Medical Science

pISSN 2321–7359; eISSN 2321–7367

Cardiovascular risk factors and co-morbidities in patients with Alopecia Areata: A case-control study

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To Cite:
Attallah DAA, Ashry MAM, Abourehab S, Mahran AM. Cardiovascular risk factors and co-morbidities in patients with Alopecia Areata: A case-control study. Medical Science, 2022, 26, ms37e1866.
doi: https://doi.org/10.54905/disssi/v26i119/ms37e1866

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Peer-Review History
Received: 12 October 2021
Reviewed & Revised: 14/October/2021 to 9/January/2022
Accepted: 10 January 2022
Published: 19 January 2022

Peer-review Method
External peer-review was done through double-blind method.

URL: https://www.discoveryjournals.org/medicalscience

ABSTRACT

Objective: Study the possible cardiovascular risk factors and co-morbidities in patients with Alopecia Areata. *Design:* Case-control study. *Setting:* Dermatology Outpatients' Clinic, Assiut University Hospitals, Egypt. *Patients and Methods:* One hundred Alopecia Areata patients and fifty age and sex matched healthy controls were recruited from the Dermatology Outpatients' Clinic, Assiut University Hospitals, Egypt at the time from June 2018 to January 2020. Each participant was exposed to full history taking, general and dermatological clinical examinations, Severity of Alopecia Tool score and Body Mass Index evaluation. Investigations included Complete Blood Count, Lipogram, random blood sugar, Electrocardiogram and Echocardiography. *Results:* Body Mass Index of patients was significantly higher than that of the controls. Patients had significantly lower levels of hemoglobin. Triglycerides, Cholesterol and Low Density Lipoproteins levels were significantly higher in patients. However, High Density Lipoproteins levels were significantly lower in patients. Positive correlations were detected between SALT score with Triglycerides, Cholesterol, Low Density Lipoproteins and random blood glucose. No definite ECG or ECHO abnormalities could be identified in patients. *Conclusion:* Patients with Alopecia Areata, especially severe forms, have several CV risk factors which make them liable for future development of definite CV diseases.

Keywords: Alopecia Areata, Cardiovascular comorbidities, SALT score

1. INTRODUCTION

Alopecia Areata (AA) is non-cicatricial hair loss of any hair bearing area, with a lifetime prevalence of 2.1%. Diagnosis is based on its characteristic clinical features, which can have different patterns. Scalp is the most commonly involved site (Mirozoyev et al., 2014). AA has wide difference in presentations and severity. Unfortunately, treatment response can be disappointing (Alkhalifah, 2013). Moreover, the disease itself can have profound psychological implications on patients and maybe associated with other co-morbidities (Garcia Hernande et al., 1999). Autoimmunity, genetic factors,

stress, disorders of vasculature and innervations, cytokines' influence and even viral infections have been incriminated (Huang et al., 2013; Mirozoyev et al., 2014). It has been suggested that AA might be associated with coronary artery diseases (CADs) (Lotufo et al., 2000).

Metabolic syndrome and cardiovascular (CV) risks have been reported to be associated with psoriasis (Huang et al., 2013), However, systemic influences of AA on the CV system have been controversial (Kang et al., 2015). Because of the significant morbidity and mortality of CV diseases as well as the commonality of AA, our hypothesis was to study the CV risk factors and co-morbidities in patients with AA. Also, we wanted to explore the predictory role of certain clinical variables in developing such risk factors.

2. PATIENTS AND METHODS

Study design

This is a case control study. An informed consent was obtained from all study participants.

Patients

One hundred patients with the clinical diagnosis of AA and 50 healthy controls were gathered from the Dermatology Outpatients' Clinic, Assiut University Hospitals, Egypt at the time from June 2018 to January 2020. Each patient was subjected to the following: history taking, general and dermatological clinical examinations, Severity of Alopecia Tool (SALT) score, Body mass index (BMI) and investigations which included: Complete Blood Count (CBC), lipogram, random blood sugar (RBS), ECG and Echocardiography.

Exclusion criteria

Patients < 16 years or >60.

Patients with any other dermatological disease

Patients on any systemic or topical therapeutic during the past one month before enrollment in the study

Body Mass Index

Body Mass Index (BMI) is a person's weight measured in Kilograms divided by the square of height in meters. BMI between 18.5 and 25 kg/m² means a normal weight. BMI of lower than 18.5 kg/m² is considered underweight. BMI between 25 kg/m² and 29.9 kg/m² is a reflectance overweight. BMI of 30 kg/m² or more is considered obese (Blackburn and Jacobs, 2014).

