Dyslipidemia in male AGA: Is it more prevalent than we think?

Roopesh Gowda1, Kannan Gopalan2, Muthusamy Kundasamy3, Navakumar Manickam4*

1Junior Resident, 2Professor, 3Professor and HOD, 4Assistant Professor, 4Dept. of Skin, Vinayaka Mission’s KirupanandaVariyar Medical College & Hospital, Research Foundation, Salem, Tamil Nadu, India

*Corresponding Author: Navakumar Manickam
Email: drnava2k3@gmail.com

Abstract

Introduction: The aim of the study was to determine if male androgenetic alopecia (AGA) patients have abnormal lipid profile as hyperlipidemia is suspected to be quite common amongst this group of individuals.

Materials and Methods: A cross sectional observational study of 100 male androgenetic alopecia patients aged between 18-55 years was conducted during a period of one year. Fasting serum lipid profile including total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL), and low density lipoprotein (LDL) was obtained and recorded in a predesigned proforma for analysis and interpretation of data. Statistical analysis was calculated using SPSS software.

Results: Amongst all four variables, mean TG (151.90mg/dl ± 59.657) was the only one which was above laboratory reference range (>150mg/dl). A total of 46% of the study group had an increased TG level while only 23% had increased LDL levels. Nearly half (48%) of the study group had an above-normal range TC.

Conclusion: Only one parameter, triglyceride was elevated in nearly half (46%) of the study group and we discovered that both triglyceride and total cholesterol in the late onset AGA group were much higher than in the early onset group which was contradictory to previous studies.

Since it is suggested that individuals with male pattern AGA have a higher likelihood for developing dyslipidemia at some point in life, awareness should be unfurled amongst such predisposed individuals. Furthermore, emphasis should be placed on early screening and lifestyle modifications which could circumvent other medical ailments that could occur secondary to hyperlipidemia.

Keywords: Male androgenetic alopecia, Prevalence, Lipid profile, Dyslipidemia.

Introduction

Androgenetic alopecia has been the most dreaded concern among society since the great plague. Though various other medical ailments like cancer, tuberculosis, diabetes, HTN etc. can creep up on and cripple the population, it is without any doubt that male androgenetic alopecia (AGA) has been the most psychologically distressing condition mankind has faced.

It is also one of the most prevalent conditions worldwide, affecting nearly fifty percent of the entire male population. This very condition has been shown to be associated with hyperlipidemia as described by various authors in the past. Numerous studies have been conducted seeking an association between male AGA and hyperlipidemia, but lack of substantial evidence, consistency, and uniformity amongst them was the reason we undertook this study.

Materials and Methods

We conducted a cross sectional observational study in our Skin and STD Outpatient Department for a period of one year from April 2017 to April 2018 after obtaining clearance from the ethical committee of our institution. One hundred male patients diagnosed with androgenetic alopecia aged between 18-55 years were included. Only those who did not smoke, consume alcohol, or have concurrent associated systemic conditions i.e CVD, hypertension, diabetes mellitus and on treatment for the same, nor have received treatment of any sort for AGA within the past 6 months were enrolled in the study after obtaining an informed written consent. Diagnosis was made based on clinical examination and the patients were subsequently subjected to a 12 hour fasting lipid profile.

Each patient was thoroughly clinically assessed and graded according to the Norwood-Hamilton classification of hair loss based on severity. Demographic details, fasting serum lipid profile including total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL), and low density lipoprotein (LDL), were recorded in a predesigned proforma for analysis and interpretation of data.

Statistical analysis was calculated using SPSS software (version 22.0 SPSS Inc., Chicago, IL, USA.)

Results

The average lipid profile of the entire study group of 100 male AGA individuals were the following; (TG) 151.90mg/dl ± 59.657, (LDL) 99.09mg/dl ± 36.987, (HDL) 47.17mg/dl ± 8.868, and (TC) 174.68mg/dl ± 38.356.

Amongst the four parameters, mean TG was the only one which was above laboratory reference range (>150mg/dl).

In comparison of the lipid profile of early and late onset AGA, the parameters that were statistically significant were triglyceride, with a P-value of 0.010 and total cholesterol with a P-value of 0.018 according to the independent T-test.

