

International Journal of Biomedical Research

ISSN: 0976-9633 (Online); 2455-0566 (Print)

Journal DOI: <https://doi.org/10.7439/ijbr>

CODEN: IJBRFA

Case Report**Vitiligo and its association with hypoacusis: A case control study****Sharmila Patil, Vasundhara Singh* and Shweta Agarwal***Department of Dermatology, Dr. D Y Patil Medical College Nerul, Navi-Mumbai, India***QR Code*****Correspondence Info:**

Dr. Vasundhara Singh
 Resident,
 Department of Dermatology,
 Dr. D Y Patil Medical College Nerul, Navi-Mumbai, India

Article History:*Received:** 17/09/2017**Revised:** 12/10/2017**Accepted:** 12/10/2017**DOI:** <https://doi.org/10.7439/ijbr.v8i10.4394>**Abstract**

Vitiligo is a pigmentary disorder with absence of functional melanocytes in the affected area. Melanocytes originate from neural crest and migrate to rest of the body in a cephalo-caudal manner in the embryonic life. Apart from epidermis and hair bulb in skin, they are also present in leptomeninges, inner ear, retina and uveal tract. In the inner ear melanocytes are present in endolymphatic sac, hair cells, vestibular organ and striavascularis.

Vitiligo is not just skin deep and is also responsible for subclinical hypoacusis in majority of patients. Patients suffering from vitiligo affecting the head and neck region at the time of origin are more prone to the auditory dysfunction. Leucotrichia is another association with asymptomatic sensori-neural hypoacusis. This is due to the loss of melanocytes which have a protective role in inner ear. TEOAE and Pure tone audiometry can be used effectively for screening patients and these patients should be informed regarding the noise exposure and ototoxic drugs.

Keywords: vitiligo, hypoacusis, sensorineural.**1. Introduction**

Vitiligo is an autoimmune disorder of hypopigmentation, characterised by localized and/or generalized depigmentation of the skin with or without mucous membrane involvement. Incidence of the disease is approximately 1-2% of the world population [1].

The disease is characterised by hypopigmented patches distributed over the body in either symmetric or asymmetric pattern.

The imperative of the disease is highlighted from the time of Vedas in our country.

Various studies have been performed to know to exact etio-pathogenesis of the disease. Multiple theories like genetic, autoimmune, neural and free radical damage etc have been proposed [2].

Human melanocytes are derived from neural crest cells and the damage to these cells are most evident on the skin, but melanocytes present in other organs like retina, uvea, leptomeninges and inner ear may also bear the brunt of this destruction[3-8].

In the inner ear melanocyte are believed to play a role in development and normal functioning of striavascularis which helps in formation of endolymph[7]. Association of various disorder of hypopigmentation with acoustic abnormalities like Wardenburg's syndrome, Albinism, Aleizandrini syndrome and Vogt-Koyanagi-Haradasyndrome are witness to the endocochlear electrophysical role of melanocytes [9,10]. Studies claim difference in levels of noise induced hypoacusis in white and black population due to the difference in melanin content[11,12,13].

Our study is a case control prospective study to determine the prevalence of sensorineural hearing loss in vitiligo patients.

2. Method

2.1 Inclusion criteria: Vitiligo patients with age and sex matched healthy controls

2.2 Exclusion criteria:

Hearing loss due to trauma/drugs/infection/surgery/Miniers disease
 Other autoimmune/ neurological/ metabolic disorder
 Systemic disease like hypertension/Diabetes mellitus
 No family history of hypoacusis

After gaining the approval of institutional ethics committee, a prospective case control study was undertaken in 50 vitiligo patients. Control consisted of healthy, age and sex match volunteers, from April 2016 to Jan 2017 at Dr D.Y. Patil Medical college and hospital, Nerul, Navi Mumbai.

A well-informed consent was taken from the study group, after a detailed discussion regarding the purpose of study and methodology. After enquiring about the onset of the disease, areas involved treatment and family history, a detailed dermatological examination was taken. Patient from dermatology OPD were referred to ENT department. The Audiologist was unaware of the patient group and carried out tuning fork tests, Pure tone audiometry, transient evoked otoacoustic emission (TEOAE). TEOAEs were recorded at and amplitude of 1,2,3 kHz, and reducibility percentage was recorded.

Average pure tone hearing threshold (APT-HT) were calculated in each patient. The degree of hearing loss was calculated using Clarks scale: minimal >16-25 dB;

mild > 25-40 dB; moderate > 40-55 Db; moderate to severe > 55-70 Db; severe > 70-90 Db and > 90 dB profound.

