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MAIN ARTICLE

Is there a relationship between premature hair greying and hearing impairment?

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Abstract

Objective: There is evidence for a strong correlation between low bone mineral density and hearing loss. Furthermore, premature hair greying has been associated with low bone mineral density. Hence, this study aimed to investigate, for the first time, the relationship between premature hair greying and hearing impairment.

Methods: Fifty patients with premature hair greying (20 women and 30 men), aged under 40 years (mean, 30.1 ± 4.9 years), who had onset of hair greying in their twenties, were recruited, along with 45 age- and sex-matched healthy control subjects (17 women and 28 men; mean age, 28.7 ± 5.1 years). Each participant was tested with low frequency audiometry at 0.125 to 2 kHz, high frequency audiometry at 4 to 8 kHz, and extended high frequency audiometry at 9 to 20 kHz.

Results: Hearing thresholds were similar at all frequencies from 0.25 to 4 kHz ($p > 0.05$); however, significant hearing loss was observed at all frequencies from 8 to 20 kHz in the premature hair greying group compared with the control group ($p < 0.05$).

Conclusion: Patients with premature hair greying had hearing impairment at extended high frequencies. Premature hair greying may be an important risk factor for hearing loss.

Key words: Aging; Premature; Hearing Loss; Hearing Loss, High Frequency

Introduction

The colour of human hair depends on melanogenesis, the process of synthesis of melanin and its subsequent distribution from the melanocytes to keratinocytes. The process is thought to be controlled genetically at diverse levels. The human hair follicles include two types of melanin: black-brown pigment eumelanin, mainly present in black and brown hair, and yellow or red pheomelanin, found in blonde and auburn hair.[1] Premature hair greying, defined as having almost all hair grey by age 40 years, is a risk marker for a variety of chronic conditions.[2,3]

The aetiology of greying is incompletely understood. Currently, it is thought to be mainly genetic, with interplay of diverse environmental factors. It may also occur in association with certain organ-specific autoimmune disorders, such as pernicious anaemia, hyperthyroidism or hypothyroidism, and as part of various premature ageing syndromes, including progeria and pangeria, and atopic diathesis.[4,5] Premature hair greying has also been associated with low bone mineral density.[3,6]

Hearing impairment is the most common sensory defect, and its possible aetiologies consist of a broad range of inherited causes (e.g. monogenic and polygenic) to environmental causes (e.g. infections, drugs and noise).[7] Hearing loss is classified according to aetiology, onset, course, side, type, frequency and severity.[7]

There are a number of studies showing a strong correlation between low bone mineral density and hearing loss.[8–10] Because premature hair greying is associated with low bone mineral density in some studies, we aimed to investigate the relationship between premature hair greying and hearing impairment. This is, to our knowledge, the first published study evaluating this relationship.

Materials and methods

Subject selection

We recruited 50 patients (20 women and 30 men) who had premature hair greying affecting almost all their hair, who were aged under 40 years and who had onset of hair greying in their twenties (Figure 1). All the participants were subjected to careful ear examination to identify any abnormalities that may interfere with hearing, such as a perforated tympanic membrane

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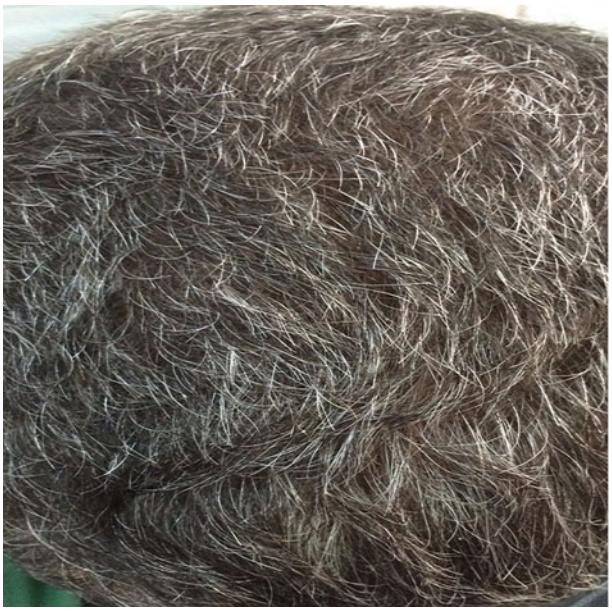

FIG. 1
Premature hair greying in one of the patients.

or other middle-ear pathologies. We also recruited 45 age- and sex-matched healthy control subjects (17 women and 28 men) who were aged less than 40 years.

Exclusion criteria were: tinnitus, middle-ear disease, diabetes mellitus, family history of hearing loss, history of acoustic trauma, conductive hearing loss, exposure to ototoxic substances, occupational noise exposure, autoimmune diseases, history of smoking, ongoing infection or inflammation, and being on any medication.

