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Chlamydia Trachomatis Infection as a Trigger For Autoimmune Reaction In Vitiligo

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ABSTRACT
The exact aetiology of vitiligo is not known. Generalised vitiligo is thought to be an autoimmune disease. Although viral infection has been implicated, the exact trigger for initiating this autoimmune phenomenon is not known. Sera of 60 cases of vitiligo were tested for the presence of IgM, IgG and IgA antibodies to Chlamydia trachomatis by ELISA method. The results were compared with 30 healthy controls. Twelve (24.0%) of generalized and focal cases of vitiligo as against 2 (6.67%) controls were seropositive for IgM/IgG/IgA antibodies, a statistically significant difference (χ^2 3.91 P < 0.05). Odds Ratio was 4.42 and there is 2.48% to 32.12% higher chance of having seropositivity for C. trachomatis with confidence interval of 95% in cases of generalized and focal vitiligo as compared to healthy controls. None of the patients of segmental vitiligo was positive for antichlamydia antibodies. Above observation does suggest that C. trachomatis infection may be a trigger for initiation of autoimmunity in cases of generalized and focal vitiligo. Hence we recommend that all such cases of vitiligo and their spouses/sexual contacts should be investigated and treated with antichlamydial drugs. Absence of any association of C. trachomatis infection and segmental vitiligo supports its neurogenic origin.
Keywords: *Vitiligo, Chlamydia trachomatis, Segmental Vitiligo, Generalised Vitiligo, Focal Vitiligo*

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INTRODUCTION

The pathogenesis of vitiligo is still an enigma as it was decades ago. Genetic predisposition, self-destruct theory of defective melanocytes and autoimmunity causing destruction of these melanocytes are some of the possible mechanisms implicated. Clinical support for the immune hypothesis includes the presence of lymphocytes in the early lesion of vitiligo, presence of circulating auto antibodies in many patients (with or without autoimmune disease), and the association with halo nevus and autoimmune diseases in 10 to 15 percent compared to one percent of the population.[1-3] Trigger of this autoimmunity is not known. Autoimmune serum factors have also been reported in cases of lymphogranuloma venereum caused by L1, L2, L3 strains of *Chlamydia trachomatis*.[4] A number of females attending our out-patient as cases of vitiligo, also had associated chronic pelvic inflammatory disease. Since *Chlamydia trachomatis* is one of the most common causes of PID and it is known to initiate HSP-60 induced CD8+ T-cell mediated autoimmunity by molecular mimicry in Reiter's disease [5], we decided to look for evidence of *C. trachomatis* infection in patients of vitiligo by studying the presence of *C. trachomatis* antibodies in the sera of these patients.

MATERIAL AND METHODS

Sixty patients of vitiligo presenting in the dermatology out-patient department formed part of the study. The patients were classified into focal, if there was one or two isolated, asymmetrical lesions in a non segmental distribution and into segmental with depigmented lesions in a segmental distribution. All other cases were classified as generalized vitiligo. Further any case with appearance of a new lesion or with extension of an existing lesion during the last six months was considered as active. Informed consent of the patients was obtained and their sera were tested for the presence of IgG, IgM and IgA antibodies to *Chlamydia trachomatis* by ELISA method with the kits supplied by Vircell, S. I., Granada, Spain. The test had sensitivity of 96% for IgG, 90% for detection of IgM and 95% for IgA with specificity of 100% for both IgM and IgG and 98% for IgA. In the assay is used COMP (Complexes of Outer Membrane Proteins) of *C. trachomatis*, free of lipopolysaccharides.

30 healthy age and sex matched controls were also tested.

χ^2 test was used as a test of significance.

RESULTS

There were 60 cases of vitiligo, with an average age of 35.19 (SD 20.15) years. Thirty four (56.67%) of them were males and 26 (43.33%) were females. Average duration of disease was 6.43 (SD 7.06) years. Forty three (71.67%) were cases of generalized vitiligo, 7 (11.67%) focal vitiligo and 10 were segmental vitiligo. ELISA tests for *C. trachomatis* antibodies were compared with 30 healthy controls with an average age of 32.43 (SD 3.85) years, 20 (66.67%) of them being females and 10 (33.33%) males (Table). The difference between mean age of the cases and controls was not significant (Standard Error 2.69).

