

- psoriatic arthritis

Photochemotherapy with oral methoxsalen (psoralen) and ultraviolet A radiation (PUVA) can increase the risk in the following conditions, therefore, medical consultation(s) should be requested:

- basal cell carcinoma
- malignant melanoma
- squamous cell carcinoma (skin)
- cutaneous genital tumours in men
- cataracts

## REFERENCES FOR PSORIASIS

1. Australia. Department of Veterans Affairs: medical research in relation to the Statement of Principles concerning Psoriasis, which cites the following as references:
  - 1) Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Fauci AS, and Kasper DL (Eds) (1994) *Harrison's Principles of Internal Medicine*. McGraw-Hill, New York, 13th Ed. p276.
  - 2) Christophers E and Kruiger GG (1987) Epidermis: disorders of cell kinetics and differentiation in *Dermatology in General Medicine*. Fitzpatrick TB, Eisen AZ, Wolff K, Freedberg IM and Austen KF (Eds.) New York: McGraw-Hill Book Company. p. 478.
  - 3) Baker H (1986) Psoriasis in *Textbook of Dermatology. Vol. 2*. Rook A, Wilkinson DS, Ebling FJG, Champion RH and Burton JL (Eds.) Fourth Edition. Oxford: Blackwell Scientific Publications. p. 1472.
  - 4) Higgins EM and du Vivier AWP (1994) Cutaneous Disease and Alcohol Misuse. *Br Med Bulletin*. Vol. 50(1) pp. 85-98.
  - 5) Krueger GG and Duvic M (1994) Epidemiology of Psoriasis : Clinical Issues. *J Invest Dermat*. Vol. 102. pp.14S-18S.
  - 6) Farber EM and Nall L (1994) Psoriasis in the Tropics. *Dermatological Clinics*. Vol. 12(4). pp. 805-616.
  - 7) Elder JT, Henseler T, Christophers E, Voorhees JJ and Nair RP (1994) Of genes and antigens: the inheritance of psoriasis. *J Invest Dermat*. Vol. 103. pp.150S-153S.
  - 8) Theeuwes M and Morhenn V (1995) Allelic Instability in the Mitosis Model and the Inheritance of Psoriasis. *J Am Academy Derm*. Vol. 32. pp. 44-52.
  - 9) Krueger, G.G. and Duvic, M. (1994) *op cit*. pp. 14S-18S.
  - 10) Brandrup F, Holm N, Grunnet N, Henningsen K and Hansen HE (1982) Psoriasis in monozygotic twins: variations in expression in individuals with identical genetic constitution. *Acta Dermatovener (Stockholm)*. Vol. 62. pp. 229-236.
  - 11) Elder JT, Nair RP and Voorhees JJ (1994) Epidemiology and the genetics of psoriasis. *J Invest Dermat*. Vol. 102. pp. 24S-27S.
  - 12) Naldi L, Parazzini F, Brevi A, Peserico A, Veller Fornasa C, Grosso G, Rossi E, Marinaro P, Polenghi MM, Finzi A, Galbiati G, Recchia G, Cristofolini M, Schena D and Cainelli T (1992) Family history, smoking habits, alcohol consumption and risk of psoriasis. *Br J Derm*. Vol. 127. pp. 212-217.
  - 13) Arnett FC, Reveille JD and Duvic M (1991) Psoriasis and psoriatic arthritis associated with HIV infection. *Rheumatic Clinics of North America*. Vol. 17(1). pp. 59-78.

