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NATURAL HISTORY AND PREVALENCE OF PSORIASIS

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ABSTRACT

The chronicle of psoriasis begins in ancient times when psoriasis, leprosy and some other chronic inflammatory dermatological disorders were thought to be the same. The disease is enormously variable in duration, periodicity of flares and extent. Morphological variants are also common. Prevalence varies according to race, genetic susceptibility, geographic location, demographic characteristics and environmental factors. Studies on the prevalence and incidence of psoriasis have contributed to better understand the burden of the disease. However, further studies are required to fill existing gap for understanding the prevalence and trends in incidence over time. Increasing prevalence of psychosocial impact on an individual affected with psoriasis is due to the altered immunity and continuous exposure to the stress which in turn resulted due to the irregular lifestyle and eating habits. Therefore, the history of psychological treatment is also important. The introduction of psychological treatment proved a substantial aid in the management of the disease, which help to improve the quality of life in psoriatic patients. The present review focused on the natural history of dermatological disorders, psoriasis, world-wide prevalence of psoriasis.

INTRODUCTION

Psoriasis is a chronic, inflammatory, T-cell-mediated autoimmune, multisystem disease that affects mainly skin and joints. It is one of the most common inflammatory skin diseases, affecting 2%–3% of the population worldwide [1].

The first description of psoriasis belongs to the period of the Roman Empire in the 1st century AD in the books of A. Cornelius Celsus 'De re medicalibriocto'. Celsus described it as the fourth variant of impetigo, a condition caused by *Staphylococcus pyogenes*. This condition appears as red patches with watery blisters on the skin. The disease was treated with medicaments containing pitch and sulphur. Interestingly, Celsus did not use the words psoriasis, psora, lepra [2]. Incidence is defined as the number of new cases of a disease in a particular time period while; prevalence is defined as the proportion of individuals in a population who have the disease of interest in a specified time period. Both measures, taken together provide important information for estimating the impact of a particular condition or illness.

The incidence and prevalence estimates in the literature, display a degree of variation and the main factors explaining this variation include: age, ethnicity, and geography. Another factor when considering the differing estimates of prevalence and incidence is different definitions of prevalence, sampling frames and age groups which are utilized in the various study designs [3]. Although, psoriasis is worldwide, its prevalence is highest in Scandinavian countries and Northern Europe (3%), it is about 2% in North America and the UK and about .2% in Japan. It contributes to approximately 1-5% of all skin disorders in Saudi Arabia. Some studies shows different prevalence rate for example, it is estimated around 3.5% in Europe and the United States which is higher when compared with Asian, native Indian and black population of African descent [4]. Population-based surveys from China and Japan have given a similar low prevalence ranging respectively from 0.05 to 1.23% and 0.29 to 1.18 %. The prevalence rate in India is (0.5%-2.3%) [5].

Psoriasis affects all age groups, genders, races and ethnicities, although some evidences suggested that psoriasis may be more prevalent in females than males. The majority of patients will present before the age of 35 with their first signs and symptoms of psoriasis. From an economic standpoint, an estimated 56 million hours of work are lost each year by people with psoriasis [6].

The prevalence and morbidity due to psoriasis is continuously increasing due to sedentary and modern stressful life style and increased incidence of metabolic co-morbidities like diabetes mellitus, hypertension, hyperlipidemia etc. It is twice more common in males compared to females and most of the patients present during their third or fourth decade of life [7].

Although traditionally psoriasis has been considered a dermatologic disease, contemporary medical literature is accumulating to support the assertion that psoriasis is actually a multisystem disorder. Psoriasis recently received a lot of scientific and medical attention in great part, because of major advances in our understanding of the disease and the consequent development of new treatments that are dramatically effective in many patients. Key areas of science in which advances have been made are genetics and immunology [8].

Various types of skin changes in the psoriatic patients are well known since biblical times. The first documentation belongs to the Old Testament in the third book of Moses, which means that humankind has dealt with this disease for at least 3000 years. Since then, the hypothesized causes of the disease have naturally evolved. The etiology of the disorder was considered to be due to hyper-proliferation and altered differentiation of the keratinocytes till the late 1970s [9-10]. After the 1980s various studies reported that activated T cells have a dominant pathogenic role in the initiation and persistence of psoriasis [11-12]. First, therapeutic success was found with medications that inhibit T-cell functions. The first of these pharmaceutical products was cyclosporinA, which reduces T-cell proliferation and cytokine production [13]. An improvement of psoriasis was also observed after treatment with other T-cell modulating drugs: antiCD4 antibodies [14-15] and a fusion protein composed of interleukin, (IL)-2 and fragments of diphtheria toxin [16].