Hearing assessment

Each participant was tested with low frequency audiometry at 0.125 to 2 kHz, high frequency audiometry at 4 to 8 kHz, and extended high frequency audiometry at 9 to 20 kHz. All subjects had normal immittance audiometry results. This assessment was conducted in the audiology unit of the Evliya Celebi Education and Research Hospital at Dumlupınar University by the same expert audiologist, who was blinded to the study and clinician.

Ethical considerations

The study protocol was approved by the ethics committee of Pamukkale University, Denizli, Turkey. All subjects included in the study provided informed oral and written consent. Subjects were enrolled in the study only after they agreed to participate in the study and had signed an informed consent form.

Statistical analysis

Statistical analysis was conducted using SPSS software, version 19 (SPSS, Chicago, Illinois, USA). Normality was assessed using a Shapiro–Wilk test. For normally distributed values, descriptive results are expressed as mean ± standard deviation (SD). The independent-samples *t*-test and Kruskal–Wallis test were used to examine differences between groups. Statistical significance was defined as $p < 0.05$.

Results

Following a through clinical examination, and otological and audiometric evaluation, 50 patients with premature hair greying and 45 healthy controls were included in the final analysis. Mean age (± SD) was 30.1 ± 4.9 years in the premature hair greying group and 28.7 ± 5.1 years in the control group. There were no significant differences between the two groups in terms of mean age or ratio of males to females (Table I).

The hearing thresholds of the two groups of participants for the left and right ears are shown in Table II. Although the hearing thresholds of the groups were similar at all frequencies from 0.25 to 4 kHz ($p > 0.05$), significant hearing loss was observed at all frequencies from 8 to 20 kHz in the premature hair greying group compared with the controls ($p < 0.05$) (Table II).

Discussion

Premature hair greying has been described as a risk marker for a variety of chronic conditions, notably various disorders of the endocrine system. The actual pathophysiology of melanin depletion in hair follicles is unknown, although it has been shown that this trait is genetically determined, as is the acquisition of bone mass.[3] It is reasonable, therefore, to hypothesise that premature hair greying might be a marker for a variety of genetic and non-genetic conditions, such as myocardial infarction,[11] congestive heart failure, cancer, stroke, pneumonia and bronchitis, cirrhosis of the liver, gastrointestinal problems, or premature mortality.[2]

The key to the significance of premature hair greying, if any, may come with further understanding of the pathophysiology of melanin loss within the hair follicle itself. One study has shown that 55 per cent of patients with pernicious anaemia were found to develop greying hair before 50 years old as compared with only 30 per cent in the control group.[12] Reversible hypopigmentation of hair has also been noted in association with nutritional deficiencies, such as chronic protein loss (due to kwashiorkor, nephrosis, celiac disease and other causes of malabsorption), severe iron deficiency and copper deficiency.[4]

TABLE I
MEAN AGE AND SEX RATIO OF PREMATURE HAIR GREYING GROUP AND CONTROL GROUP

Variable	Study group	Control group	*p*
Age (mean ± SD; years)	30.1 ± 4.9	28.7 ± 5.1	0.195
Male/female (*n*)	30/20	28/17	0.982

SD = standard deviation

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TABLE II
HEARING THRESHOLDS IN PREMATURE HAIR GREYING GROUP AND CONTROL GROUP

Frequency (kHz)	Ear	Study group* (mean ± SD)	Control group† (mean ± SD)	*p*
0.25	Right	10.0 ± 4.1	9.7 ± 4.3	0.798
	Left	10.1 ± 4.5	10.9 ± 4.4	0.39
0.5	Right	8.1 ± 3.1	8.7 ± 3.4	0.394
	Left	8.9 ± 3.9	7.4 ± 3.3	0.056
1	Right	9.5 ± 4.1	9.9 ± 4.8	0.671
	Left	11.3 ± 7.5	9.0 ± 4.5	0.078
2	Right	10.7 ± 4.4	10.4 ± 4.4	0.778
	Left	11.9 ± 4.3	10.8 ± 4.8	0.229
4	Right	14.9 ± 5.2	13.2 ± 5.7	0.135
	Left	15.4 ± 6.1	13.7 ± 6.9	0.197
8	Right	46.4 ± 17.7	28.3 ± 12.9	<0.001
	Left	49.0 ± 17.1	29.1 ± 13.7	<0.001
10	Right	51.7 ± 12.9	34.7 ± 12.9	<0.001
	Left	52.3 ± 16.2	34.4 ± 13.6	<0.001
12	Right	57.4 ± 19.1	40.1 ± 15.5	<0.001
	Left	56.9 ± 20.2	37.9 ± 16.2	<0.001
14	Right	69.4 ± 20.9	50.1 ± 16.5	<0.001
	Left	71.7 ± 20.6	51.6 ± 16.7	<0.001
16	Right	86.9 ± 16.3	75.4 ± 23.1	0.007
	Left	89.6 ± 19.5	71.6 ± 20.9	<0.001
18	Right	101.5 ± 11.4	94.7 ± 13.3	0.009
	Left	103.1 ± 8.6	93.9 ± 14.4	<0.001
20	Right	107.2 ± 5.3	103.8 ± 6.3	0.005
	Left	107.7 ± 6.2	103.3 ± 6.7	0.001