- 14) White SH, Newcomer VD, Mickey MR and Terasaki PI (1972) Disturbance of HLA Antigen Frequency in Psoriasis. *NEJM*. Vol. 287(15) pp. 740-743.
- 15) Barker JNWN, Mitra RS, Griffiths CEM, Dixit VM and Nickoloff BJ (1991) Keratinocytes as Initiators of Inflammation. *The Lancet*. Vol. 337, Jan. 26. pp. 211-214.
- 16) Ros AM (1992) Photosensitive Psoriasis. *Seminars in Derm*. Vol. 11(4). pp. 267-268.
- 17) O'Doherty CJ and MacIntyre C. (1985) Palmoplantar pustulosis and smoking. *BMJ*. Vol.291. pp. 861-864.
- 18) Mills CM, Srivastava ED, Harvey IM, Swift GL, Newcombe RG, Holt PJA and Rhodes J (1992) Smoking habits in psoriasis: a case control study. *Br J Dermatology*. Vol. 127. pp. 18-21.
- 19) Poikolainen K *et al.* (1994) Smoking, alcohol and life events related to psoriasis. *Br J Derm*. Vol. 130. pp. 473-477.
- 20) Poikolainen K, Reunala T, Karvonen J, Lauharanta J and Kärkkäinen P (1990) Alcohol intake: a risk factor for psoriasis in young and middle aged men? *BMJ*. Vol. 300. pp. 780-783.
- 21) Farber EM and Nall L (1993) Erythrodermic Psoriasis. *Cutis*. Vol. 51. pp. 79-82.
- 22) O'Doherty CJ and MacIntyre C (1985) *op cit.* p. 863.
- 23) Chaput JC, Poynard T, Naveau S, Penso D, Durrmeyer O and Suplisson D (1985) Psoriasis, alcohol and liver disease. *BMJ*. Vol. 291. p. 25.
- 24) MacGregor RR (1986) Alcohol and Immune Defense. *JAMA*. Vol. 256(11). pp. 1474-1479.
- 25) Fitzpatrick TB *et al.* (Ed.) (1987) *Dermatology in General Medicine*. New York: McGraw-Hill Book Company. p. 478.
- 26) Menter A. and Barker JNWN. (1991) Psoriasis in Practice. *The Lancet*. Vol. 338. July 27. pp. 231-234.
- 27) Duvic M, Rios A and Brewton GW (1987) Remission of aids-associated psoriasis with zidovudine. *The Lancet*. Sept. 12, p. 627.
- 28) Belsito DV, Sanchez MR, Baer RL, Valentine F and Thorbecke GJ (1984) Reduced Langerhans` cell IA antigen and ATPase activity in patients with AIDS. *NEJM*. Vol. 310(20). pp. 1279-1282.
- 29) Forster SM, Seifert MH, Keat AC, Rowe IF, Thomas BJ, Taylor-Robinson D, Pinching AJ and Harris JRW (1988) Inflammatory joint disease and HIV infection. *BMJ*. Vol. 296. pp. 1625-1627.
- 30) Ellis, C.N. Gorsulowsky DC, Hamilton TA, Bilings JK Brown MD, Headington JT, Cooper KD, Baadsgaard O, Duell EA, Annesley TM Turcotte JG and Voorhees JJ (1986) Cyclosporin Improves Psoriasis in a Double-blind Study. *JAMA*. Vol. 256(22). pp. 3110-3116.
- 31) Ramsay B and O`Reagan M (1988) A Survey of the Social and Psychological Effects of Psoriasis. *Br J Derm*. Vol. 118. pp. 195-201.

- 
- 32) Menter A. and Barker JNWN. (1991) Psoriasis in Practice. *The Lancet*. Vol. 338. July 27. pp. 231-234
  - 33) Peserico A, Zanetti G, Padovan S, Bertoli P, Veller Fornasa C, Cipriani R, Ambrosio GB, Zamboni S and Pagnan A (1988) Relationship Between Body Weight and Blood Pressure and Some Metabolic Parameters in Psoriatic Patients. *Br J Derm*. Vol. 118. pp. 191-194.
2. Bork, Konrad, and Brauninger, W. *Diagnosis and Treatment of Common Skin Diseases*. Toronto: W. B. Saunders, 1988.
  3. Cotran, Ranzi. S., Vinay Kumar, et al. *Robbins Pathologic Basis of Disease*. 6<sup>th</sup> ed. Toronto: W. B. Saunders, 1999.
  4. Fauci, Anthony S. and Eugene Braunwald, et al, eds. *Harrison's Principles of Internal Medicine*. 14<sup>th</sup> ed. Montreal: McGraw-Hill, 1998.
  5. Fitzpatrick, Thomas B., Arthur Z. Eisen, et al, eds. *Dermatology in General Medicine*. 4<sup>th</sup> ed. Montreal: McGraw-Hill, 1993.
  6. Goldman, Lee and J. Claude Bennett. *Cecil Textbook of Medicine*. 21<sup>st</sup> ed. Toronto: W. B. Saunders, 2000.
  7. Goroll, Allan H. and Albert G. Mulley, Jr. *Primary Care Medicine. Office Evaluation and Management of the Adult Patient*. 4<sup>th</sup> ed. Philadelphia: Lippincott Williams & Wilkins, 2000.
  8. Habif, Thomas P. *Clinical Dermatology*. 3<sup>rd</sup> ed. Toronto: Mosby-Year Book, 1996.