Assessment of AA severity using SALT score

The National Alopecia Areata Foundation working committee has devised "Severity of Alopecia Tool score" (SALT score) to assess the AA severity. The SALT score is calculated by measuring the percentage of hair loss in each of 4 areas of the scalp (40% vertex area, 18% right profile area, 18% left profile area, 24% posterior area) and adding the total to achieve a composite score (Olsen et al., 2004). Summing the result of each diseased area will give the result of SALT score. Score can be interpreted as follows:

*S0 = no hair loss.

*S1 = 25% hair loss.

*S2 =25-49% hair loss.

*S3 = 50-74% hair loss.

*S4 = 75-99% hair loss. (a = 75-95% hair loss, b = 96-99% hair loss).

*S5 = 100% hair loss.

Laboratory investigations

Complete blood picture was evaluated by Cell-DYN 3700 (Abbott diagnostics, UK). Lipid profile was evaluated with an auto-analyser Cobas c311 (Roche/Hitachi, Cobas c system, Basel, Switzerland), after fasting for 12 hours. RBS was performed with an auto-analyser Cobas c311 (Roche/Hitachi, Cobas c system, Basel, Switzerland).

Electrocardiography (ECG)

Resting 12-lead ECG recognizes any little electrical change that originates from heart muscles. ECG can be used to evaluate the rate and rhythm of heartbeats, heart chambers and the presence of any disease of the heart muscles or conduction system.

Echocardiography

Echocardiogram (ECHO) and Doppler was done using Philips (HD11XE) with an S3-1probe. ECHO evaluates wall motion abnormalities, cardiac function and cardiac valves abnormalities.

Statistical Analysis

An Excel sheet was developed for data entry. Data were then transferred to version 23 SPSS (Statistical Package for Social Science) (Chicago, IL, USA). Descriptive statistics included: numbers, percentages, means and standard deviation (SD). Chi- square test was used to examine difference between qualitative variables. T-test was used to compare quantitative variables between groups and ANOVA test was used for more than two groups. A probability value (P value) was considered significant when $p<0.05$.

3. RESULTS

This study included 100 patients with the clinical diagnosis of AA and 50 healthy volunteers. Study subjects were chosen from the Dermatology Outpatients' Clinic, Assiut University Hospitals, Assiut, Egypt. Of the 100 patients, 65% were females. Regarding BMI, we found that patients have significantly higher values than controls. Demographic data are represented in (Table 1).

Table 1 Demographic data & BMI of study subjects

Item	Patients "No =100"	Control group "No =50"	p value
1- Age "years" (Mean± SD)	34.78±8.15	36.92±6.28	p=0.372
2-Sex (no, %)			
Male	35(35.0%)	18(36.0%)	p=0.593
Female	65(65.0%)	32(64.0%)	
3-Residence: (no, %)			
Rural	30(30.0%)	14(28.0%)	p=0.274
Urban	70(70.0%)	36(72.0%)	
4-Marital status : (no, %)			
Single	33(33.0%)	13(26.0%)	p=0.385
Married	77(77.0%)	37(74.0%)	
5-Weight'Kg"(Mean± SD)	79.46±9.87	74.26±6.45	p<0.01
6-Height"cm" (Mean± SD)	165.09±9.37	167.42±8.21	p=0.264
7-BMI"kg/m²"(Mean± SD)	32.89±4.55 (obese)	29.00±4.46	p<0.02
8-Family history: (no, %)	15(15%)	0	p< 0.03
9-History of other autoimmune disease: (no, %)	2(2%)	0	p=0.463

Clinical Data

The mean ± SD of disease duration in our patients was found to be 9.59±5.45 years. The disease had sudden onset in 77% of patients and progressive course in 80% of patients (Figure 1). Only 17% of patients had past history of AA and 6% had history of Alopecia Totalis (AT) (Table 2). We found that 85% of patients have patchy hair loss pattern, 8% have Ophiasis, 4% have Alopecia Universalis (AU) and only 3% have AT (Figure 2). In addition, 16% of patients had nail changes, of which only 8% had twenty nail dystrophic changes. Also, we found that 30% of patients have associated eyebrows involvement. Surprisingly, no patients had any ECG or ECHO abnormalities (Table 2 and Figure 3).

