

Clinical patterns of vitiligo and its associated co morbidities: A prospective controlled cross-sectional study in South India

D. S. Krupa Shankar, K. Shashikala¹, Rama Madala

Department of Dermatology, Old Airport Road, Manipal Hospital, Bangalore, India.
¹Skin Diagnosis Centre, Mahabodhi Mallige Hospital, Jayanagar 1st Block, Bangalore, India.

ABSTRACT

Aim: The purpose of this study is to assess the clinical patterns and associations of vitiligo, audiometric functions, and ocular involvement and to correlate the morphology, clinical behaviour and comorbidities associated with vitiligo. **Settings and Design:** For this prospective and cross-sectional study 80 self-reporting patients in the age group 7-75 years with vitiligo attending the outpatient department of Manipal hospital during the period August 2008 to February 2010 were selected and the data was analysed. **Materials and Methods:** The patients were subjected to detailed history, clinical examination and investigations [complete blood count (CBC), absolute eosinophil count (AEC), erythrocyte sedimentation rate (ESR), thyroid stimulating hormone (TSH), vitamin B12 estimation, fasting blood sugar (FBS), and post prandial blood sugar (PPBS), antibody titre estimations that is antithyroid peroxidase (ATPA), antithyroglobulin (ATA), antinuclear antibodies (ANA), urine analysis], audiometric evaluation and ophthalmic examination. **Statistical Analysis Used:** The Fisher exact test has been used to find the significance of study parameters on categorical scale between two or more groups. **Results:** In the present series of 80 cases, 41 (51.25%) were males and 39 (48.75%) were females. The male to female ratio was 1.05:1. In our study 20% cases gave definite family history of vitiligo and patients in the age group of 20 - 30 years were the most commonly affected. Generalized vitiligo (31.3%) was the most common type followed by segmental (30%), focal (18.8%), acrofacial (8.8%), and mucosal vitiligo (11.3%). In the present study there was a high incidence of autoantibodies (22.5%), vitamin B12 deficiency (30%), hypothyroidism (11.3%), elevated absolute eosinophil count (16.3%), hypoacusis (10%) and retinal changes (8.8%). This suggests multisystem autoimmunity in vitiligo.

Keywords: Deafness, retinal changes, thyroid disease, vitiligo, vitamin B12 deficiency

Access this article online

Website: www.idoj.in

DOI: 10.4103/2229-5178.96705

Quick Response Code:



INTRODUCTION

Vitiligo is a commonly acquired, idiopathic, heritable depigmentary disorder of the skin and/or mucous membranes.^[1] It is of major social and cosmetic concern in India, characterised by depigmented macules of varying size and shape.^[2] There are no other textural changes besides loss of color.^[2] Vitiligo occurs worldwide with an overall prevalence of 1%.^[3-6] However, its incidence ranges from 0.1 to >8.8% across the country and other parts of the world.^[3] Widespread prejudices, ignorance, taboos, lack of scientific appraisal about vitiligo and confusion with leprosy all make it a social embarrassment for the patient.^[7,8] However, life expectancy is unaffected.^[7,9]

The present study was conducted to assess the clinical patterns of vitiligo and its associated

comorbidities, audiometric function, and ocular involvement.

MATERIALS AND METHODS

This study was conducted on 80 vitiligo successive patients reporting to the outpatient department at Manipal hospital during the period of August 2008 to February 2010. All patients diagnosed to be having vitiligo willing to enrol for the study and above the age of 5 years were included in the study. Unwilling patients, patients below the age of 5 years and patients with established auditory or ophthalmic abnormalities due to other causes were excluded from the study.

Details of dietary habits and family history were taken in each case. History of associated diseases such as diabetes mellitus, thyroid

Address for correspondence:
 Dr. D. S. Krupa Shankar
 Department of Dermatology, Manipal Hospital, Old Airport Road, Bangalore - 590 017, India.
 E-mail: dermakrupa@gmail.com

disorders, pernicious anaemia and alopecia were noted. History of precipitating or initiating factors especially physical trauma, sun exposure, acute mental or emotional stress, contact with chemicals or synthetic footwear was noted.