NATURAL HISTORY

The Vedas are the world's oldest literature. The period of Vedas traced back to 6000-5000 BC. Among the four Vedas, the Atharva-Veda is by far the most important source of early references to medicine and it forms the foundation to the future systems of medicines especially Ayurvedic system of medicine. The Atharva Veda contains 700 hymns which contains the 6000 stanzas. Among these hymns, 114 are devoted to medical topics [17].

The CharakSamhita is the most important among the ancient comprehensive book on medicine and also considered the most authoritive text on medicine in Ayurvedic system of medicine. CharakSamhita described the various types of dermatological disorders under the roof of

'Kushtha'. It gives the detail description including etio-pathogenesis, clinical presentation, classification and management including topical and internal medication [18].

The CharakaSamhita clearly described the importance of psychosocial factors in the etio-pathogenesis of dermatological disorders. The described etiological factors can be summarized in to three categories which include Aharaja (dietary), Viharaja (lifestyle related) and Mansika (psychological). It also clearly described the psychological aspect of management for dermatological disorders including DaivavyapashrayaChikitsa (divine therapy) and SatvavajayaChikitsa (psycho-behavioral therapy) [19].

Psoriasis (OMIM 177900) is one of the oldest recorded skin diseases. Today, it is a well-known multi-system disorder, in which genetic, environmental and immunologic factors participate in etio-pathogenesis. The famous Hippocrates and his school (460–377 B.C.) used the term 'psora' which means 'to itch' and produced objective and meticulous descriptions of many skin disorders. In their classification, dry scaly eruptions were grouped together under 'lopoi' (epidermis). This group probably included psoriasis and leprosy. Psoriasis is basically resulted due to interaction between the genetic and environmental factors and frequently inherited and passed from one generation to the next, but not following a classical autosomal Mendelian profile [20].

A short description of a cutaneous disease called zaraath (Lepra in Greek)was found in the Leviticus. Some authors compared the lepra with leprosy, psoriasis, vitiligo, fungal diseases or even some other skin condition [21].

Psoriasis was again mentioned in the first century by Cornelius celsus, a Roman author. Galen (131-201 AD) was the first who used the term psoriasis. He used the term psoriasis for an itchy, scaly eruption of the eyelids and scrotum that was probably seborrhoeic dermatitis [22]. From 1000 – 1400 A.D. the prevalence of leprosy was very high. Many psoriatic patients, diagnosed as leprosy, received the same brutal treatment as leprosy patients; they were isolated from the community. The confusion between Psoriasis and leprosy remained for many centuries. The English dermatologist, Robert Willan (1757-1812) was the first person who recognized psoriasis as an independent disease and gave accurate description of psoriasis and its different manifestations. He identified two categories. "LeprosaGraecorum" was the term he used to describe the condition when the skin had scales. PsoraLeprosa described the condition when it became eruptive [23].

In 1841, Ferdinand Von Herba definitely separated the clinical picture of psoriasis from that of leprosy. Psoriasis has been described in great detail by Pussy (1933) and Bechet (1936). Simon (1949) has studied Psoriasis in Tropics and reported that psoriasis was 30 times more common in Europeans. Wright and Reed (1964) had proved definite link between Psoriatic arthritis and Reiter's disease. In 1977 –78 Seville has investigated the relationship between psoriasis and psychological stress [24-25]. Early concepts regarding the pathogenesis of psoriasis focused primarily on keratinocyte hyper-proliferation. However, the recent data suggest the role of the immune system in the pathogenesis. Psoriasis is a complex immune-mediated disease in which T-lymphocytes and dendritic cells play a central role. The complex interplay between cells from the skin and the immune system leads to chronic inflammation within the skin. Novel CD4+ T-helper (Th) cells, called Th17 cells, are recognized to be important in the pathogenesis of psoriasis [26]. The dependence of these diseases on Th17 cells rather than Th1 cells has led some to reclassify psoriasis and similar diseases as Th17 diseases [27].

In the past, physicians used many different com-pounds and serendipitously found several that helped to treat psoriasis, including arsenic (Fowler's solution) and ammoniated mercury. In many instances, neither the specific target nor the mechanism of action for the treatment was known. However, current treatments include a new wave of selective therapeutic agents[28]. The current therapeutic strategies include the topical medicament which is usually the first line of treatment, phototherapy and the internally used systemic agents. The systemic therapeutics includes conventional agents like methotrexate, systemic corticosteroids and the biological agents. Biological agents are a set of different engineered proteins. Biological therapies provide a targeted approach to treatment through interaction with specific components of the underlying immune and inflammatory disease processes.