Table 2 Patients' Clinical Data

Item	Patients "No=100"
1-Duration of disease (years) (Mean ± SD)	9.59±5.45
2-Onset: (no, %)	

Sudden	77(77.0%)
Gradual	23(23.0%)
3-Course: (no, %)	
Progressive	80(80.0%)
Stationary	14(14.0%)
Regressive	6(6.0%)
4-Prior history of Alopecia Aerata: (no, %) Yes No	 17(17.0%) 83(83.0%)
5-History of AT or AU: (no, %) Yes No	 6(6.0%) 94(94.0%)
6-Pattern of hair loss: (no, %)	
Patchy Ophiasis Universalis	85(85.0%) 8(8.0%) 4(4.0%)
Totalis	3(3.0%)
7-Body hair loss: (no, %)	
No body hair loss	37(37.0%)
Some body hair loss	59(59.0%)
100% body hair loss	4(4.0%)
8-Nail changes: (no, %)	
No nail involvement	84(84.0%)
Some nail involvement	8(8.0%)
Twenty nail dystrophy/trachonychia	8(8.0%)
9-Involvement of eyebrows: (no, %) Yes No	 30(30.0%) 70 (70.0%)
10-ECG "normal" (no, %)	100(100%)
11-ECHO "normal" (no, %)	100(100%)

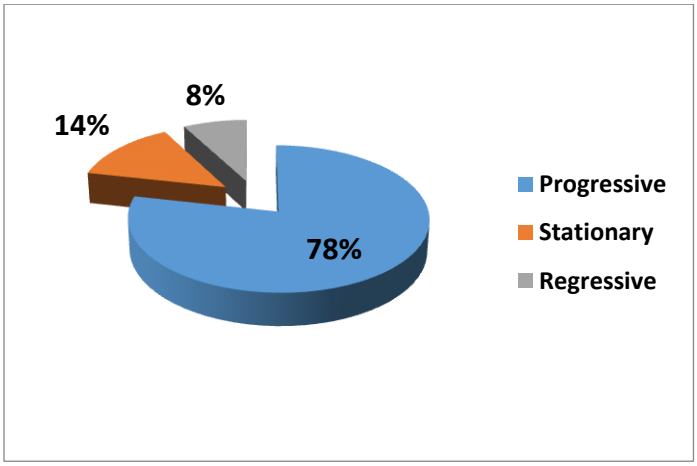

Figure 1 Disease course in the studied group.

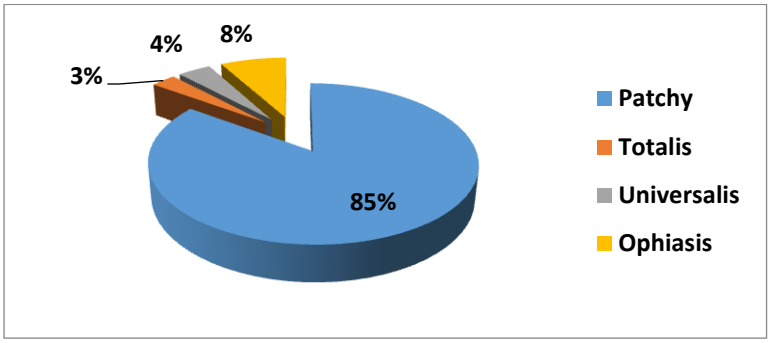

Figure 2 Hair loss pattern in the studied group.

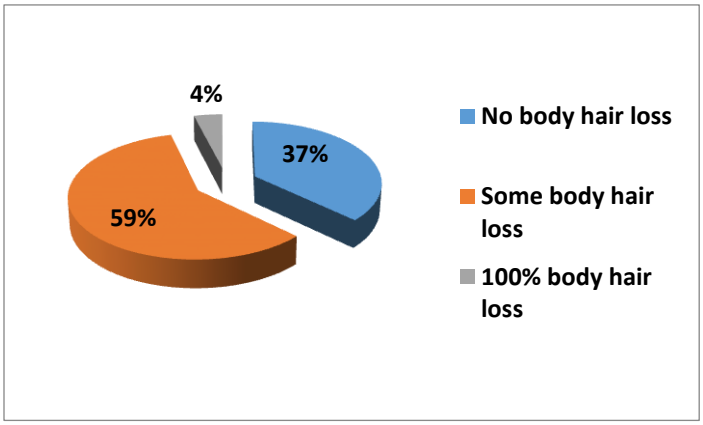

Figure 3 Body hair losses in studied population.

Laboratory findings

Regarding Hemogram, HB and WBCs were significantly lower in patients ($p<0.02$). However, platelets' count was significantly more in patients ($p<0.000$). Lipogram, Triglycerides (TG); Low Density Lipoproteins (LDL) and Cholesterol were significantly more in patients than controls ($p < 0.000$, $p<0.000$ and $p <0.002$, respectively). However, High Density Lipoproteins (HDL) levels were significantly less in patients. In addition, RBS levels were of a higher significance in patients ($p< 0.000$) (Table 3).

