INTRODUCTION

Hair graying, one of the prototypical signs of human aging, is caused by a progressive loss of pigmentation from growing hair shafts. In normal aging, the onset of hair graying occurs at 34 ± 9.6 years of age in Caucasians and 43.9 ± 10.3 years in African Americans. Hair graying represents an impaired ability of melanocytes to maintain normal homeostasis and replenish melanin, pigment for the newly growing hair. Whether hair graying, early or otherwise, is a risk factor/predictor for osteopenia is controversial. Previous studies have shown a correlation of early hair graying with osteopenia, indicating that premature graying could serve as an early marker of osteopenia. However, some studies showed that there is no correlation between the two. We conducted a case–control study to compare the degree of osteopenia in healthy individuals with this condition.

A gradual loss of bone mass occurs with aging leading to osteopenia and osteoporosis. The diagnostic difference between osteopenia and osteoporosis is based on the measure of bone mineral density (BMD). Osteopenia and osteoporosis markedly increase the risk of skeletal fractures.
premature graying of hair (PGH) and those without PGH.

SUBJECTS AND METHODS

We conducted an observational case–control study (from February 2018 to April 2019) and a total of 132 normal individuals, who accompanied the patients attending the outpatient department of Department of Dermatology, Era’s Lucknow Medical College and Hospital, were enrolled. We included adults of either sex in the age group of 18–30 years and willing to participate in the study. The participants were divided into two groups: cases (n = 82) and controls (n = 50). Individuals who had graying of hair were included in the case group. The control group included age-, sex-, and bone mass index (BMI)-matched individuals without graying of hair. The exclusion criteria were all individuals with recent or old fracture, chronic debilitating disease, malabsorption syndrome, gross malnutrition, arthritis, hormonal disorders, neurologic disorders, on long-term treatment with systemic corticosteroids, chloroquine, hormone replacement therapy >6 months, calcitonin, bisphosphonates, antiepileptics, and psychiatry drugs.

A history of age of onset of PGH, intake of vegetarian and nonvegetarian food, amount of milk/day, smoking, and back pain was taken from all individuals. Family history of PGH was also taken. All the individuals were examined for PGH, and hair whitening score (HWS) was calculated (1: pure black; 2: black > white; 3: black = white; 4: white > black; and 5: pure white). BMD was assessed using Furuno CM-200 ultrasound bone densitometer [Figure 1]. This machine uses ultrasound (500 Hz) to measure the speed of sound in heel (calcaneus). The machine’s %CV (coefficient of variation) is 0.5%, and it provides accurate results by compensating the heel temperature. T-score of BMD between 1.0 and 2.5 was considered as osteopenia.

The data were analyzed using Statistical Package for the Social Sciences, Version 21.0 (IBM Corporation, Armonk, New York, USA). Chi-square test and Student’s t-test were used to compare the data. Logistic regression analysis was performed to see the simultaneous effect of various explanatory variables. The confidence level of the study was kept at 95%; hence, P < 0.05 indicated statistically significant association. Ethical clearance was taken from the institutional ethical committee for the purpose of the study.

RESULTS

The mean age of cases (23.63 ± 3.33 Years) and controls (22.66 ± 2.81) was not significantly different (P = 0.086). The mean BMI of case and control groups was matched (22.22 ± 3.04 Vs. 22.64 ± 3.88, P = 0.522). The mean
Age of onset of graying of hair among the cases was 20.62 ± 3.74 Years [Table 1].

Among the individuals with PGH, maximum cases had a HWS of 2 (95.12%), followed by 3 (3.66%) and 5 (1.21%) [Figure 2 and Table 2].

The mean BMD of the case group was 0.76 ± 1.00 and the control group was 0.68 ± 1.11, but the difference was not statistically significant (P = 0.649) [Table 3]. The proportion of cases having low BMD was slightly more than the control group (odds ratio = 1.54), though the risk was not found to be significant [Figure 3].

Among the biodemographic factors, a higher age group of 25–30 years (P = 0.016) and family history of PGH (P < 0.001) were found to be significant risk factors for PGH. However, the intake of vegetarian/nonvegetarian food (P = 0.016) and amount of milk intake per day (P = 0.008) and history of back pain (P = 0.031) and smoking (P = 0.401) did not alter the risk of PGH.

**DISCUSSION**

Hair graying scientifically termed as canities is a physiological phenomenon that occurs with chronological aging, regardless of the gender or race. When graying begins before the usual age of onset, it is termed as PGH or premature canities. PGH has been proposed as a clinical marker of osteopenia in various studies, but the association of PGH with osteopenia has not been validated in large studies.

In our study of 132 participants between the age group of 18 and 30 years, graying of hair was present in 82 (62.12%) cases and graying was absent in 50 (37.87%) controls. In our study, the mean age of onset of graying of hair was 20.62 ± 3.74 years. Similar age distribution of PGH was observed by Shin et al. [11] (20.2 ± 1.3 years). However, Daulatabad et al. [12] observed PGH in age group as low as 11.6 ± 3.6 years.

Out of 132 individuals, osteopenia was present in 56 (42.4%) participants and absent in 76 (57.6%) participants. The proportion of osteopenia in cases with PGH was slightly more than the normal individuals. However, the risk was not found to be significant. Our result was similar to that of Chakrabarty et al. [13] who reported that there was no significant association between PGH and serum calcium concentration. Other studies reported a significant association between low calcium levels and PGH. [14,15] In our study, the mean BMD difference between cases and controls was not statistically significant (P = 0.649), which was similar to the observations of Orr-Walker et al. [16] However, Rosen et al. [4] had reported that individuals with PGH were 4 times more likely to have osteopenia than individuals without graying. The possibility of association found between osteopenia and PGH in previous studies being purely coincidental cannot be denied. It is proposed that the etiopathogenesis mechanisms of both the said conditions follow different paths and are not related to each other.

We observed that family history is a significant risk factor of PGH. Similar observations have been recorded in previous studies. [11,12,17] It is proven that PGH has genetic roots. It follows an autosomal dominant inheritance. [18] but has a multifactorial etiology. The various causes include stress, autoimmune conditions, inflammation, environmental factors, nutritional deficiencies, and premature aging.
syndrome. It has been postulated that PGH could be due to exhaustion of melanocyte’s capability to produce pigment for the hair after a defined age.

**CONCLUSION**

To conclude, we did not find any significant association between osteopenia and PGH. Also, a higher age group (25-30 years) and family history of PGH were found to be the significant risk factors for PGH. The limitations of our study include small sample size and lack of long-term follow-up. The number of control group was less due to logistics (less availability of controls in the stipulated time frame of the study). This could have caused some reduction in the power of study. Despite this, the observed results of the study are above the cutoff value of 80%. The accuracy of the study is still more than 80% and the findings are not altered markedly.

Since premature canities has a strong bearing on patients sociocultural acceptance and self-esteem, prospective studies on a large scale are warranted for a better understanding of the etiopathogenesis of this condition.

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**Conflicts of interest**

There are no conflicts of interest.

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