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Original article

ASSOCIATION BETWEEN PREMATURE SCALP HAIR GRAYING AND SUBSTANCE ABUSE AMONG THE BENGALEE HINDU YOUNG ADULTS OF KOLKATA, INDIA: A BRIEF REPORT

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ABSTRACT

Background: Premature graying of hair (PGH) is one of the common public health problems worldwide. The factors include genetic, stress, autoimmune disorder, vitamin deficiency and smoking. The last one that is substance abuse is relatively less studied or very few and far between. Objectives: The present study was therefore undertaken to study the association of PGH with substance abuse as no such studies found in this part of the world. Sample and Methods: The data consisted of 116 cases (PGH+) of males and 134 controlled (PGH-) males and 119 cases of females with 131 controlled females (total 250) with age range between 20 and 30 years from the Bengalee Hindus of the urban areas of Northern fringe of Kolkata, India. Subjects were graded as 1-5 scale for the cases depending on the presence of premature gray hair. Subjects with smoking history of more than 3 pack-years as smokers and alcohol intake more than three times a week as drinkers were selected (Jo SJ et al, 2012). Results: Among males with regular smoking showed an odds ratio of 5.4586 indicating that the odds the developing the risk is over 5 times higher among cases (PGH+) than controls (PGH-). Similarly among females, the odds ratio of 3.1837 indicating the odds of developing the risk is 3 times higher among PGH+ than PGH-. Among males with regular alcohol consumption showed an odds ratio of 6.4286 indicating that the odds the developing the risk is over 6 times higher among PGH+ than their counterparts. Among females, the odds ratio of 2.5750 indicating that the odds the developing the risk is 2.5 times higher among cases than controls. *Conclusion*: The association of PGH with substance abuse seems to be lifestyle disorder and has been reported in the present study, irrespective of sex.

Key words: Premature Graying, Scalp hair, Smoking, Alcohol, Bengalee, Asian Indians

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INTRODUCTION

Graying is one of the ubiquitous signs of physiological ageing (Trüeb, 2006) which suggest an age-related exhaustion of the genetically regulated pigmentary potential of each individual hair follicle (Tobin et al., 2001). But, on the other hand, premature graying of hair (PGH) with no clear etiology (Naieni et al, 2012) is considered as a sign of rapid progressing old age, ill health and often leads to loss of self-esteem (Pandhi and Khanna, 2013). This physical phenomenon has a common dermatological entity (Naieni et al, 2012) and it also has major psychosocial and socioeconomic impact (Pandhi and Khanna, 2013). There is no specific definition of premature graving of hair, but researchers tried to define the phenomenon since many years. Gould, et al. (1978) and Glasser (1991) defined PGH as if 50% of the scalp hair is gray before the age of 50. On the other hand, others considered if all or most of one individual's hair is gray before the age of 40 is considered as PGH (Morton et al, 2007). Trueb (2006) adopted the threshold of PGH to be at the age of 20 in Caucasian, and among Africans at the age of 30, but he did not specify the percentage of gray hair. Cichorek et al (2013) proposed that after the age of 30, for every decade in pigment-producing epidermal melanocytes there is a decrease of 10-20% and this decrease in pigment production is one of the proposed mechanisms of hair graving.

Pathogenesis of PGH is not clear (Kocaman et al, 2012), but it had been stated by Acer et al. (2020) that the oxidative (Seiberg, 2013, Akin et al, 2016) and emotional stress (Akin et al, 2016) may play a significant role in pathogenesis of PGH. Moreover, it is associated with various systemic diseases (Kocamon et al, 2012, Agarwal et al. 2020). Premature graying of hair is subjected to be associated with so many factors like low serum ferritin, Vitamin B12, and HDL-C levels (Chakrabarty et al., 2016) and also with low serum copper concentration (Naieni et al, 2012). More importantly different environmental factors like lifestyle practices also play important role in PGH. Substance consumption is one those factors that are closely associated with the occurrence of PGH.