Table 3 Laboratory findings in patients and controls

Item (Mean ±SD)	Patients "NO=100"	Control group "NO=50"	P-value
1-HB g/dl	11.56±0.23	13.47±0.80	P<0.02
2-Platelets k/ul	218.96±62.60	170.40±17.83	P<0.000
3-WBCs k/ul	5.91±2.15	6.64±0.89	P<0.02
4-TG mg/dl	105.60±8.36	67.90±5.33	P<0.000
5- TC mg/dl	223.07±32.98	152.00±26.49	P<0.002
6-HDL mg/dl	50.21±15.13	72.44±5.61	P<0.000
7-LDL mg/dl	94.00±22.12	76.60±5.57	P<0.000
8-Random Blood Glucose mmol/l	6.52±0.61	5.13±0.77	P<0.000

Correlations between clinical data & disease severity

We detected negative correlations between SALT score with age of onset ($p<0.000$) (Figure 4) and disease duration ($p<0.000$) (Figure 5). On the opposite hand, a positive relationship was detected between SALT score and systolic blood pressure ($p<0.001$) (Figure 6 and Table 4)

Table 4 Correlation between clinical data & SALT score

		SALT score
Age	R	0.002
	P	0.982
Age onset	R	-0.421
	P	0.000
Onset	R	0.047
	P	0.656
Duration of disease	R	-0.997
	P	0.000
Pulse	R	-0.630
	P	0.000
SBP	R	0.380
	P	0.001
DBP	R	0.144
	P	0.222