Late onset of AGA patients had a higher average TG of (164.64mg/dl ± 62.988) than the early onset group (133.56mg/dl ± 49.711) as well as a higher average TC (182.19mg/dl ± 40.677) than the early onset (163.88mg/dl ± 32.238), contrary to a few previous studies.

Based on our study, we could concur that the late onset group had a significantly higher lipid profile than the early onset.
Discussion
The hypothesis that male AGA could be a risk factor for hyperlipidemia which could in turn lead to secondary manifestations such as metabolic syndrome, and vascular abnormalities was first suggested by Cotton et al.\(^1\)

AGA is considered as an autoimmune disorder with genetic predisposition that ensues with a high tendency to progress inevitably though individual disparity in pattern can occur. A sequence of events initially manifesting as frontal hair line recession ends with balding of the vertex. Eventually, only a brim of normal hair growth will be present over the suboccipital and temporal areas of the scalp.

Due to increased level DHT and androgen receptor binding in such predisposed individuals, a transition transpires from healthy terminal hair to shorter, thinner, vellus hair which in due course will disappear completely.

Salvador Arias- Santiago et al.\(^2\) had the most resemblance to our study in terms of methodology and results since theirs yielded a higher average TG, LDL and TC than the control. Our study also portrayed similar data with a higher TG in the study group. We perceived remarkably higher and statistically significant P-values of 0.010 for TG and 0.018 for TC in the late onset group which contradicts assertions made by past investigators that have enrolled only early onset AGA subjects with the presumption that hyperlipidemia could develop at some point of time in such a predisposed group of individuals.

Though the mean TC was only on the borderline to the “cut-off” mark for dyslipidaemia, it was indeed still higher than in the control group. The level of LDL in Santiago et al.\(^2\) was considerably higher in the study population than the control. However, in our study, the mean LDL was not of much importance since it was not elevated as much as we expected.

Our mean HDL was almost the same as in a study by Santiago et al.\(^2\) in which both were within the normal limits. Apart from this current study, a total of three previous ones by Salvador Arias-Santiago et al.\(^2\) Ola Ahmed Bakry et al.\(^3\) & K.C Dharam Kumar et al.\(^4\) had abnormal TG levels (>150mg/dl) thus satisfying one element of the criteria for dyslipidaemia.

Out of the 6 previous studies we used to compare the lipid profile with, Al-Sadat Mosbeh et al.\(^5\) was the only one that showed abnormal mean LDL (147.40mg/dl). It was evident that an elevated LDL was not as common as we expected to be amongst male AGA individuals.

Our study results were very similar to Harmee Singh Banger et al.\(^6\) except for the fact that TG was not included in their study. The remaining three parameters were within normal limits as were in our study.

The studies conducted by Ola Ahmed Bakry et al.\(^3\) and K.C. Dharam Kumar et al.\(^4\) measured only two parameters of the lipid profile, namely TG, and HDL. The mean TG of both studies were deemed abnormal, (160.91mg/dl and 169.030mg/dl). This allows us to form a rationale for male AGA individuals having consistently higher lipid profile than non AGA group.

Limitations of the study
A control group consisting of age-matched individuals without AGA should have been implemented, thus allowing us to compare the lab values with a similar aged non-AGA population which would have assisted in thorough assessment of the association if any between AGA and abnormal lipid profile.

Conclusion
This study could not substantiate the possibility of an association between hyperlipidemia and AGA since only one parameter; triglyceride was increased in 46 % of the study population among 100 male AGA patients. We observed that the late onset AGA group has a higher mean TG and TC as opposed to the early onset group, contradictory to what was concluded in a couple of previous studies which focused on only early onset AGA with the presumption that presence of hyperlipidemia at an earlier age could be an indicator of abnormal lipid profile later in life.

More studies need to be conducted with precise comparison between, early and late onset AGA with an age matched control group, excluding those with history of smoking and who consume or have consumed alcohol for a significant period of time in their life. Many variables could alter the mean lipid profile values thus making it onerous to correlate the presence of an association between male AGA and hyperlipidemia.

