



Original Research Article

Decoding vitiligo: A review of demography, clinical features and associations at a tertiary care hospital in western Gujarat

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Abstract

Introduction: Vitiligo is a potentially harmless autoimmune skin condition causing white geographic patterns. It is a social taboo especially in India since ancient times. Vitiligo decreases the matrimonial prospects of the affected individuals. There is a variance in prevalence of vitiligo in different populations all over the world, Indian studies found the incidence to be 3-4% throughout India but an epidemic surge of 8.8% in the tropical states of Gujarat and Rajasthan. Oxidative stress is another causative factor. Even though extensively studied a lot still remains unknown about this disease. With this study we aim to shed light on some of the demographic, etiological, dietary and associated factors of vitiligo.

Objectives: 1. To determine demographic factors of vitiligo; 2. To describe clinical types of vitiligo; 3. To describe associated factors in patients with vitiligo.

Materials and Methods: All the patients diagnosed with vitiligo coming to the outpatient department (OPD) of the dermatology department of the medical college from 1st September to 31st December 2024 were part of this study. Additionally wood's lamp and dermoscopy were used to aid diagnosis.

Conclusion: Vitiligo is most frequent disorder in the tropical geographic region of western India in all age groups of both genders. Measures need to be taken to provide affordable and effective management for the patients along with psychological support to the affected individuals. Majority of the patients had dietary restrictions so nutrient deficiencies should be looked into as studies show role of diet in vitiligo. BMI in most of the patient was found to be average or below average so, diet habits seem to have role in vitiligo. Sunscreens should be added to treatment to prevent photo damage in sun exposed skin.

Keywords: Vitiligo, Generalized vitiligo, Acral vitiligo, Focal vitiligo, Mucosal vitiligo, Genital vitiligo, Segmental vitiligo, Non-segmental vitiligo, White patches.

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1. Introduction

Vitiligo is a cutaneous disorder in which melanin particularly disappears and causes bizarre geographic white patterns on the skin. The depigmented skin is left exposed to harmful Ultraviolet (UV) rays and sunburn especially on exposed body parts. It does not hinder physically but has a profound psychosocial effect as the patients feel alienated.¹ In certain regions including western India vitiligo has been given misnomer of leprosy in local language since ages further impacting the mental health of patients.² In depth studies of vitiligo have found it to be due to autoimmune causes along with environmental, genetic factors and oxidative stress.³ Tyrosinase, an enzyme involved in melanin biosynthesis encoded by TYR gene acts as an autoantigen for demelanisation.^{4,5} IFN- γ secreted by T cells trigger CXCL 9 and

CXCL 10 production by keratinocytes which further recruit T cells thus progressing vitiligo.^{6,7} Throughout world vitiligo has been reported to be 0.5-1%⁸ but it has epidemic proportions in Gujarat of 8.8%.⁹ The disease is more evident in the tropical region due to the sharp contrast of skin colour occurring due to depigmentation.¹⁰ Vitiligo most frequently occurs in genetically susceptible individuals and over 50 gene loci have been identified.^{11,12} The pigmentary disorder has also been found to be associated with various other autoimmune diseases.¹³ Diet low in proteins and minerals is also thought to be the causative factor of this pigmentary disorder.¹⁴ As per 2011 consensus segmental vitiligo has been diversified from non-segmental vitiligo because of its prognosis. Segmental type is associated with leucotrichia and is unusually unilateral and resistant to treatment. Vitiligo particularly constitutes non segmental vitiligo which is