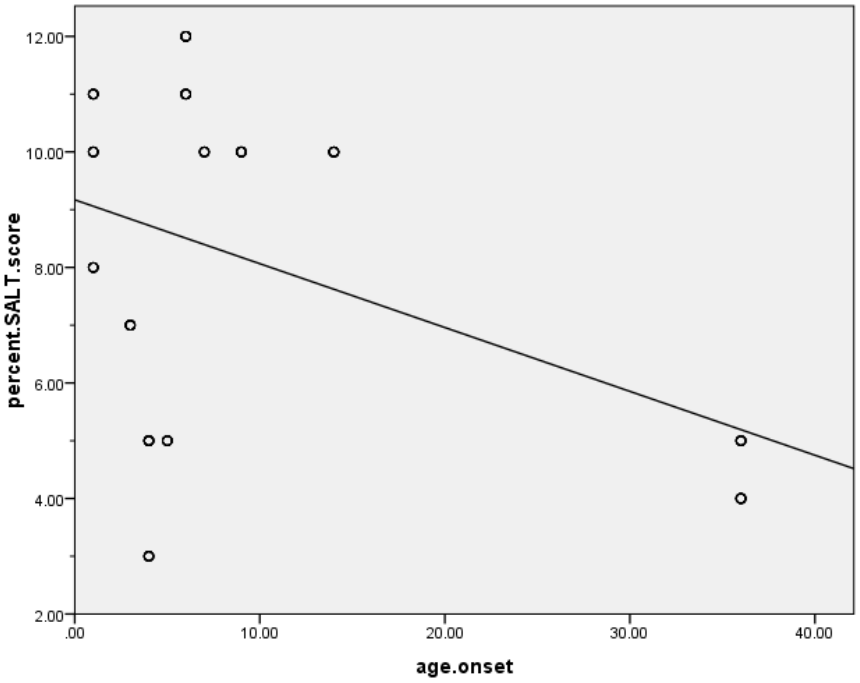

Figure 4 Correlation between age of onset & SALT Score

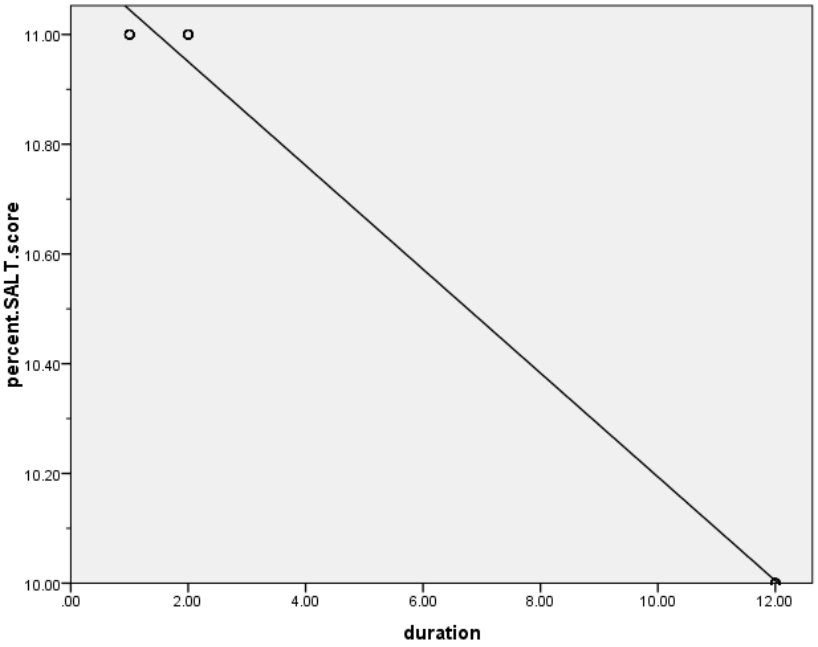

Figure 5 Correlation between disease duration & SALT Score

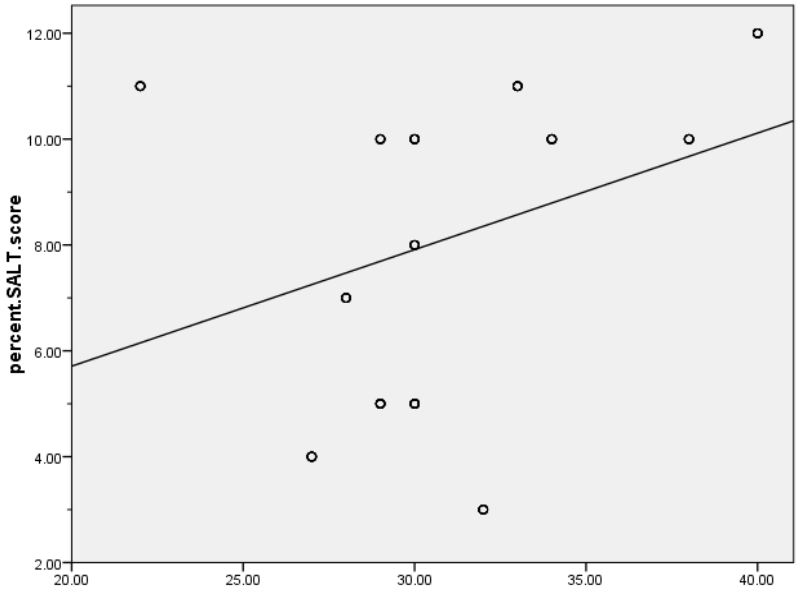

Figure 6 Correlations between SBP & SALT Score

Correlations between Laboratory data and severity

We detected positive correlations between SALT score with WBCs (p<0.000), TG (p<0.001), Cholesterol (p<0.009) (Figure 7), LDL (P<0.001) and RBS (P<0.000). On the other hand, the relationship between SALT score and HDL was of a negative nature (p<0.000) (Table 5).

Table 5 Correlation between SALT score and laboratory data

		SALT score
Hb	R	-0.178
	P	0.077
Platelets	R	0.145
	P	0.149

WBCs	R	0.457
	P	0.000
Triglyceride	R	.0462
	P	0.001
Cholesterol	R	0.534
	P	0.009
HDL	R	-0.457
	P	0.000
LDL	R	0.452
	P	0.001
Random Glucose	R	0.356
	P	0.000