All data was statistically analysed using student’s T test. A p value of less than or equal to 0.05 was statistically significant.

3. Results

Table 1 shows gender and age distribution in vitiligopatients. Two groups of age and sex matched consisting of 50 each were taken. There were 21 males and 29 females with a ratio of 1:1.3.

Table 1: Gender and Age distribution details

Age of onset(years)	Male	Female	Total	Percentage
0-20	6	16	22	44
21-40	9	9	18	36
41-60	4	5	9	18
61-80	2	1	3	6
Total	21	29	50	100

Type of vitiligo with acoustic characteristic is given in Table 2. Conductive deafness was not observed in any patient. Hypoacusis in vitiligo group was 34% (17 patients) and in control group was 4% (2 patients). Unilateral sensorineural deafness was noted in 6 patients and bilateral in 11 patients. Family history was positive in 32% (16 patients) of vitiligo. Leukotrichia was positive in 34% (17 patients).

Table 2: Type of vitiligo with acoustic characteristic

Type of Vittigo	Hypoacusis Bilateral	Hypoacusis Right ear	Hypoacusis Left ear	Vitiligo with Hypoacusis	Vitiligo with Normal hearing	Frequency of vitiligo type	Percentage of hypoacusis
Vulgaris	6	2	-	8	10	18	44.44
Focal	2	-	2	4	2	6	66.66
Segmental	1	-	-	1	18	19	5.26
Universal	1	1	-	2	2	4	50
Acrofacial	1	-	1	2	1	3	66.66
Total	11	3	3	17	33	50	

The site of origin and association between hypoacusis is depicted in Table 3. Most common site of origin was mucosa (15 patients). Vitiligo of head and neck was strongly associated with hypoacusis(58.33%).

Table 3: Association between origin site and hypoacusis

Site of origin	With hypoacusis(17)	With normal hearing(33)	Frequency at origin site	Incidence at each site %
Head and neck	7	5	12	58.33
Upper limb	1	3	4	25
Lower limb	2	6	8	25
Mucosa	4	11	15	26.67
Trunk	3	8	11	27.27

Average pure tone hearing threshold in each ear of all patients is represented in Table 4.

Table 4: Average pure tone hearing threshold in each ear of all patients

Frequency	Right ear: Vitiligo Mean±SD	Right ear: Control Mean±SD	Left ear: Vitiligo Mean±SD	Left ear: Control Mean±SD	P value
500-2000 Hz	10.32±3.59	10.13±2.21	10.26±2.23	10.19±2.43	0.067
250-8000 Hz	16.79±5.36	10.52±2.29	15.93±6.17	9.93±6.10	0.039

A higher APT-HT was noted in patients suffering from vitiligo as compared to control group. This data was statistically significant with a p value of 0.039. TEOAE reproducibility was reduced in vitiligo patients(41.5%) as compared to normal patients(96%).

4. Discussion

Vitiligo is a pigmentary disorder with absence of functional melanocytes in the affected area. Melanocytes originate from neural crest and migrate to rest of the body in a cephalo-caudal manner in the embryonic life [14,15]. Apart from epidermis and hair bulb in skin, they are also present in leptomeninges, inner ear, retina and uveal tract. In the inner ear melanocytes are present in endolymphatic sac, hair cells, vestibular organ and striavascularis[16,17].

The variations of melanocyte-melanosome content in inner ear of individuals of various races and their correlation with their constitutional colour has been studied.

The semi-conductive property of melanin has brought to light its role in converting energy state into molecular vibration and rotation and its ability to response to phonic, electrical stimulation and acoustics[18]. Apart from these functions, melanocyte helps in normal development of striavascularis[9] and has angioprotective role in cochlea which maintains endolymphatics homeostasis and is crucial for hair cell survival[19].

In vitiligo a synchronous loss of melanin activity has been found in the affected area and inner ear. Loss of functional melanocytes increases the vulnerability of the inner ear to various damaging factors due to the absence of homeostasis and angioprotective function[20,21].

A study by Garber *et al* has noted increase incidence of auditory seizures, auditory fatigue and hearing loss in patients with reduced inner ear melanin content[22].