Apart being a risk factor for much preventable morbidity, including cerebrovascular, pulmonary disease, malignancy, and oral mucocutaneous disease, more recently, the role of smoking in hair loss and skin aging has been a great topic of interest and research (Mahendiratta et al., 2020, Trüeb 2006). Belli et al. (2016) in a study found that, the environmental factors,

including tobacco smoke exposure in mice can lead to premature graying of hair (PGH) as well as alopecia. The nicotine in tobacco smoke are accumulated in hair, and thus become reliable biomarkers for determining smoke exposure (Belli et al. 2016). Smoking also causes a relative hypo-estrogen state in women via hydroxylation of estradiol and inhibition of aromatase, and thus it is potentially affecting androgen dependent hair patterns (Skurnik and Shoenfeld, 1998). Many more additional studies have also suggested the association between smoking and PGH (Mosley and Gibbs 1996, Mahendiratta et al. 2020). The melanosomes in the melanocytes in gray hair are found within vacuoles or autophagolysosomes causing the defect. In addition, oxidative stress causes the hair bulbs tend to be increasingly vacuolated and this causes increase in reactive oxygen species (ROS) and oxidative stress around the follicle which leads to the damage to melanocytes and decrease the melanin production (Trüeb, 2006). Alcohol abuse is also a known contributor to oxidative stress and thus significantly associated with the occurrence of PGH (Belli et al., 2016).

In view of the previous studies the present study was an attempt to understand the association between PGH and substance abuse among Bengali Hindu population in West Bengal, India.

SAMPLE AND METHODS

The present study is a cross sectional case control study having information of substance consumption with PGH consisted data of 116 cases of males and 134 controlled males (total 250 males) and 119 cases of females with 131 controlled females (total 250 females) with age range between 20 and 30 years (mean age 25.6 years SD \pm 3.9) from the Bengalee Hindus of the urban areas of North 24 pgs, West Bengal, India. Participants were graded as 1-5 scale for the cases depending on the presence of gray hair, Grade 1 (less than 20% of total hair), grade 2 (20~40%), grade 3 (40~60%), grade 4 (60~80%), and grade 5 (more than 80%) (Jo et al, 2012). Subjects with a smoking history of more than 3 pack-years were considered as smokers and regarding alcohol abuse, those who drank alcohol more than three times a week were regarded as drinkers (Jo et al, 2012). The participants of the present study are mostly students, in service, and some of them are homemakers (especially females).

RESULTS

The association of premature graying of hair (PGH) with substance abuse which include smoking and alcohol consumption and shown are given in Table1 and 2 respectively. Associations between individuals with and without PGH by smoking status are shown in Table 1a and 1b respectively for males and females. Among the males the differences between individuals with and without PGH by smoking status showed significant difference ($\chi^2 = 38.11$, p < 0.00001) with an odds ratio of 5.4586 indicating that the odds the developing the risk is over 5 times higher among PGH+ than PGH-. Similarly among females the difference ($\chi^2 = 19.76$, p < 0.00001) with an odds ratio of 3.183 indicating that the odds the developing the risk is 3 times higher among PGH+ than PGH-.

Males	PGH+	PGH-	Total	Chi-square test
Smoker	90	52	142	38.11
Non smoker	26	82	108	p<0.00001
Total	116	134	250	-

Table 1a: Association of PGH with smoking among males

Odds Ratio = 5.4586; 95% CI = 3.1248 to 9.5354; z-statistics = 5.963; p<0.0001

Table 1b: Association of PGH with smoking among females

Females	PGH+	PGH-	Total	Chi-square test
Smoker	78	49	127	19.76
Non smoker	41	82	123	p<0.00001
Total	119	131	250	

Odds Ratio = 3.1837; 95% CI = 1.8968 to 5.3437; z-statistics = 4.383; p<0.0001

Associations between individuals with and without PGH by alcohol consumption are shown in Table 2a and 2.b respectively for males and females. Among the males the differences between individuals with and without PGH by alcohol drinking showed significant difference ($\chi^2 = 46.19$, p < 0.00001) with an odds ratio of 6.4286 indicating that the odds the developing the risk is over 6 times higher among PGH+ than PGH-. Similarly among females the differences between individuals with and without PGH by alcohol drinking showed significant differences ($\chi^2 = 11.47$, p < 0.0007) with an odds ratio of 2.5750 indicating that the odds the developing the risk is 2.5 times higher among PGH+ than PGH-.