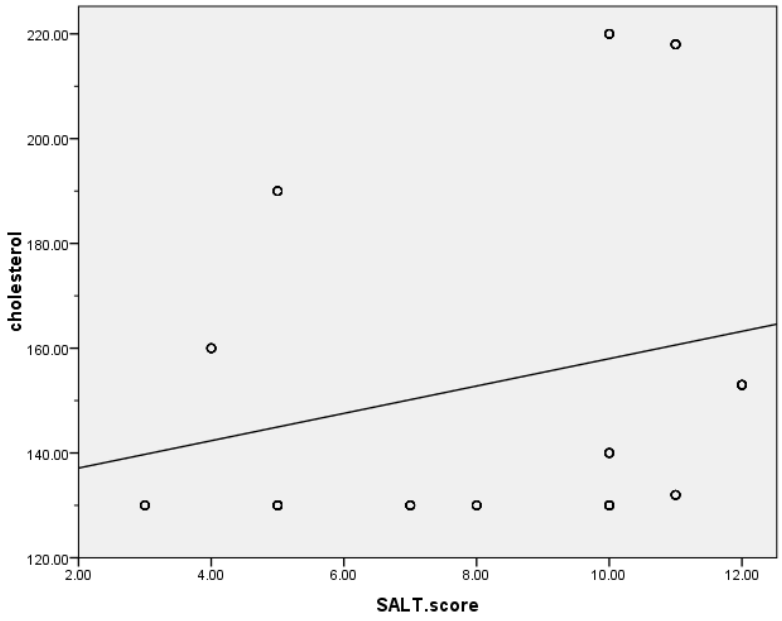

Figure 7 Correlations between Cholesterol & SALT Score

Correlations between clinical & laboratory data

We detected significant positive correlations between Age with TG (p<0.000), Cholesterol (P <0.001), LDL (p<0.003) and RBS (p<0.000). Also, we found positive correlations between disease duration with TG (p< 0.007), Cholesterol (p< 0.029), LDL (p<0.000) and RBS (p<0.000). On the other hand, negative correlations were detected between age and disease duration with HDL (p <0.000). Regarding Hemogram, HB was found to be positively correlated with age (P< 0.000). PLT was found to be positively correlated with age of onset (P< 0.000) and disease duration (P<0.000) (Table 6).

Table 6 Correlation between clinical & laboratory data

		Age	Age of onset	Duration
Triglyceridesmg/dl	R	0.589	0.370	0.532
	P	0.000	0.000	0.007
Cholesterolmg/dl	R	0.320	0.317	0.445
	P	0.001	0.001	0.029
LDL mg/dl	R	0.294	0.325	0.997
	P	0.003	0.001	0.000
Random Glucose (mmol/l)	R	0.838	-0.159	0.936
	P	0.000	0.113	0.000

HDL mg/dl	R	-0.428	0.115	-0.997
	P	0.000	0.253	0.000
HB g/dl	R	0.576	-0.075	-0.394
	P	0.000	0.461	0.057
PLTk/ul	R	-0.108	0.358	0.901
	P	0.284	0.000	0.000
WBCk/ul	R	0.044	-0.039	0.413
	P	0.663	0.703	0.045

Relations between laboratory findings with pattern of hair loss, nail changes and eyebrows involvement

Regarding TG ($p<0.02$), Cholesterol ($p<0.04$), LDL ($p<0.03$) and RBS ($p< 0.01$), we found a significant rise in the levels from the mild forms to severe forms with the lowest in the patchy pattern followed by AT, AU and the ophiasis pattern. The reverse was observed in HDL levels ($p< 0.03$). As regards nail changes, we found that platelets' levels were significantly lower in patients with twenty nail affection than in patients with no or some nail affection ($p<0.001$). Also, we detected that TG ($p < 0.000$), Cholesterol ($p<0.000$) and LDL ($p=0.04$) were significantly higher in patients with twenty nail dystrophy than in patients with no or some nail changes. However, HDL ($p <0.02$) was significantly lower in patients with twenty nail dystrophy. We found that TG ($p < 0.04$), Cholesterol ($p < 0.002$), LDL ($p < 0.03$) and RBS ($p<0.02$) were significantly higher in patients with eyebrows involvement. On the other hand, HDL was significantly lower ($p<0.03$) in patients with eyebrows involvement.

4. DISCUSSION

Alopecia areata (AA) is a patchy, non-scarring hair loss of any hairy part. It represents a challenging autoimmune disease in Dermatology. Until now, there is no available therapy with predictable efficacy (Hordinsky and Donati, 2014). The pathogenesis of AA depends on both genetic predisposition and environmental exposures. Predisposing factors, such as stress and infection, decrease the immune privilege of the hair follicle in genetically susceptible persons (Gilhar, 2010). Autoimmune theory has always been strongly suggested (Huang et al., 2013). Previous studies proved the relation between AA and metabolic syndrome. However, few studies have reported the relation of CV risk factors and diseases (CVDs) in AA patients (Huang et al., 2016). We have evaluated CV risk factors and CVDs in patients with AA. Also, we explored the possible relations between the clinical variables and CV diseases' liability factors.

In this study, females represented 65%. Our finding of female predominance is in concordance with the findings of Eileen et al., (2002); Tan et al., (2002) and Trink et al., (2013). This is because females are generally more concerned about cosmetic issues. In this study, the onset of AA was sudden in 77% of patients. This result is similar to those of Kumar et al., (2016). Regarding clinical types, we found that 85% of patients have patchy hair loss, 8% have Ophiasis, 4% have AU and 3% have AT. These findings are almost in concordance with those of Eileen et al., (2002); Thomas and Kadyan, (2008) and Amer et al., (2009). Nail changes were observed in 16% of our patients' population in the form of nail pitting, striations and dystrophy. Almost similarly, Amer et al., (2009) found nail changes in 20% of patients. Previous reports about nail involvement in patients with AA were highly variable from 7-66%.