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further diversified as focal, acrofacial, mucosal, generalized, universal, mixed and rare types. The course of vitiligo is unpredictable and may evolve into segmental or non-segmental.¹⁵ As observed in studies a quarter of patients develop vitiligo in the first decade, half of them in the second decade and up to 80% are affected by thirty years of life.¹⁶ Genetic association of vitiligo has been shown by involvement of 20% of first degree relatives with similar condition.¹⁷ Focus on nutritious diet has also been considered for recovery from this condition.¹⁸ In a study conducted at Birmingham Medical Centre at Alabama, folic acid and Vitamin B12 was found to be low in vitiligo patients and replacement of these vital nutrients took 3 years for cutaneous repigmentation in patients.¹⁹ Vitamin D deficiency has also been indicated in causation of vitiligo and evidence has been shown in decreased progression of disease on taking Vitamin D daily for 6 months.²⁰ Polypodium leukotomas, khellin and Ginkgo bilboa have been seen to enhance pigmentation in patients of vitiligo.²¹ Cytotoxic CD8+ T cells precipitate melanocyte destruction and autoimmunity in vitiligo.²² Histopathology though not imperative for diagnosis reveals nearly complete loss of pigment in epidermis and melanocytes in basal layer by Fontana-Masson stain. Initially epidermis shows interface dermatitis with CD8+ cytotoxic cells near melanocytes. The borders of lesion show perivascular and perifollicular infiltrate.²³ Vitiligo is diagnosed clinically with appearance of typical white macules presenting most frequently around mouth, eyes, lips, genitals, finger tips, toes and other frictional sites.²⁴ On wood's lamp examination - a hand held device emitting UVA light, lesions are accentuated with bluish white fluorescence hence, assisting the diagnosis of vitiligo (8). Dermoscopic examination of vitiligo reveals lingering perifollicular pigmentation in active stage and telengectasia (Figure 1) differentiating it from other hypopigmentary disorders.²⁵

2. Materials and Methods

All the patients who were diagnosed with vitiligo coming to the OPD of dermatology of the medical college from 1st September to 31st December 2024 were part of this study. This study was approved by the ethics committee of the institute. The diagnosis of patients was clinical and further evaluated by wood's lamp examination and dermoscopy. Patient's detailed family history along with history of associated diseases, dietary preferences, height, weight and addictions were recorded. Demographic analysis was also done using MS excel to deduce mean and percentage, which included age, sex and occupation of the patient. Body mass index (BMI) of all the patients was also calculated.

2.1. Inclusion criteria

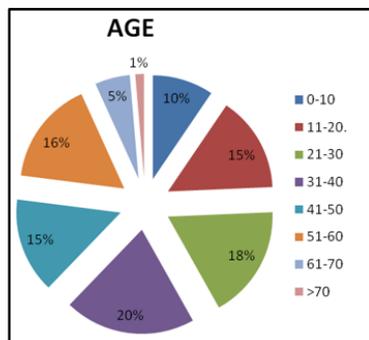
All the patients who were diagnosed with vitiligo by clinical examination, wood's lamp and dermoscopy.

2.2. Exclusion criteria

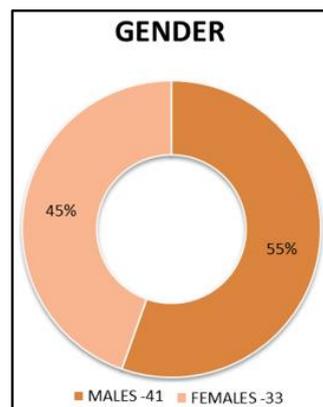
Patients who had hypo pigmented lesions like psoriasis, nevus depigmentosus, pityriasis versicolor, idiopathic guttate hypomelanosis, nevus anemicus, ash leaf macule, post inflammatory hypopigmentation etc.

3. Results

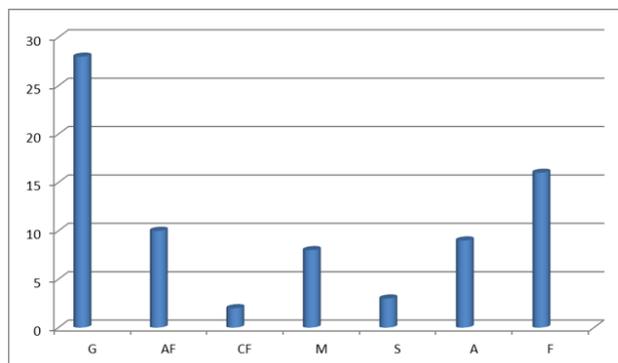
The study comprised of seventy-four patients of vitiligo attending dermatology OPD of Medical College out of a total of six thousand and three hundred patients which was 1.17% of the patients.