Table 1: The mean age of male subjects with AGA was 38.95 ± 11.659.

| Age group: | Frequency | Percent |
|-----------|-----------|---------|
| 18-25     | 16        | 16.0    |
| 26-35     | 25        | 25.0    |
| 36-45     | 27        | 27.0    |
| ≥46       | 32        | 32.0    |
| Total:    | 100       | 100.0   |

Table 2: The age of onset for patients with androgenetic alopecia varied from 18 to 45 years.

| Age of onset | Mean | Std. Deviation |
|--------------|------|----------------|
| Duration of Alopecia | 11.505 ± | 6.6193 |
Table 3:

| Lipid Profile | Mean (in mg/dl) | Std. Deviation (mg/dl) |
|---------------|-----------------|------------------------|
| TG            | 151.90          | 59.657                 |
| LDL           | 99.09           | 36.987                 |
| HDL           | 47.17           | 8.868                  |
| Total Cholesterol | 174.68          | 38.356                 |

Table 4:

|             | Early            | Late             | P-value (independent t test) |
|-------------|------------------|------------------|-----------------------------|
|             | Mean             | Std. Deviation   | Mean                        | Std. Deviation |
| TG          | 133.56           | 49.711           | 164.64                      | 62.988         | 0.010**         |
| LDL         | 92.17            | 34.387           | 103.90                      | 38.240         | 0.119           |
| HDL         | 48.66            | 12.371           | 46.14                       | 5.104          | 0.163           |
| Total Cholesterol | 163.88          | 32.238           | 182.19                      | 40.677         | 0.018**         |

**Statistically Significant

Table 5:

|             | Onset            | Total            | P value (chi square test) |
|-------------|------------------|------------------|--------------------------|
|             | Early (n=41)     | Late (n=59)      |                          |
| TG          | Normal           | 28               | 26                        | 54                        | 0.017**         |
|             | Above normal     | 13               | 33                        | 46                        | 0.097           |
| LDL         | Normal           | 35               | 42                        | 77                        | 0.303           |
|             | Above normal     | 6                | 17                        | 23                        | 0.718           |
| HDL         | Normal           | 41               | 58                        | 99                        | 0.303           |
|             | Above normal     | 0                | 1                         | 1                         | 0.718           |
| Total Cholesterol | Sub-normal     | 9                | 17                        | 26                        | 0.718           |
|             | Normal           | 23               | 29                        | 52                        | 0.718           |
|             | Above normal     | 9                | 13                        | 22                        | 0.718           |

As the previous Table 5 depicts, the late onset group had conspicuously more subjects (33) with an increased TG than the early onset group (13) which is why this parameter was the only statistically significant one with a P-value of 0.017 based on chi square test.

Graph 1: Nearly 60% of the study population had an early onset of AGA.

Graph 2: The most common types of hair loss pattern were Grades 3, 3A, 2, and 2A in decreasing order of frequency.
Roopesh Gowda et al.  

Dyslipidemia in male AGA: Is it more prevalent than we think?

Grade: 1

Grade: 2A

Grade: 3

Grade: 3: Vertex

Grade: 4A

Grade: 5A
Grade: 6

Acknowledgement: None.

Conflict of Interest: None.

References
1. Cotton SG, Nixon JM, Carpenter RG, Evans DW. Factors discriminating men with coronary heart disease from healthy controls. Br Heart J 1972;34:458–64.
2. Salvador Arias Santiago et al. A Comparative Study of Dyslipidaemia in Men and Women with Androgenic Alopecia. Acta Derm Venereol 2010;90: 485–7
3. Ola Ahmed Bakry et al. Androgenetic alopecia, metabolic syndrome, and insulin resistance: Is there any association? A case–control study. Indian Dermatol Online J 2014;5(3):276–81.
4. K. C. Dharam Kumar. Association of Androgenetic Alopecia with Metabolic Syndrome: A Case–control Study on 100 Patients in a Tertiary Care Hospital in South India. Indian J Endocrinol Metab 2018;22(2):196-9

Grade: 7

5. Al-Sadat Mosbeh. Dyslipidemia in patients with early onset androgenetic alopecia and risk of coronary artery disease. Gulf J Dermatol Venereology 2014;21(1):23-8.
6. Harmeet Sing Banger et al. Is Early Onset Androgenic Alopecia a Marker of Metabolic Syndrome and Carotid Artery Atherosclerosis in Young Indian Male Patients? Int J Trichology 2015;7(4):141–7.

How to cite this article: Gowda R, Gopalan K, Kundasamy M, Manickam N, Dyslipidemia in male AGA: Is it more prevalent than we think?. Indian J Clin Exp Dermatol 2019;5(2):116-120.