We found that BMI in patients have significantly greater values than in controls ($p<0.01$). In addition, the patients' mean weight was significantly higher than controls ($P<0.02$). These finding indicated that AA patients are at risk of developing complications of obesity, including CVDs. Our explanation of this finding is that stress as a major precipitating factorcan makes AA patients eat more. Also, hormonal changes in AA patients have been previously reported (Moore and Cunningham, 2012). Moreover, many epidemiological studies showed that stress and BMI are related (Moore and Cunningham, 2012). In contrast to our finding, Severi et al., (2003) reported that BMI and waist circumference has no relation with AA.

In our study, blood glucose levels were significantly higher in patients. Association with DM was also previously reported by Karadag et al., (2013). Moreover, hypertension and diabetes were found to be more common in patients with late onset alopecia (Karadag et al., 2013). So, this finding implies that AA patients are liable to develop DM and its complications. In our study, we established lower levels of HB in AA patients. This finding is in concordance with Kantor and co-workers (2003) who suggested a common pathogenesis. So, curative benefits of anemia on hair regrowth in patients of AA should have received more attention. Ferritin is a micronutrient which has many immunological activities. So, abnormal serum ferritin and iron levels might have a role in AA pathogenesis (Esfandiarpour et al., 2008). The finding of non-drug induced Leukopenia in our patients might be explained by the possibility of a shared autoimmune etiology. Regarding Thrombocytosis which was noticed in our patients, we suggest that it could be of a reactive nature to a mild associated systemic inflammation with AA.

Regarding lipogram, we found that TG, LDL and cholesterol were significantly higher in patients than in controls. Moreover, HDL levels were significantly less in patients. In (2018), Lim and co-workers reported hyperlipidemia in patients with AA. Pinar Incel-Uysal et al., (2019) observed a link between lipid metabolism, cholesterol synthesis and AA. Authors suggested that metabolic intermediates produced in the cholesterol synthetic pathway affect the immune responses including JAK-signal transducer and activator of transcription and peroxisome proliferator–activated receptor signaling. Hence, a connected pathogenesis is very much possible (Pinar et al., 2019). As it is a well-known fact that hyperlipidemia and DM are definite risk factors of CVD, we can conclude that AA itself can be considered a risk factor for CVDs. However, we could not detect definite ECG or ECHO abnormalities in patients with AA. Also, Wang et al., (2018) reported that troponin levels (diagnostic marker for ischemic stroke) were markedly expressed in AA patients.

Regarding correlation between AA severity and the clinical data, we found that SALT score as a marker of severity was correlated negatively with age of onset and disease duration. On the other hand, a positive correlation was detected between SALT score and SBP. This means that AA is more severe in children and chronic cases. Similarly, it was found that long established disease and earlier onset of AA were seen more in AT and AU patients compared to mild and patchy ones (Tosti et al., 2008). Seong-Jin Kim and co-workers in (2017) reported that male gender, nail changes and disease duration were the main markers of severity.

As regards correlation with laboratory data, we detected positive correlations between SALT score with WBCs, TG, cholesterol, LDL and RBS. On the other hand, a negative correlation was detected between SALT score and HDL. This means that severe AA cases are associated more with dyslipidemia and DM. Hence, the severity of AA is linked with a more liability of developing CV illnesses. However, the association of AA severity with hypertension, DM and dyslipidemia was denied by Hye Rin you and Seong-Jin Kim et al., (2017). Interestingly, we found that patients with extensive nail involvement and eyebrows affection are more liable to have dyslipidemia and DM. This means that AA patients with nail and eyebrows affection require more attention in terms of CV risk factors' screening.

Our study has limitations. The relatively small sample size is a limitation, however, our exclusions criteria was the major obstacle. Moreover, further confirmatory investigations of MI like cardiac catheterization were not performed. In fact, we found it unethical to convince AA patients to go through such an invasive and stressful investigation without having obvious symptoms. From our previous findings, we can suggest that AA, especially severe forms, by itself and by its association with obesity; hyperlipidemia and DM can be a definite risk factor to develop CVDs. However, the systemic inflammatory process might be insufficient to cause obvious ischemic cardiac changes. That may be the reason why we could not detect any abnormalities of the ECG and ECHO in AA patients. Moreover, evident CV illness like MI might not be detected by ECG or ECHO and may necessitate further investigations like cardiac catheterization in order to be diagnosed (Wolk et al., 2013).

5. CONCLUSION

Patients with Alopecia Areata, especially severe forms, have several CV risk factors which make them liable for future development of definite CV diseases. So, based on the previous finding, we do recommend laboratory screenings of AA patients, especially the severe forms, for anemia, dyslipidemia and DM. Also, we think that patients with AA and remarkable CV risk factors should be carefully investigated for definite CVDs, in case ECG and ECHO are normal. Also, future studies with larger sample sizes and long follow up periods about CV co-morbidities in patients with AA are very much warranted.