Graph 1: Age distribution of vitiligo patients



Graph 2: Gender wise distribution



Graph 3: Types of Vitiligo Presentation: Generalized(G), Acro-facial(AF), Confetti(C), Mucosal(M), Segmental(S), Acral(A), Focal(F).

3.1. Age range of subjects

The clinico-epidemiological profile, along with associated disorders and various patterns of vitiligo, was analyzed. The age range of patients varied from 2.5 months to seventy-five years (**Figure 1**), with a mean age of 36.3 years. The highest number of cases (fifteen patients, 20%) was observed in the thirty-one to forty years age group, followed by thirteen patients (18%) in the twenty-one to thirty years age group, twelve patients (16%) in the fifty-one to sixty years age group, and eleven patients each (15%) in the eleven to twenty years and forty-one to fifty years age groups. Among children from birth to ten years, 7 cases (10%) were reported. The lowest number of cases was seen in the sixty-one to seventy years age group, with 4 patients (5%), and only one patient (1%) was over seventy years old (**Graph 1**).

3.2. Gender, occupation and associated diseases profile

Gender wise male patients dominated with forty-one (55%) as compared to females which were thirty three (45%) (**Graph 2**). The age of onset of vitiligo was from adolescent to thirty-five years of age in fifty patients (67.5%). Associated diseases were present in twenty-two patients of this autoimmune disorder- 3 patients had hypertension, 4 had diabetes mellitus in middle age group, 2 children had atopic

dermatitis, 3 had obesity with acanthosis nigricans. One patient each had hypothyroidism, asthma, rheumatoid arthritis, lichen planus, alopecia areata, Parkinson's disease, prurigo, eczema, pityriasis versicolor (**Table 1**). Family history of vitiligo in first degree relative was positive in twelve patients (16.3%). Eighteen patients (24.4%) were addicted to tobacco smoking. Majority of patient were farmer by occupation and housewives each fifteen in number, fourteen were students, eleven were daily wagers or manual workers, 3 were in executive job & rest of the patients were in extremes of age either infants or retired persons. Number of vegetarian patients were fifty-four out of seventy-four (74%), remaining nineteen patients were non vegetarian.

3.3. BMI of patients

Fourteen (18.9%) patients were underweight with BMI below eighteen. Thirty-seven (50%) had BMI towards lower side between eighteen - twenty-five. Eighteen (24.3%) had BMI in the range of twenty-five - thirty while 5 (6.7%) were overweight with 30+ BMI. Approximately 70% patients of vitiligo patients had average or below average BMI (**Table 2**).

Table 1: Associated diseases in vitiligo patients

S.No.	Associated disease	Age	Sex	Type of vitiligo
1	Acanthosis Nigricans with Obesity	16	F	Focal
2	Hypertension	58	M	Focal
3	Diabetes	40	F	Acral
4	Vision	20	M	Focal
5	Hypertension	64	F	Generalized
6	Thyroid	40	F	Segmental
7	Hypertension	33	F	Focal
8	Atopic dermatitis	14	F	Focal
9	Asthma	50	F	Generalized
10	Obesity	15	F	Focal
11	Atopic dermatitis	2	M	Segmental
12	Lichen Planus	40	F	
13	Diabetes mellitus 1	47	F	Generalized
14	Eczema	52	M	Generalized
15	Prurigo	26	F	Acral
16	T. Corporis / Keloid	21	F	Acral
17	Alopecia Areata	23	M	Generalized
18	P. Versicolor	32	M	Focal
19	Facial Palsy	40	M	Generalized
20	Diabetes mellitus and Parkinsons	69	M	Acral
21	Diabetes mellitus 1	47	F	Generalized
22	Rheumatoid arthritis	60	F	Mucosal