Patients were subjected to a detailed history, clinical examination, and investigations which included complete blood count (CBC), absolute eosinophil count (AEC), erythrocyte sedimentation rate (ESR), thyroid stimulating hormone (TSH), vitamin B12 estimation, fasting blood sugar (FBS), postprandial blood sugar (PPBS), antibody titre estimations like antithyroid peroxidase (ATPA), antithyroglobulin (ATA), antinuclear antibodies (ANA), urine analysis, audiometric evaluation, and ophthalmic examination.

RESULTS

Of all patients included in the study, 41 (50.25%) were males and 39 (49.75%) were females and no gender preponderance as with other studies [Table 1]. The age of patients ranged from 7 - 75 years, with a mean age of 32.4 years. The duration of illness ranged from half a month to 60 years with a mean of 46.9 months [Table 2]. Twenty percent of patients gave a family history of vitiligo and first-degree relatives were most commonly affected [Table 3]. There were 59 (73.8%) cases of unstable vitiligo, and 21 (26.3%) stable patients. Stable vitiligo was statistically associated with a significant family history [Table 4]. Majority of cases were generalized type (31.3%) followed by segmental (30%), focal (18.8%), acro-facial (8.8%) and mucosal type (11.3%) [Table 5]. Complete audiological examination revealed that 8 (10%) patients were suffering from hypoacusis [Table 6]. Retinal examination showed that 7 (8.8%) patients had abnormal retinal findings such as lattice degeneration, retinal atrophy and pigmentation [Table 7]. A disease duration of less than 6 months was statistically associated with generalised vitiligo and duration of 6 - 12 months with focal type of vitiligo [Table 8]. Disease duration of more than 6 months was associated with higher absolute eosinophil count (AEC) and higher PPBS. Patients with vitiligo aged more than 30 years were significantly associated with high PPBS, high incidence of autoantibodies (22.5%) [Figure 1], vitamin B12 deficiency (30%), hypothyroidism (11.3%), high AEC (16.3%) [Figure 2], hypoacusis (10%) and retinal changes (8.8%). Incidence of auditory ($P=0.638$) and retinal abnormalities ($P=0.973$) was not statistically associated with type of vitiligo.

DISCUSSION

Vitiligo is a commonly acquired, idiopathic, heritable depigmentary disorder of skin and/or mucous membrane.^[1] Statistics about vitiligo are variable. Vitiligo occurs worldwide with an overall prevalence of 1%.^[3,4] Patients suffer from a poor body image and low self-esteem and also experience a considerable level of psychological burden.^[11] This disorder does not result in restriction of working capacity or life expectancy but it causes cosmetic disfigurement leading to psychological trauma to the patient. Since ancient times, patients with vitiligo have suffered the same mental abuse as patients with leprosy, and the two diseases have often been confused clinically.^[11,12] Although vitiligo affects both sexes equally,^[13] most of the studies show a female preponderance.^[14,15] The cause of female preponderance is probably because of greater cosmetic awareness and the impact of the disease on their social life.^[7] Our study had a male to female ratio of 1.05:1 and there was no gender preponderance. Vitiligo is considered to be a multifactorial disorder. An assortment of hypotheses, such as genetic, neural, biochemical (autocytotoxic), and autoimmune have been put forth to elucidate its aetiopathologic mechanism.^[2]

Three major factors seem to be involved in the destruction of melanocytes in patients of vitiligo.^[16,17] The first is that vitiligo patients inherit a set of three vitiligo genes that predispose them to destruction of melanocytes.^[16,18-20] The second factor is that melanocytes in vitiligo patients differ from that of normal persons. Melanocytes in vitiligo patients are more sensitive to phenolic chemicals and require different, more fastidious culture conditions than those of normal individuals.^[21-23] The third factor is destruction of melanocytes occur due to activation of vitiligo genes by environmental agents, which in turn leads to activation of autoimmunity and apoptosis of melanocytes.^[16,24-30] Clinically, vitiligo is classified depending upon the site and extent of involvement into following types: generalized which is the most common, segmental, focal, acro-facial and mucosal type.^[31] In our study, generalized vitiligo (31.3%) was the most common type followed by segmental and other types.