In our Study there was a female preponderance, with the Male to Female ratio being 1:1.3. This may be due to increased cosmetic concern of the latter group. A study reported by Hann *et al* showed comparable results (1:1.6)[23]. Maximum number of cases (44%) was noted in first two decades of life with gradually diminishing in geriatric population. Various studies have noted maximum incidence below 19-30 years of age[24,25].

The most common type of vitiligo observed in study cases was Segmental vitiligo (19 patients) followed by vulgaris(18 patients). Maximum hypoacusis was present in acrofacial and focal type of vitiligo and least in segmental type. In a study performed by Sharma *et al*, generalised vitiligo was associated maximum with hypoacusis as compared to other clinical variants [4,26].

In our study 34% of vitiligo patients were suffering from subclinical sensorineural hypoacusis. Incidence of hypoacusis in control group was 2% which was sensorineural type. Conductive hearing loss was not found in any patient group. The incidence of sensorineural hearing loss in patients suffering from vitiligo has been reported as 16%,18.89%,68% and 38%[10,4,27,28]. Unilateral involvement was noted in 6 patients(35.2%) whereas bilateral was noted in 11 patients(68.6%). Researchers have reported 25% and 75%, and 36.9% and 63.1% respectively [29,30].

The most common site of origin encountered in our study was mucosa (30%). Vitiligo originating from head and neck was most closely associated with hypoacusis(58.3%). We noticed that 90% of patients with hypoacusis had leucotrichia. Leucotrichia was present in 34% of vitiligo patients as compared to 44.3% of patients noted in a study by Akay *et al*[10]. Family history positivity was seen in 32%, others have found it in 37.5% and 27.5% respectively.

Our study shows a statistically significant(p value=0.039), APT-HT value(250-8000 Hz) in vitiligo patients as compared to the control group. However in frequencies of 500-200 Hz APT-HT was statistically not significant. Fleissig *et al* and Sharifian *et al*[28,29] compounded similar observation in term of APT-HT in their study. Thus we can say that the relative sparing of speech frequency in vitiligo patients explains their asymptomatic hypoacusis.

The TEOAE reproducibility % was 41.5% and 96% in vitiligo and normal patients respectively which was similar to reproducibility % reduction noted by Shalaby *et al*(33.3%) and Bassiouny *et al*(49%). This observation indicates a loss of cochlear emission in vitiligo patients[27,31,32].

5. Conclusion

Vitiligo is not just skin deep and is also responsible for subclinical hypoacusis in majority of patients. Patients suffering from vitiligo affecting the head and neck region at the time of origin are more prone to the auditory dysfunction. Leucotrichia is another association with asymptomatic sensori-neural hypoacusis. This is due to the loss of melanocytes which have a protective role in inner ear. TEOAE and Pure tone audiometry can be used effectively for screening patients and these patients should be informed regarding the noise exposure and ototoxic drugs.