Table 2: BMI of vitiligo patients

S.No	Category	BMI =kg/m2	BMI of Vitiligo patients
1.	Underweight	<18.5	14(18.9%)
2.	Normal weight	18.5 -24.9	37(50%)
3.	Overweight	25 -29.9	18(24.3%)
4.	Obese	>30	5(6.7%)

3.4. Types of vitiligo

Generalized vitiligo had the highest frequency of occurrence (**Graph 3**). A large number of patients had vitiligo patches on sun exposed sites, thirty-two had lesions on face, head and neck while twenty-four had acral presentation, 2 had involvement of genital mucosa. Segmental and confetti like lesions were in minimal number of patients.

4. Discussion

Generalized vitiligo involves large surface area of the body usually bilaterally symmetrical macules frequently on the pressure sites and those associated with friction. Vitiligo universalis (**Figure 2**) involves 80-90% body surface area with complete depigmentation of skin and hair. Acro-facial and acral vitiligo (**Figure 3**) involves sun exposed sites of face and distal extremities. Lip tip variety is also included under this. Mucosal vitiligo depigments oral (**Figure 4**) and genital mucosa (**Figure 5**). Focal vitiligo (**Figure 6**) is a singular lesion which lingers for a year or two before progressing to segmental (**Figure 7**) or non-segmental vitiligo. A combination of the lesions may be present which is called mixed vitiligo. Rare variants are Punctate vitiligo where lesions are confetti like 1-1.5mm size (**Figure 8**) and Hypochromic vitiligo or vitiligo minor present at the sites of seborrheic distribution such as trunk and scalp.



Figure 3: Acral vitiligo



Figure 4: Oral vitiligo (lip)

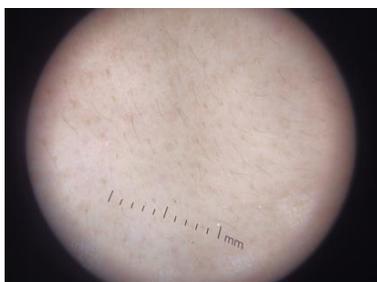


Figure 1: Dermoscopy image of vitiligo with perifollicular pigmentation



Figure 5: Genital vitiligo



Figure 2: Universal vitiligo



Figure 6: Focal vitiligo



Figure 7: Segmental vitiligo



Figure 8: Confetti vitiligo

Head is involved in 50% of the cases of vitiligo most commonly trigeminal dermatome.²⁶ Vitiligo makes an occurrence before 30 years of age usually and regression of the disease is rare on itself as it progresses for decades.^{27,28} Similar findings are revealed in our study. As reported earlier vitiligo precipitated in young adults below 35 years according to this data. About 20% cases have occurrence of vitiligo in close relatives¹⁷ and as per the above results 16.3% reported positive family history. Management of the patient requires evaluation and detailed history of the patient including personal and family history along with association of thyroid and other autoimmune diseases.²⁹ Studies have shown coexistence of thyroid and other autoimmune disorders like alopecia areata, rheumatoid arthritis, diabetes mellitus and Systemic lupus erythematosus, psoriasis and atopic dermatitis.³⁰ Recent study of vitiligo has shown it to be associated with lower BMI which is consistent with our study's findings as well as the results showed that 70% of our patients had an average or below average BMI.³¹