Various studies have shown family history ranging from 7-36% and first-degree relatives were the most commonly affected. About 20% of vitiligo patients have at least one first-degree relative with vitiligo and the relative risk of vitiligo for first-degree relatives (parents, children, siblings) is elevated 7-10 fold as per the observations made in other similar

Table 1: Sex distribution in comparison with other studies

Parameters	Our study (80 pts)	Gopal <i>et al.</i> ^[36]	Dave <i>et al.</i> ^[34]	Shajil <i>et al.</i> ^[48]	Shah <i>et al.</i> ^[49]	Moradi <i>et al.</i> ^[41]
Males	51.25%	54%	51.2%	35.44%	31.6%	35%
Females	48.8%	46%	48.8%	61.56%	68.4%	65%
M : F	1.05:1	1.17:1	1.05:1	1:1.6	1:2.1	1:1.86

Table 2: Age distribution in comparison with other studies

Parameters	Our study (80 pts)	Gopal <i>et al.</i> ^[36]	Shajil <i>et al.</i> ^[48]
Age (years)	7-75	10-55	
Mean age (years)	32.4	23	25.59
Duration of disease (months)	0.5-720	0.5-372	0.5-720
Mean duration(months)	46.9	43.2	39.6
Mean ± SD: 32.39±16.90			

Table 4: Stable vitiligo and family history

Family history of vitiligo	Number of patients	Stable	
		Yes	No
Absent	66	16 (24.2%)	50 (75.8%)
Present	14	5 (35.7%)	9 (64.3%)
Total	80	21 (26.3%)	59 (73.8%)

Stable vitiligo patients had significant family history with P=0.504

Table 6: Age and audiological findings

Age in years	Number of patients (n=80)	Audio logical findings	
		Normal	Abnormal
1-10	8	8 (11.1%)	0
11-20	9	9 (12.5%)	0
21-30	23	23 (31.9%)	0
31-40	21	20 (27.8%)	1 (12.5%)
41-50	5	4 (5.6%)	1 (12.5%)
51-60	7	5 (6.9%)	2 (25.0%)
>60	7	3 (4.2%)	4 (50.0%)
Total	80	72 (100.0%)	8 (100.0%)

Patients aged more than 30 years significantly associated with abnormal auditory findings with P=0.005**

studies.^[32] Second-degree relatives also have significantly elevated relative risks. In our study, 20% of patients gave a definite family history of vitiligo, of which first-degree relatives were 8.75%, second degree relatives 7.5% and third degree relatives were 3.5%.

Younger people were more frequently affected and had active vitiligo compared to older people according to few studies.^[33] In our study also the younger age groups were commonly affected. This suggests a genetic factor in vitiligo. The presence of a positive family history, mucosal involvement, the isomorphic Koebner's phenomenon, nonsegmental vitiligo and mucosal vitiligo are associated with progressive disease as per the observation made by other authors and also in our study.^[34] Autoimmunity seems to play a significant role in its causation in a considerable number of patients.^[35-39] Various studies suggest that patients with vitiligo have an increased risk of developing autoimmune disease such as thyroid disease, Addison's disease,

Table 3: Family history of vitiligo

Parameters	Our study (80 pts)	Gopal. <i>et al.</i> ^[36]	Dave <i>et al.</i> ^[34]	Shajil <i>et al.</i> ^[48]	Behl <i>et al.</i> ^[33]	Shah <i>et al.</i> ^[49]
Family history	20%	36%	7.5%	21.93%	8.4%	13.7%
1 st degree	8.75%	22%			4.8%	
2 nd degree	7.5%	14%			2.9%	
3 rd degree	3.75%				0.86%	