References

- [1]. Sun XK, Xu AE, Meng W, Wei XD, Jiang ZM, Yan XF et al. Study on genetic epidemiology in 815 patients with vitiligo in Zhejiang area. *J Eur Acad Dermatol.* 2005; 26(11):911-4.
- [2]. 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; 16:208-214.
- [3]. Mills MD, Albert DM. Ocular and otic findings in vitiligo. In: Hann SK, Nordlund JJ, eds. Oxford: Blackwell Science Ltd.; 2000:81-8.
- [4]. Sharma L, Bhawan R, Jain RK. Hypoacusis in vitiligo. *Indian J Dermatol Venereol Leprol.* 2004; 70:162-4.
- [5]. Cowan CL Jr, Halder RM, Grimes PE. Ocular disturbances in vitiligo. *J Am Acad Dermatol.* 1986; 15:17-24.
- [6]. Ozuer MZ, Sahiner T, Aktan S. Auditory evoked potentials in vitiligo patients. *Scand Audiol.* 1998; 27:255-8.
- [7]. Ortonne JP, Bose SK. Vitiligo: where do we stand? *Pigment Cell Res.* 1993; 6:61-72.
- [8]. Hann S-K, Nordlund JJ. Definition of Vitiligo. In: Hann S-K, Nordlund JJ eds. Vitiligo: A monograph on the basic and clinical science. Blackwell Science, Oxford, 2000:3-5.
- [9]. Steel KP, Barkway C. Another role for melanocytes: their importance for normal striavascularis development in the mammalian inner ear. *Development.* 1989; 107:453-63.
- [10]. Akay BN, Bozkir M, Anadolu Y, Gullu S. Epidemiology of vitiligo, associated autoimmune diseases and audiological abnormalities: Ankara study of 80 patients in Turkey. *J Eur Acad. Dermatol Venereol.* 2010; 24:1144-50.
- [11]. Karsai LK, Bergman M, Choo YB. Hearing in ethnically different longshoremen. *Arch Otolaryngol.* 1972; 96:499-504.
- [12]. Ortonne JP, Bahadoran P, Fitzpatrick TB, Mosher DB, Hori Y. Hypomelanoses and hypermelanosis. In : Freedman IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI, editors. Fitzpatrick's Dermatology in General Medicine. McGraw Hill: New York; 2003. p. 836-81.
- [13]. Aslan S, Serarslan G, Teksoz E, Dagli S. Audiological and transient evoked otoacoustic emission findings in patients with vitiligo. *Otolaryngol Head Neck Surg.* 2010; 142:409-14.
- [14]. Halaban R, Heberi DN. Biology of melanin. In: Fitzpatrick's Dermatology in General Medicine. Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI, eds. 6th edn. New York: McGraw-Hill, 2003:127-48.
- [15]. Goldgeier MH, Klein LE, Klein-Angerer S, Moellmann G, Nordlund JJ. The distribution of melanocyte in the leptomeninges of the human brain. *J Invest Dermatol.* 1984; 82(3):235-8.
- [16]. Spritz RA. The genetics of generalized vitiligo and associated autoimmune diseases. *J ematol Sci.* 2006; 41:3-10.
- [17]. Barrenas ML, Axelsson A. The development of melanin in the stria vascularis of the gerbil. *Acta Otolaryngol.* 1992; 112:50-8.
- [18]. McGinness J, Corry P, Proctor P. Amorphous semiconductor switching in melanins. *Science* 1974; 183(4127):853-5.
- [19]. Conlee JW, Parks TN, Creel DJ. Reduced neuronal size and dendritic length in the medial superior olivary nucleus of albino rabbits. *Brain Res.* 1986; 363:28-37.
- [20]. LaFerriere KA, Kaufman-Arenberg I, Hawkins JE, Johnsson LG. Melanocytes of the vestibular labyrinth and their relationship to microvasculature. *Ann Otol Rhinol Laryngol.* 1974; 83:685-94.
- [21]. Hilding D, Ginzberg R. Pigmentation of the stria vascularis. *Acta Otolaryngol.* 1977; 84:24-37.
- [22]. Garber SR, Turner CW, Creel DJ, Witkop CJ. Auditory system abnormalities in human albinos. *Ear Hear.* 1982; 3:207-11.
- [23]. Hann SK, Chun WH, Park YK. Clinical characteristics of progressive vitiligo. *Int J Dermatol.* 1997; 36:353-355.
- [24]. Handa S, Kaur I. Vitiligo: clinical findings in 1436 patients. *J Dermatol.* 1999; 26:1295-7.
- [25]. Arican O, Koc K, Kutluk R, Ersoy L. Vitiligoluhastalarda serum vitamin B12 vefolikasitduzeyleri. *Turkiye Klinikleri Dermatol.* 2003; 13:4-10.
- [26]. Hong CK, Lee MH, Jeong KH, Cha CI, Yeo SG: Clinical analysis of hearing levels in vitiligo patients. *Eur J Dermatol.* 2009; 19:50-56.
- [27]. El-SayedShalaby M, El-Zarea GA, Nassar AI. Auditory Function In Vitiligo Patients. *Egyptian Dermatology Online Journal.* 2006; 2(1):7.
- [28]. Fleissig E, Gross M, Ophir I, Elidan J, Bdolah-Abram T, Ingber A. Risk of Sensorineural Hearing Loss in Patients with Vitiligo. *Audiol Neurotol.* 2013; 18:240-6.
- [29]. Ayodogan K, Turan OF, Onart S, Karadogan SK, Tunali S: audiological abnormalities in patients with vitiligo. *Clin Exp Dermatol* 2006; 31:110-3.
- [30]. Sharifian MR, Maleki M, Honarvar H. The correlation between vitiligo and hearing loss. *Iranian J Otorhinolaryngol.* 2006; 17(42):3-7.
- [31]. Bassiouny A, Farid S, El Khoust M. Hearing abnormalities in vitiligo. Egypt. *J Otolaryngol.* 1998; 15(1):51-60.
- [32]. Schrott A, Spoendlin H. Pigment anomaly associated inner ear deafness. *Acta Otolaryngol(Stockh).* 1987; 103:451-7.