European Dermatology Forum formulated guidelines for vitiligo management with four graded options. Topical corticosteroids and calcineurin inhibitors are the first choice of treatment. Phototherapy in the form of NB-UVB, Psoralen PUVA and oral corticosteroids are given in the next step if first line is ineffective. Various surgical grafting techniques are tried when oral and topical treatment does not work. Last resort is the depigmentation to even out skin colour.³² Presently Ruxolitinib and Tofacitinib have been found to be

promising drugs and approved to manage vitiligo by targeting JAK.³³

5. Conclusion

Vitiligo is a very common disease in western India associated with a number of other diseases and it is a psychological havoc for the patients. Its exact cause is yet to be unearthed. BMI below average and average towards lower side signify role of proper diet and protein intake in vitiligo. Micronutrient deficiencies should be covered as studies show the relevance in vitiligo. Sunscreens should be added to treatment to prevent photo damage to sun exposed parts. Measures need to be taken to provide affordable management for the patients along with psychological support incorporated in the guidelines.

6. Source of Funding

None.

7. Conflict of Interest

None.

References

- Ezzedine K, Grimes P, Meurant JM, Seneschal J, Léauté-Labrèze C, Ballanger F, et al. Living with vitiligo: results from a national survey indicate differences between skin phototypes. *Br J Dermatol*. 2015;173(2):607–9.
- Porter JR, Beuf AH, Lerner A, Nordlund J. Psychosocial effect of vitiligo: a comparison of vitiligo patients with “normal” control subjects, with psoriasis patients, and with patients with other pigmentary disorders. *J Am Acad Dermatol*. 1986;15(2):220–4.
- Picardo M, Dell'Anna ML, Ezzedine K, Hamzavi I, Harris JE, Parsad D, et al. Vitiligo. *Nat Rev Dis Primers*. 2015;1:15011.
- Baharav E, Merimski O, Shoenfeld Y, Zigelman R, Gilbrud B, Yechezkel G, et al. Tyrosinase as an autoantigen in patients with vitiligo. *Clin Exp Immunol*. 1996;105(1):84–8.
- Spritz RA, Hearing Jr VJ. Genetic disorders of pigmentation. *Adv Hum Genet*. 1994;22:1–45.
- Rashighi M, Agarwal P, Richmond JM, Harris TH, Dresser K, Su MW, et al. CXCL10 is critical for the progression and maintenance of depigmentation in a mouse model of vitiligo. *Sci Transl Med*. 2014;6(223):223ra23–ra23.
- Richmond JM, Masterjohn E, Chu R, Tedstone J, Youd ME, Harris JE. CXCR3 depleting antibodies prevent and reverse vitiligo in mice. *J Invest Dermatol*. 2017;137(4):982–5.
- Taïeb A, Picardo M. The definition and assessment of vitiligo: a consensus report of the Vitiligo European Task Force. *Pigment Cell Res*. 2007;20(1):27–35.
- Behl P, Kapoor T, Majumdar M. Epidemiological study of vitiligo. *Dermatol Times*. 1988;9:1–3.
- Sehgal VN, Srivastava G. Vitiligo: compendium of clinico-epidemiological features. *Indian J Dermatol Venereol Leprol*. 2007;73(3):149–56.
- Shajil E, Agrawal D, Vagadia K, Marfatia Y, Begum R. Vitiligo: clinical profiles in Vadodara, Gujarat. *Indian J Dermatol*. 2006;51(2):100–4.
- Spritz RA, Andersen GH. Genetics of Vitiligo. *Dermatol Clin*. 2017;35(2):245–55.
- Dutta A, Mandal S. A clinical study of 650 vitiligo cases and their classification. *Indian J Dermatol*. 1969;14(3):103–11.
- Behl PN. Practice of dermatology: Thomson Press (India); 1975.
- Ezzedine K, Lim HW, Suzuki T, Katayama I, Hamzavi I, Lan CC, et al. Revised classification/nomenclature of vitiligo and related