First-degree relatives were most commonly affected

Table 5: Clinical types of vitiligo

Parameters	Our study (80 pts)	Gopal. <i>et al.</i> ^[36]	Martis <i>et al.</i> ^[7]	Shajil <i>et al.</i> ^[48]	Behl <i>et al.</i> ^[33]	Shah <i>et al.</i> ^[49]
Generalized	31.3%	48	39	52.36	47.5	64.9
Segmental	30%	13.34	3	6.84	5.3	1.4
Acro-facial	8.8%	22.67	18	7.55	41.6	0.8
Focal	18.8%	16	27	28.54		18.6
Mucosal	11.3%	0	5	2.83	5.6	14.8
Universal			1	1.89		8.2
			7			

Generalized type was the commonest in all studies and even in our study

Table 7: Age and retinal findings

Age in years	No of patients (n=80)	Ocular findings	
		Normal	Abnormal
1-10	8	8 (10.9%)	0
11-20	9	9 (12.3%)	0
21-30	23	23 (31.5%)	0
31-40	21	16 (21.9%)	5 (71.4%)
41-50	5	4 (5.5%)	1 (14.3%)
51-60	7	6 (8.2%)	1 (14.3%)
>60	7	7 (9.6%)	0
Total	80	73 (100.0%)	7 (100.0%)

Patients aged more than 30 years significantly associated with abnormal retinal findings with P=0.012*

pernicious anaemia, diabetes mellitus, pemphigus vulgaris and alopecia areata.^[38-44] In the present study, there was a high incidence of autoantibodies (22.5%) and hypothyroidism (11.3%) favouring autoimmunity in vitiligo. Other conditions with proposed autoimmune mechanism such as alopecia areata, pernicious anaemia were however not found in any of our cases.

Vitiligo patients may present various pigment changes in the fundus, in particular atrophic spots in the retinal pigment epithelium or chorioretinal scars, probably related to previous inflammatory events. Uveitis is the more common in patients with vitiligo.^[10]

Table 8: Duration of disease versus clinical types

Duration in months	No. of patients	Type of Vitiligo					
		Generalized (%)	Segmental (%)	Focal (%)	Mucosal (%)	Mucosal + Acral (%)	Acro-facial (%)
Less than 6 months	32	4 (12.5)	10 (31.3)	10 (31.3)	6 (18.8)	1 (3.1)	1 (3.1)
6-12 months	13	5 (38.5)	5 (38.5)	3 (23.1)	0 (0)	0 (0)	0 (0)
12-60 months	20	9 (45.0)	5 (25)	1 (5)	1 (5)	2 (10)	2 (10)
>60 months	15	7 (46.7)	4 (26.7)	1 (6.7)	2 (13.3)	1 (6.7)	0 (0)
Total	80	25 (31.3)	24 (30)	15 (18.8)	9 (11.3)	4 (5)	3 (3.8)
P value		0.022*	0.872	0.063+	0.284	0.562	0.416

Duration is statistically associated with type of Vitiligo with $P=0.075$ (4x6 Fisher exact test)

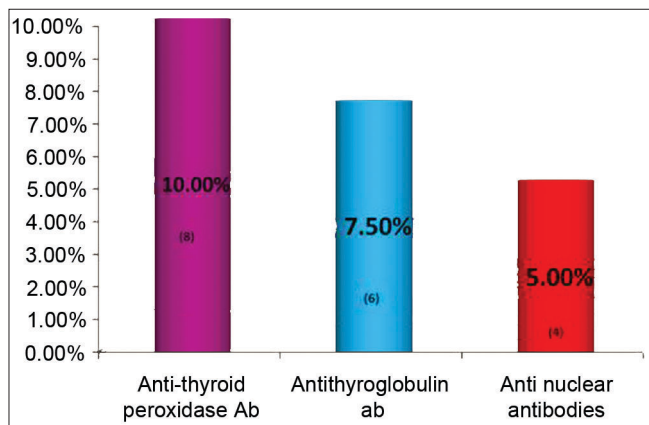


Figure 1: Antibody titre estimation of vitiligo patient studied

Deficiency of vitamin B12 associated with vitiligo was reported many years ago^[45,46] probably due to defective absorption. Studies reported excellent repigmentation in patients with vitiligo after 1-2 years of treatment with oral folic acid, ascorbic acid and parental B12 (100 mg intramuscular).^[46,47] Pteridine part of folic acid could interfere with the recycling of the reduced pterins in vitiligo. Vitamin B12 downregulates the formation of homocysteine, which appears to cause the depigmentation in vitiligo.^[45] Systemic oxidative stress has a pathophysiological role in precipitating all clinical types of vitiligo.^[48]

The incidence of audiological and retinal abnormalities were not statistically associated with type of vitiligo, which is in agreement with other studies.