Many studies validated the importance of stress and related psychological factors in the etio-pathogenesis of psoriasis. Therefore, introduction of psychological agent said to the treatment of psoriasis [29-30]. Psoriasis is reported to exert the psychosocial impact especially due to its visibility, chronicity and relapse which may change the behavior of affected individuals, resulting in obesity, increased alcohol consumption and an increased incidence of smoking. Smoking has been suggested to elicit psoriasis, whereas obesity appears to be a result of behavioral change in response to the condition. Reported relationship between alcoholism and psoriasis is possibly due to the psychological impact of psoriasis on affected individuals [31].

PREVALENCE

Psoriasis can manifests at any age, however, it exhibit a bimodal distribution of onset with majority of cases, approximately 75%, present before the age of 40 years, with a peak at 20–30 years. The remaining 25 % cases present after the age of 40 years. Patients with early onset tend to have a positive family history, frequent association with histocompatibility antigen (HLA)-Cw6, and more severe form of disease. Patients with later onset (after the age of 40) usually have a negative family history and a normal frequency of the Cw6 allele [32]. On the basis of these two types of onset Henseler and Christophers, differentiated into two subgroups: type I, which begins before the age of 40, and type II, which begins after the age of 40 [33].

Prevalence of psoriasis varies in different parts of the world. According to published reports, prevalence in different populations varies from 0% to 11.8% [34-37]. There is considerable variation of the prevalence between different populations of a comparable ethnic background or geographical location. A declarative survey from the USA has found 1.3% prevalence in African-Americans compared to 2.5 % in "Caucasians" [38]. In South Africa, the respective figures in Blacks (Bantu population) and Whites have been evaluated to be 1.5 % vs. 4% [39]. In South America, it seems more frequent in persons of Indian descent than in those of African descent [40]. Several epidemiological studies revealed that many disorders quite frequently associated with psoriasis, which are termed as co-morbidities. The common co-morbidities include arthritis, cardio-metabolic disorders including myocardial infarction, stroke, diabetes, obesity, dyslipidemia and non-alcoholic fatty liver disease. These comorbidities confer a higher mortality rate. The presence of any co-morbid diseases worsens the psoriasis. In contrast, atopic dermatitis and allergies are less frequently seen to be associated with psoriasis[41-43]. Prevalence of psychological co-morbidities is continuously increasing among the patients of psoriasis which also associated with reduced quality of life [44]. The psychological problems include poor self-esteem, sexual dysfunction, anxiety, depression and suicidal ideation, reported as high as 67% in one study [45]. In some studies it is found that the prevalence of depression was significantly higher in patients with psoriasis when compared with general population [46-49]. Prevalence studies from India are mostly hospital based. A study collected a comprehensive data from various medical colleges of India and found that the incidence of psoriasis among total skin patients ranged between 0.44 and 2.2%, with overall incidence of 1.02% [50]. Another study from north India reported the prevalence rate of 2.8 % [51].

CONCLUSION

Psoriasis is one among the oldest recorded skin disorders. History of dermatological disorders traced back to the most ancient literature of knowledge, the Vedas. The concept related to the pathophysiology of psoriasis changed continuously as the researches advanced. Previously it is considered as the disorder of keratinization which resulted due to the hyper-proliferation and differentiation of keratinocytes. But, now days it is well acknowledged that psoriasis is genetically determined, T-cell mediated multisystem disorder brought on by stress.

The historical review of psoriasis and its treatment history from the ancient time till today illustrate efficiently the complexity of its etio-pathogenesis and chronicity. Prevalence of psychosocial impact is continuously increasing due to urban lifestyle, irregular eating habits and regular exposure to stress. The further studies needed to better understand the prevalence of psoriasis including the prevalence of psychosocial impact. Due to the psychosocial and economic impact on an individual, psoriasis need the support of other scientific disciplines like molecular medicine, genetics, immunology and pharmaco-genetics for better understanding and management.