Males	PGH+	PGH-	Total	Chi-square test
Drinker	88	44	132	46.19
Non drinker	28	90	118	p<0.00001
Total	116	134	250	

Table 2a: Association of PGH with alcohol among males

Odds Ratio = 6.4286; 95% CI = 3.6811 to 11.2266; z-statistics = 6.541; p<0.0001

Females	PGH+	PGH-	Total	Chi-square test
Drinker	49	28	77	11.47
Non drinker	70	103	173	p<0.0007
Total	119	131	250	_

Odds Ratio = 2.5750; 95% CI = 1.4786 to 4.4845; z-statistics = 3.342 p<0.0001

DISCUSSION

The prooxidant effect of smoking on the body increases the reactive oxygen species (ROS) damage to hair follicle melanocytes (Mosley and Gibbs, 1996, Trüeb, 2003, Jo et al., 2012,

Zayed et al, 2013) which leads to PGH. Alcohol abuse is also a known contributor to oxidative stress and thus significantly associated with the occurrence of PGH (Belli et at., 2016). Consumption of alcohol [in case of males significant difference ($\chi^2 = 46.19$, p < 0.00001) with an odds ratio of 6.4286 and in case of females significant difference ($\chi^2 = 11.47$, p < 0.0007) with an odds ratio of 2.5750] and smoking (in case of males significant difference [($\chi^2 = 38.11$, p < 0.00001) with an odds ratio of 5.4586 and in case of females significant difference ($\chi^2 =$ 19.76, p < 0.00001) with an odds ratio of 3.183)] was reported to be significantly associated with PGH in the present study as evident in other studies in other parts of the world [Nath et al. (2020), Triwongwaranat et al. (2019), Chakrabarty et al. (2016), Akin et al. (2016), Yeo et al. (2014), Sabharwal et al. (2014), Gatherwright et al (2013), Su and Chen (2007), Severi et al. (2003)] among both the sexes. Compared to controls, the smoker groups usually show an earlier onset and higher prevalence of hair graying [Sharma and Dogra (2018), Aggarwal et al. (2015), Sabharwal et al. (2014), Zayed et al. (2013)]. An individual with a positive smoking history also show increased risk of hair graying each year of continued smoking (Jo et al., 2012), but this correlation was not found to be true among smokers below age of 21-year old, mainly because of pressure of study and shorter duration of smoking [Belli et al. (2019), Acer et al. (2020), Gould et al. (1978)].

Regarding alcohol drinking, the result of this study is contradictory to that obtained in the study conducted by Kansal et al (2021) which showed that there is no significant difference between individuals with PGH and without PGH as the subjects of the studied population were below the age of 21 years. As the consumption of smoking and alcohol has significant relationships with CAD [Jalali et al. (2021), Hobden et al., 2020, O'Keefe et al. (2018)], osteoporosis (Yang et al., 2021), and so many other lifestyle diseases and also have significantly higher association with PGH as well, it can be said that PGH can be considered as a risk marker for those lifestyle diseases among both the sexes.

As the questionnaire is self-administered, there might be a possibility of recall bias. Moreover, the present study shows the associations and relationships of the risk factors that are attributed with PGH but without any proven causal relationship. The generalizability of the observations is limited.

CONCLUSION

Young adults with regular substance abuse are more susceptible to premature graying of human scalp hair. This also indicates an early sign of ageing and other associated health hazards. Necessary steps are required for proper screening and awareness to overcome this public health burden.

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