*$n = 50$; †$n = 45$. SD = standard deviation

It has also been hypothesised that premature hair greying is associated with osteopenia.[3]

Orr-Walker *et al*. found that in a population of normal post-menopausal women screened to exclude diseases and medications known to influence bone metabolism, the majority of hair greying before the age of 40 years was associated with a lower bone mineral density at most skeletal sites.[6] They suggested that premature hair greying may be linked to genetic factors that influence bone mineral density. Alternatively, the processes that led to loss of scalp melanin production might have an impact on bone turnover.

Rosen *et al*. also investigated the relationship between premature hair greying and osteopenia in 36 men and women with osteopenia, comparing the findings with 27 men and women without osteopenia.[3] They concluded that those with premature hair greying but no other identifiable risk factors were 4.4 times more likely to have osteopenia than people without premature hair greying. They also noted that the association between premature hair greying and low bone mass could be related to genes that control peak bone mass or to factors that regulate bone turnover. Premature hair greying may therefore be an important risk marker for osteopenia.

A number of studies have investigated the relationship between low bone mineral density and hearing loss. Osteoporosis is a common metabolic disorder that causes progressive changes in bone structure. Metabolic changes and possible degeneration of middle-ear ossicles or the cochlear capsule may cause hearing loss in patients with osteoporosis. Kahveci *et al*. investigated the relationship between osteoporosis and hearing loss, and found that patients with osteoporosis showed a higher incidence of the sensorineural type of hearing loss.[8] Mendy *et al*. suggested that low bone mineral density was associated with balance and hearing impairment.[9] Monsell investigated the mechanism of hearing loss in Paget's disease of bone.[13] The findings supported the existence of a general, underlying cochlear mechanism of pagetic hearing loss that was closely related to loss of bone mineral density in the cochlear capsule. Swinnen *et al*. investigated the association between bone mineral density and hearing loss in osteogenesis imperfecta.[14] They hypothesised that osteogenesis imperfecta patients with lower bone mineral density might be more sensitive to aggregating microfractures, which may interfere with the bone remodelling inhibition pathways in the temporal bone, and therefore contribute to stapes footplate fixation and a conductive hearing loss component. Overall, these studies have similar findings, supporting an association between low bone mineral density and hearing impairment.

Different studies in the literature have shown that on the one hand premature hair greying is associated with low bone mineral density, and on the other hand low bone mineral density is associated with hearing loss. We therefore decided to investigate the hypothesis that there is a relationship between premature hair greying and hearing loss. Our study findings showed that hearing impairment at extended high frequencies was significantly greater in those with premature hair greying compared with a control group.

- **In this study, patients with premature hair greying had hearing impairment at extended high frequencies**
- **Premature hair greying may be an important risk factor for hearing loss**

There are several studies that have used extended high frequency audiometry to evaluate early hearing damage. Sulaiman *et al*. used extended high frequency audiometry to investigate early hearing damage in users of personal listening devices, and showed the presence of an early stage of hearing damage.[15] Kucur *et al*. evaluated hearing impairment in patients with polycystic ovary syndrome using extended high frequency audiometry, and claimed that hearing loss at high frequencies was more susceptible to vascular damage caused by the disease.[16] They also noted that extended high frequency audiometry was more effective than pure tone audiometry in identifying early hearing loss. They found more cases of hearing loss at high frequencies in patients with polycystic ovary syndrome compared with a control group. Patients with secretory otitis media were evaluated

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using extended high frequency audiometry by Sharma *et al*., who found pronounced impairment at extended high frequencies in cases of middle-ear pathologies.[17] Because early hearing damage can be detected by means of extended high frequency audiometry, we too evaluated the hearing of people with premature hair greying and a control group using extended high frequency audiometry, together with pure tone audiometry.

Conclusion

In conclusion, our study, which is the first to investigate the relationship between premature hair greying and hearing impairment, found that patients with premature hair greying had hearing impairment at extended high frequencies. Premature hair greying may be an important risk factor for hearing loss. Further studies are needed to help elucidate the mechanism behind hearing impairment associated with premature hair greying and to determine whether the impairment of extended high frequencies in these cases is progressive. It might be possible to prevent progression of hearing impairment by revealing the underlying factors.

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