REFERENCES

- 1. Lukas Flatz, Curdin Conrad. Role of T-cell-mediated inflammation in psoriasis: pathogenesis and targeted therapy. Psoriasis: Targets and Therapy, 2013;3: 1–10.
- 2. Bechet PE. Psoriasis. A brief historical review, Arch DermSyph, 1936; 33(2): 327-334.
- 3. Parisi R., D. P. Symmons, C. E. Griffiths, D. M. Ashcroft. "Global epidemiology of psoriasis: a systematic review of incidence and prevalence." J Invest Dermatol, 2013; 133(2): 377-385.
- 4. Chandran V, Environmental factors of psoriasis and psoriatic arthritis. J Autoimmun, 2010; 34: J314-321.
- 5. Gudjonsson JE, Elder JT. Psoriasis: epidemiology. Clin Dermatol, 2007; 25: 535-546.
- 6. The psoriasis and psoriatic arthritis pocket guide. Authored by: Abby Van Voorhees, M.D., Steven R. Feldman, M.D., Ph.D., John Y. M. Koo, M.D., Mark G. Lebwohl, M.D., Alan Menter, M.D. Editor, Mary Bellotti, Editor, National Psoriasis Foundation. National Psoriasis Foundation 2009. Pp:1.
- 7. PreethaSelva, A clinical study to evaluate the effect of topical tazarotene in the treatment of plaque type psoriasis in a tertiary care hospital.International Journal of Phytopharmacology, 2015; 6(1): 42-46.
- 8. Menter A, Gottlieb A, Feldman SR, et al. Guidelines of care for the management of psoriasis and psoriatic arthritis. Section 1. Overview of psoriasis and guidelines of care for the treatment of psoriasis with biologics. J Am AcadDermatol, 2008; 58:826-850.
- 9. Hodge L, Comaish J S. Psoriasis: current concepts in management. Drugs, 1977; 13: 288–296.
- 10. Voorhees J J. Pathophysiology of psoriasis. Annu Rev Med, 1977; 28: 467–473.
- 11. Barker J N. Psoriasis as a T cell-mediated autoimmune disease. Hosp Med, 1998; 59: 530–533.
- 12. Ozawa M, Aiba S. Immunopathogenesis of psoriasis. Curr Drug Targets Inflamm Allergy, 2004; 3: 137–144.
- 13. Mueller W, Herrmann B. Cyclosporin A for psoriasis. N Engl J Med, 1979; 301: 555.
- 14. Nicolas J F, Chamchick N, Thivolet J, Wijdenes J, Morel P, Revillard J P. CD4 antibody treatment of severe psoriasis. Lancet, 1991; 338: 321.
- 15. Prinz J, Braun-Falco O, Meurer M et al. Chimaeric CD4 monoclonal antibody in treatment of generalized pustular psoriasis. Lancet 1991: 338: 320–321.
- 16. Gottlieb S L, Gilleaudeau P, Johnson R et al. Response of psoriasis to a lymphocyte-selective toxin (DAB389IL-2) suggests a primary immune, but not keratinocyte, pathogenic basis. Nat Med, 1995; 1: 442–447.