- issues: the Vitiligo Global Issues Consensus Conference. *Pigment Cell Melanoma Res.* 2012;25(3):E1-13.
16. Lee H, Lee M-H, Lee DY, Kang HY, Kim KH, Choi GS, et al. Prevalence of vitiligo and associated comorbidities in Korea. *Yonsei Med J.* 2015;56(3):719–25.
 17. Nath SK, Majumder PP, Nordlund JJ. Genetic epidemiology of vitiligo: multilocus recessivity cross-validated. *Am J Hum Genet.* 1994;55(5):981–90.
 18. Di Nardo V, Barygina V, França K, Tirant M, Valle Y, Lotti T. Functional nutrition as integrated approach in vitiligo management. *Dermatol Ther.* 2019;32(4):e12625.
 19. Montes LF, Diaz M, Lajous J, Garcia N. Folic acid and vitamin B12 in vitiligo: a nutritional approach. *Cutis.* 1992;50(1):39–42.
 20. Finamor DC, Sinigaglia-Coimbra R, Neves LC, Gutierrez M, Silva JJ, Torres LD, et al. A pilot study assessing the effect of prolonged administration of high daily doses of vitamin D on the clinical course of vitiligo and psoriasis. *Dermatoendocrinol.* 2013;5(1):222–34.
 21. Dutta RR, Kumar T, Ingole N. Diet and vitiligo: the story so far. *Cureus.* 2022;14(8):e28516.
 22. Van Den Boom JG, Konijnenberg D, Dellemijn TA, Van Der Veen JW, Bos JD, Melief CJ, et al. Autoimmune destruction of skin melanocytes by perilesional T cells from vitiligo patients. *J Invest Dermatol.* 2009;129(9):2220–32.
 23. Kim YC, Kim YJ, Kang HY, Sohn S, Lee ES. Histopathologic features in vitiligo. *Am J Dermatopathol.* 2008;30(2):112–6.
 24. Alikhan A, Felsten LM, Daly M, Petronic-Rosic V. Vitiligo: a comprehensive overview: part I. Introduction, epidemiology, quality of life, diagnosis, differential diagnosis, associations, histopathology, etiology, and work-up. *J Am Acad Dermatol.* 2011;65(3):473–91.
 25. Kumar Jha A, Sonthalia S, Lallas A, Chaudhary R. Dermoscopy in vitiligo: diagnosis and beyond. *Int J Dermatol.* 2018;57(1):50–4.
 26. Hann SK, Park YK, Chun WH. Clinical features of vitiligo. *Clin Dermatol.* 1997;15(6):891–7.
 27. Millington GW, Levell NJ. Vitiligo: the historical curse of depigmentation. *Int J Dermatol.* 2007;46(9):990–5.
 28. Nordlund JJ. The medical treatment of vitiligo: An historical review. *Dermatol Clin.* 2017;35(2):107-16.
 29. Mason C, Gawkrödger D. Vitiligo presentation in adults. *Clin Exp Dermatol.* 2005;30(4):344–5.
 30. Alkhateeb A, Fain PR, Thody A, Bennett DC, Spritz RA. Epidemiology of vitiligo and associated autoimmune diseases in Caucasian probands and their families. *Pigment Cell Res.* 2003;16(3):208–14.
 31. Sharifzadeh A, Wetstone R, Chen LC, Guilarte-Walker Y, Flahive J, Harris JE. Vitiligo is associated with lower body mass index: a retrospective case-control study. *Br J Dermatol.* 2025;192(4):753–5.
 32. Taieb A, Alomar A, Böhm M, Dell'anna M, De Pase A, Eleftheriadou V, et al. Vitiligo European Task Force (VETF); European Academy of Dermatology and Venereology (EADV); Union Européenne des Médecins Spécialistes (UEMS). Guidelines for the management of vitiligo: the European Dermatology Forum consensus. *Br J Dermatol.* 2013;168(1):5–19.
 33. Jackson SW, Jacobs HM, Arkatkar T, Dam EM, Scharping NE, Kolhatkar NS, et al. B cell IFN- γ receptor signaling promotes autoimmune germinal centers via cell-intrinsic induction of BCL-6. *J Exp Med.* 2016;213(5):733–50.

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