CONCLUSION

Findings in our study correlate with other studies, and this clearly establishes that the pathophysiology of vitiligo is complex and systemic rather than focal. Therefore all vitiligo patients irrespective of the duration of illness or morphology must be investigated for hypothyroidism, thyroid and nuclear autoantibodies, vitamin B12 deficiency, hearing defects and retinal changes. Duration less than 6 months is statistically associated with generalized and 6-12 months with focal type of Vitiligo. Age and gender is not associated with any specific

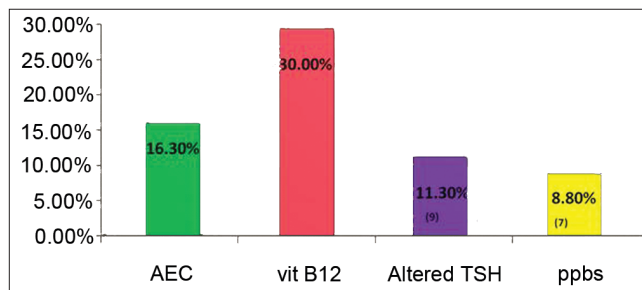


Figure 2: Laboratory evaluation of vitiligo patients studied. [AEC-absolute eosinophil count, vit-vitamin, TSH-thyroid stimulating hormone, ppbs-post prandial blood sugar]

type of vitiligo. Age of more than 30 years is significantly associated with abnormal audiometry, abnormal retinal findings, high AEC and high PPBS. Our study does not address adrenal disease and myasthenia gravis, which are rare but known associations of vitiligo. Notwithstanding this, we recommend that this short list of investigations and consultations be offered to all newly diagnosed adult patients of vitiligo, given the high diagnostic yield and its implications for general health. Our study underlines the fact that vitiligo is the skin manifestation of an internal disease.

REFERENCES

- Garg BJ, Saraswat A, Bhatia A, Katore OP. Topical treatment in vitiligo and the potential uses of new drug delivery systems. *Indian J Dermatol Venereol Leprol* 2010;76:231-8.
- Kar PK. Vitiligo: A study of 120 cases. *Indian J Dermatol Venereol Leprol* 2001;67:302-4.
- Sehgal VN, Srivastava G. Vitiligo: Compendium of clinico-epidemiological features. *Indian J Dermatol Venereol Leprol* 2007;73:149-56.
- Nogueira LS, Zancanaro PC, Azambuja RD. Vitiligo and emotions. *Bras Dermatol* 2009;84:41-5.
- Moscher DB, Fitzpatrick TB, Ortonne JP, Hori Y. Hypomelanism and hypermelanosis. In : Eisen AZ, Wolff K, Austen, KF, Goldsmith LA, Kats SI, Fitzpatrick TB, editors. *Dermatology in General Medicine*. New York: MC Graw Hill; 1999. p. 945-1017.
- Agrawal D, Sahani MH, Gupta S, Begum R. Vitiligo etipathogenesis and therapy: A Review. *J Maharaja Sayajirao Univ of Baroda* 2001; 48:97.
- Martis J, Bhat R, Nandakishore B, Shetty JN. A clinical study of vitiligo. *Indian J Dermatol Venereol Leprol* 2002;68:92-3.
- Koronke RV, Sachdevo KG. Vitiligo. *Int J Dermatol* 1998;27:676-81.
- Nair BK. Vitiligo: A retrospect. *Int J Dermatol* 1978;17:755-7.