- 17. News, Notes and Queries-dermatological writings of Ancient India.http://citeseerx.ist.psu.edu/viewdoc/download;jsessionid=BF651D9679C10EE53A03B7F27DF0E5EE?do i=10.1.1.284.3403&rep=rep1&type=pdf. Accessed on 03/02/2014 & 24/05/2016
- 18. ShashtriRajeshwarDutta, upadhyaya Y, Pandey GS, Gupta B, editors. CharakaSamhita. Varanasi: Chaukhambhabharati academy; 2005.
- 19. ShashtriRajeshwarDutta, upadhyaya Y, Pandey GS, Gupta B, editors. CharakaSamhita. Varanasi: Chaukhambhabharati academy; 2005.
- 20. Brian J. Nickoloff1 and Frank O. Nestle. Recent insights into the immunopathogenesis of psoriasis provide new therapeutic opportunities. J. Clin. Invest., 2004; 113:1664–1675.
- 21. Goldman L., Moraites RS., Kitzmiller K. White spots in Biblical times. Arch Dermatol, 1966; 93(6):748-753.
- 22. Ines Brajac, Franjo Gruber. History of psoriasis. PDF available at http://cdn.intechopen.com/pdfs/32469/InTech-History of psoriasis.pdf. Accessed on 16/03/2014 & 24/05/2016.
- 23. Dr. Singh Satyapal. Clinical study of KitibhaKushtha (psoriasis) and its management with Samshodhan (Virechan) and Samshaman therapy. M.D. (Ay.) thesis. Banaras Hindu University: Varanasi, December 2010
- 24. Dr. Singh Satyapal. Clinical study of KitibhaKushtha (psoriasis) and its management with Samshodhan (Virechan) and Samshaman therapy. M.D. (Ay.) thesis. Banaras Hindu University: Varanasi, December 2010
- 25. Bechet PE. Psoriasis- A brief historical review. Arch DermSyph, 1936; 33(2):27-334.
- 26. Nakajima H, Nakajima K, Tarutani M, Sano S. Clear association between serum levels of adipokines and Thelper 17-related cytokines in patients with psoriasis. ClinExpDermatol, 2013; 38:66-70.
- 27. Nakajima K. Critical role of the interleukin-23/T-helper 17 in psoriasis. J Dermatol, 2012; 39:219-224.
- 28. Krueger J.G. The immunologic basis for the treatment of psoriasis with new biologic agents. J. Am. Acad. Dermatol., 2002; 46:1–23.
- 29. Gaston L, Lassonde M, Psoriasis and stress: a prospective study. J Am AcadDermatol, 1987; 17:82-86.
- 30. Griffiths CE, Richards HL: Psychological influences in psoriasis. ClinExpDermatol, 2001; 26: 338–342.
- 31. Singh Satyapal, Tripathi J.S., Rai N.P. An overview of Ayurvedic& contemporary approaches to Psychodermatology. The Journal of Phytopharmacology, 2014; 3(4): 286-299
- 32. Juliana L Basko-Plluska, VesnaPetronic-Rosic. Psoriasis: epidemiology, natural history and differential diagnosisPsoriasis: Targets and Therapy, 2012;2:67–76.
- 33. Henseler T, Christophers E. Psoriasis of early and late onset: characterization of two types of psoriasis vulgaris. J Am AcadDermatol, 1985; 13: 450–456.
- 34. Kaur I, Epidemiology of psoriasis in a clinic from India. Indian J Dermatol Venereol Leprol, 1986;52:208-12.
- 35. Bedi TR. Psoriasis in north India. Geographical variations. Dermatologica, 1977;155:310-4.
- 36. Swanbeck G, A population genetic study of psoriasis. Br J Dermatol, 1994;131:32-9.
- 37. Farber EM, Nall L. Epidemiology: natural history and genetics. In: RoenigkJr HH, Maibach HI, editors. Psoriasis. New York: Dekker; 1998. p. 107-57.
- 38. Gelfand JM, The prevalence of psoriasis in African Americans: results from a population-based study. J Am AcadDermatol.2005:52: 23-26.
- 39. Farber EM, Nall L (1998) Epidemiology: natural history and genetics. Psoriasis. New York: Dekker 107-157.
- 40. Suite M. The epidemiology of psoriasis in a dermatology clinic in a general hospital in Port-of-Spain, Trinidad and Tobago, West Indies. West Indian Med J, 2006; 55: 399-402.
- 41. Christophers E. Psoriasis epidemiology and clinical spectrum. ClinExpDermatol, 2001: 26: 314–320.
- 42. Gisondi P, Tessari G, Conti A, et al. Prevalence of metabolic syndrome in patients with psoriasis: a hospital-based case-control study. Br J Dermatol., 2007;157(1):68–73.
- 43. Singh Satyapal, Rai N.P.Co-morbidites with Psoriasis A Review. SangyaharanShodha, 2015; 18(1): 76-85.
- 44. Krueger G, Koo J, Stern RS, Rolstad T. The impact of psoriasis on quality of life: results of a 1998 National Psoriasis Foundation patient-membership survey. ArchDermatol, 2001;137(3): 280–284.
- 45. Esposito M, Chimenti S. An Italian study on psoriasis and depression. Dermatology, 2006; 212:123-7.
- 46. Sharma N. Psychiatric morbidity in psoriasis, vitiligo: a comparative study. J Dermatol, 2001; 28 (8):419-23.
- 47. Esposito M, Chimenti S. An Italian study on psoriasis and depression. Dermatology, 2006; 212 (2):123-7.
- 48. Akay A, Pekcanlar A, Bozdag KE, Altintas L, Karaman A. Assessment of depression in subjects with psoriasis vulgaris and lichen planus. J EurAcadDermatolVenereol, 2002; 16 (4):347-52.
- 49. Kurd SK, Troxel AB, Crits-Christoph P, Gelfand JM. The risk of depression, anxiety, and suicidality in patients with psoriasis: a population-based cohort study. Arch Dermatol, 2010;146(8):891–895.
- 50. Okhandiar RP,. Psoriasis in the tropics: An epidemiological survey. J Indian Med Assoc, 1963; 41: 550-6.
- 51. Bedi TR. Clinical profile of psoriasis in North India. Indian J DermatolVenereolLeprol, 1995;61: 202-5.