Table. *Chlamydia trachomatis* antibodies in cases of vitiligo

Group	Number	Positive (%)	IgM positive (%)	IgG positive (%)	IgA positive (%)
Generalized vitiligo	43	10 (23.26)	6 (13.95)	4 (9.30)	3 (6.98)
Focal vitiligo	7	2 (28.57)	2 (28.57)	0	0
Segmental vitiligo	10	0	0	0	0
Controls	30	2 (6.67)	2 (6.67)	0	1 (3.33)

Taking generalized and focal vitiligo as a group with autoimmunity as their pathogenesis, odds ratio (OR) was 4.42 and with confidence interval of 95%, there was 2.48 to 32.12% higher chance of *C. trachomatis* antibodies in patients of vitiligo as compared to healthy controls, the difference was also found statistically significant (χ^2 3.91, P <0.05). None of the ten cases of segmental vitiligo had antichlamydial antibodies. Further 8 (22.22%) of 36 patients with active vitiligo as compared with 4 (16.66%) cases with inactive vitiligo were positive for *C. trachomatis* antibodies. Although higher proportion of patients with active vitiligo were positive for *C. trachomatis* antibodies as compared to inactive cases, the difference was not found statistically significant.

DISCUSSION

Neural hypothesis in the pathogenesis of vitiligo especially in segmental variety was supported by absence of *C. trachomatis* antibodies in all our cases of segmental Vitiligo. [6] Autoimmune hypothesis has been proposed for generalized and acrofacial variety of Vitiligo. [1-3] The trigger for this autoimmune process is not known. Role of viral infection as a trigger has been proposed by Orotonne and Bose [7] in 1993, Akcan et al [8] inconclusively studied the role of hepatitis B virus and Toker et al [9] of CMV virus. Nimba et al [10] reported a case of HIV infection in a black patient with vitiligo, who improved with antiretroviral therapy. However, there has been no further progress in this direction. We have found significant association of *C. trachomatis* seropositivity and generalized vitiligo, there by suggesting that *C. trachomatis* may be acting as trigger of autoimmunity in certain cases of vitiligo.

Hence we suggest, that those with genetic predisposition, having similar molecular structure as *Chlamydia trachomatis*, develop autoimmune reaction when the primed T-cells cross react with melanocytes when they are exposed to the immune system due to any type of trauma. Trauma exposes the hidden antigens to the immune system, which will also explain the Koebner's phenomenon seen in vitiligo.

6.67% of our healthy controls also tested positive for IgM *C. trachomatis* antibodies. These may be cases with subclinical infection. Almost 60% of the cases of PID are subclinical. [11] Hence we recommend that pelvic inflammatory disease in females and chronic genitourinary infection in males should be looked in all cases of generalized/focal vitiligo. Those who have clinical or serological evidence of PID/genitourinary infection should be treated with antichlamydial drugs along with their sexual partners, to take away this trigger of autoimmunity.

REFERENCES

1. Nordlund JJ, Lerner AB. Vitiligo(1982): It is important. Arch Dermatol 118: 5-8.
2. Betterle C, Peserico A, Bersani(1979): G. Vitiligo and autoimmune polyendocrine deficiencies with autoantibodies to melanin-producing cells. Arch Dermatol 115: 364.
3. Naughton GK, Reggiardo D, Bystryn JC.(1986): Correlation between vitiligo antibodies and extent of depigmentation in vitiligo. J Am Acad Dermatol 15: 978-81.
4. Sonck CE, Rasanen JA, Mustakallio KK, Lassus A.(1973): Autoimmune serum factors in active and inactive lymphogranuloma venereum. Br J Vener Dis 49: 67-68

5. Dunlop EM. (1983): Venereal disease: *Chlamydia trachomatis* and *Ureaplasma urealyticum* in a patient with Reiter's disease. Br J Hosp Med 29: 6-11.
6. Orecchia GE. Neural pathogenesis. In: Hann SK, Nordlund JJ, editors. Vitiligo. London, Blackwell Science 2000; 142.
7. Ortonne JP, Bose SK. Vitiligo (1993): Where do we stand? Pigment Cell Res 6: 61-72.
8. Akcan Y, Kavak A, Sertbas Y, Olut AL, Korkut E, Bicik Z, Kisacik B.(2006): The low seropositivity of hepatitis B virus in Vitiligo patients. J Eur Acad Dermatol Venereol 20: 110-111.
9. Toker SC, Sarycaoglu H, Karadogan SK, Mistik R, Baskan EB, Tunaly S. (2007): Is there any relation between Vitiligo and cytomegalovirus? J Eur Acad Dermatol Venereol 21: 141-42.
10. Niamba P, Traoreʹ A, Taiʹeb A. (2007): Vitiligo in a black patient associated with HIV infection and repigmentation under antiretroviral therapy. Ann Dermatol Venereol 134: 272-273.
11. Westrom L and Eschenbach D. Pelvic inflammatory disease. In: Holmes KK, Mardh PA, Sparling PF, Lemon SM, Stamm WE, Piot P, Wasserheit JN editors. Sexually Transmitted Diseases, Thired. New York, McGraw-Hill 1999, 783-810.

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