10. Tosti A, Piraccini BM, Matildelozzo, Tosti G. Ocular and Audio logical Disorders. In: Lotti T, Hercogova J, editors. Vitiligo, problems and solutions: Italy: crechrepublic; 2004. p. 201-6.
11. Mashayekhi V, Javidi Z, Kiafar B, Manteghi AA, Saadatian V, Esmaeili AH, *et al.* Quality of life in patients with vitiligo: A descriptive study on 83 patients attending a PUVA therapy unit in Imam Reza Hospital, Mashad. *Indian J Dermatol Venereol Leprol* 2010;76:592.
12. Schmid-ott G, Kunsebeck HW, Jecht E. Stigmatization experience, coping and sense of coherence in vitiligo patients. *J Eur Acad Dermatol Venereol* 2007;21:456-61.
13. Sarin RC, Kumar AJ. A clinical study of vitiligo. *Indian J Dermatol Venereol Leprol* 1977;43:311-14.
14. Howitz J, Brodhagen H, Schwartz M, Thomsen K. Prevalence of vitiligo. *Arch Dermatol* 1977;113:47-52.
15. Bar S, Feiweil M, Chanarin I. Vitiligo and its aetiological relationship to organ specific auto-immune disease. *Br J Dermatol* 1969;81:83.
16. Nordlund JJ. Vitiligo: A review of some facts lesser known about depigmentation. *Indian J Dermatol* 2011;56:180-9
17. Boissy R, Nordlund JJ. Vitiligo: Current medical and scientific understanding. *G Ital Dermatol et Venereol* 2011. p. 46.
18. Majumder PP, Das SK, Li CC. A genetical model for vitiligo. *Am J Hum Genet* 1988;43:119-25.
19. Majumder PP, Nordlund JJ, Nath SK. Pattern of familial aggregation of vitiligo. *Arch Dermatol* 1993;129:994-8.
20. Nath SK, Majumder PP, Nordlund JJ. Genetic epidemiology of vitiligo: Multilocus recessivity cross-validated. *Am J Hum Genet* 1994;55:981-90.
21. Puri N, Mojamdar M, Ramaiah A. In vitro growth characteristics of melanocytes obtained from adult normal and vitiligo subjects. *J Invest Dermatol* 1987;88:434-8.
22. Puri N, Mojamdar M, Ramaiah A. Growth defects of melanocytes in culture from vitiligo subjects are spontaneously corrected in vivo in repigmenting subjects and can be partially corrected by the addition of fibroblast-derived growth factors in vitro. *Arch Dermatol Res* 1989;281:178-84.
23. Medrano EE, Nordlund JJ. Successful culture of adult human melanocytes obtained from normal and vitiligo donors. *J Invest Dermatol* 1990;95:441-5.
24. Nordlund J, Le Poole IC, Boissy R. Vitiligo vulgaris, in clinical and basic immunodermatology. In: Tyring AG, editor. London: Springer Verlag; 2008. p. 661-90.
25. Le Poole IC, van den Wijngaard RM, Westerhof W, Das PK. Presence of T cells and macrophages in inflammatory vitiligo skin parallels melanocyte disappearance. *Am J Pathol* 1996; 148:1219-28.
26. Palermo B, Campanelli R, Garbelli S, Mantovani S, Lantelme E, Brazzelli V, *et al.* Specific cytotoxic T lymphocyte responses against Melan-A/MART1, tyrosinase and gp100 in vitiligo by the use of major histocompatibility complex/peptide tetramers: The role of cellular immunity in the etiopathogenesis of vitiligo. *J Invest Dermatol* 2001;117:326-32.
27. Le Poole IC, Stennett LS, Bonish BK, Dee L, Robinson JK, Hernandez C, *et al.* Expansion of vitiligo lesions is associated with reduced epidermal CDw60 expression and increased expression of HLA-DR in perilesional skin. *Br J Dermatol* 2003;149:739-48.
28. Basak PY, Adiloglu AK, Ceyhan AM, Tas T, Akkaya VB. The role of helper and regulatory T cells in the pathogenesis of vitiligo. *J Am Acad Dermatol* 2009;60:256-60.
29. Le Poole IC, Luiten RM. Autoimmune etiology of generalized vitiligo. *CurrDirAutoimmun* 2008;10:227-43.
30. Huang CL, Nordlund JJ, Boissy R. Vitiligo: A manifestation of apoptosis? *Am J ClinDermatol* 2002;3:301-8.
31. Singh M, Singh G, Kanwar AJ, Belhaj MS. Clinical Pattern of vitiligo in Libya. *Int J Dermatol* 1985;24:233-5.
32. Partha P. Majumder. Genetics and Prevalence of Vitiligo Vulgaris. In: Seung - Kyung Hannm, James J. Nordlundm, editors. Vitiligo, a Monograph on the Basic and Clinical Science. Korea: USA; 2000. Pp. 18-22.
33. Behl PN, Kotia A, Sawal P. Vitiligo: Age-group related trigger factors and morphological variants. *Indian J Dermatol Venereol Leprol* 1994; 60:275-9.
34. Dave S, Thappa DM, DSouza M. Clinical predictors of outcome in vitiligo. *Indian J DermatolVenereolLeprol*2002; 68:323-5.
35. Sinha A. An immunological study of vitiligo. *Indian J DermatolVenereolLeprol* 1997; 63:91-4.
36. Gopal K, Rama Rao GR, Kumar YH, AppaRao MV, Vasudev PS. Vitiligo: A part of a systemic autoimmune process. *Indian J DermatolVenereolLeprol*2007; 73:1625.
37. Spritz RA. The genetics of generalized vitiligo and associated autoimmune diseases. *Pigment Cell Res* 2007; 20:271-27.
38. Gould IM. Vitiligo in diabetes mellitus. *Br J Dermatol* 1985; 113:153.
39. Cunliffe WJ, Hall R, Newell DJ, *et al.* Vitiligo, thyroid disease and autoimmunity. *Br J Dermatol* 1968; 80:135-139.
40. Koranne RV, Sehgal VN, Sachdeva KG. Association of Pemphigus Vulgaris with Vitiligo. *Indian J DermatolVenereolLeprol* 1986; 52:107-109.
41. Moradi S, Ghafarpour G. Thyroid dysfunction and thyroid antibodies in Iranian patients with vitiligo. *Indian J Dermatol*2008; 53:9-11.
42. Huggins RH, Janusz CA, Schwartz RA. Vitiligo: A sign of systemic disease. *Indian J DermatolVenereolLeprol*2006; 72:68-71.
43. Sehgal VN, Srivastava G. Vitiligo: Auto-immunity and immune responses. *Int J Dermatol* 2006; 45:583-90.
44. Kurtev A, Dourmishev AL. Thyroid function and auto-immunity in children and adolescents with vitiligo. *J EurAcadDermatolVenereol* 2004; 18:109-11.
45. Glovanni E, Orecchia, Alternative Therapies for Vitiligo. In: Kyunghann SK, Nordlund JJ editors. Vitiligo, A monograph on the basic and clinical science. Korea: USA; 2000; p. 227.
46. Banerjee AK, Banerjee DK, Chaudhury, DS. Serum vitamin B12 level in vitiligo: A preliminary study. *Bull Calcutta School Trop Med.* 1970;18:73-5.
47. Montes LF, Diaz ML, Lajous J, Garcia NJ. Folic acid and vitamin B12invitiligo: a nutritional approach. *Cutis* 1972;50:39-42.
48. Shajil EM, Agrawal D, Vagadia K, Marfatia YS, Begum R. Vitiligo: Clinical profiles in Vadodara, Gujarat. *Indian J Dermatol* 2006;51:100-4.
49. Shah H, Mehta A, Astik B. Clinical and sociodemographic study of vitiligo. *Indian J Dermatol Venereol Leprol* 2008;74:701.

Cite this article as: Krupa Shankar DS, Shashikala K, Madala R. Clinical patterns of vitiligo and its associated co morbidities: A prospective controlled cross-sectional study in South India. *Indian Dermatol Online J* 2012;3:114-8.

Source of Support: Nil, **Conflict of Interest:** None declared.