Authors' contribution

All authors were involved in the work and contributed equally.

Ethical Approval

The study was approved by and monitored by the Medical Ethics Committee, Faculty of Medicine, Assiut University IRB number (17101476).

Funding

The study did not receive any external funding

Conflict of interests

The authors declare that there are no conflicts of interests.

Data and materials availability

All data associated with this study are present in the paper.

REFERENCES AND NOTES

1. Alkhalifah A. Alopecia areata update. Dermatol Clin 2013; 31:93-108.
2. Amer E, Khalid J, Majid S. Pattern and profile of alopecia areata in Pakistan. J Pak Assoc Dermatol 2009; 19: 136-140.
3. Blackburn H, Jacobs D. Commentary: Origins and evolution of body mass index (BMI): continuing saga. Int J Epidemiol 2014; 43: 665–674.
4. Eileen T, Yong-Kwang T, Chee-Leok G, Yoke C. The pattern and profile of alopecia areata in Singapore – a study of 219 Asians. Int J Dermatol 2002; 41: 748–753.
5. Esfandiarpour I, Farajzadeh S, Abbaszadeh M. Evaluation of serum iron and ferritin levels in alopecia areata. Dermatol online J 2008; 14: 21-21.
6. Garcia Hernande Z. Alopecia areata, stress and psychiatric disorder a review. J Dermatol 1999; 26:625-632.
7. Gilhar A. Collapse of immune privilege in alopecia areata: coincidental or substantial?. J Invest Dermatol 2010; 130:2535-2537.
8. Hordinsky M, Donatti A. Alopecia areata: an evidence-based treatment update. Am J Clin Dermatol 2014; 15: 231-246.
9. Huang K, Joyce C, Topaz M. Cardiovascular risk in patients with alopecia areata (AA): a propensity-matched retrospective analysis. J Am Acad Dermatol 2016; 75:151-154.
10. Huang K, Mullangi S, Guo Y, Qureshi A. Autoimmune, atopic and mental health comorbid condition associated with alopecia areata in the United States. JAMA Dermatol 2013; 14:789-794.
11. Hye R, Seong-Jin K. Factors Associated with Severity of Alopecia Areata. Ann Dermatol 2017; 29: 565–570.
12. Kang J, Line H, Kaostsaimc-Chung S. Alopecia areata increases the risk of stroke- 3 years follow up study. Sci rep 2015; 1:7-8.
13. Kantor J, Kessler L, Brooks D. Decreased serum ferritin is associated with alopecia in women. J Invest Dermatol 2003; 121:985-988.
14. Karadag A, Ertugrul D, Bilgili S. Insulin resistance is increased in alopecia areata patients. Cutan Ocul Toxicol 2013; 32:102-106.
15. Kumar A, Sharma R, Bali S, Arya P. Role of platelet rich plasma therapy in Alopecia areata- A prospective study. IJCMR 2016; 3: 2499-2502.
16. Lim C, Rachel K, Petukhova L. Big Data Reveal Insights into Alopecia Areata Comorbidities. J Investig Dermatol 2018; 19: 557–561.
17. Lotufo P, Chae C, Ajani U, Hennekens C, Manson J. Male pattern baldness and coronary heart disease: The Physicians' Health Study. Arch Intern Med 2000; 160: 165-171.
18. Mirozoyev S, Schrum A, Davis M, Torgerson P. Lifetime incidence risk of alopecia areata estimated at 2.1% by Rochester epidemiology project. J Invest Dermatol 2014; 134:1141-1142.
19. Moore C, Cunningham S. Social position, psychological stress, and obesity: a systematic review. J Acad Nutr Diet 2012; 112:518–526.
20. Olsen E, Hordinsky M, Price V, Roberts J, Shapiro J, Canfield D. Alopecia areata investigational assessment guidelines. Part II. National Alopecia Areata Foundatin. J Am Acad Dermatol 2004; 51:440-7.
21. Pinar I, Neslihan A, Nuran A, Ayse O, Tuba C, Canan T, Turan T. Assessment of metabolic profile and ischemia-modified albumin level in patients with alopecia areata: A case–control study. Indian J Dermatol 2019; 64: 12-18.
22. Severi G, Sinclair R, Hopper J, English D, McCredie M, Boyle P, Giles G. Androgenetic alopecia in men aged 40-69 years: prevalence and risk factors. Br J Dermatol 2003; 149: 1207-1213.
23. Tosti A, Whiting D, Iorizzo M, Pazzaglia M, Misciali C, Vincenzi C. The role of scalp dermoscopy in the diagnosis of alopecia areata incognito. J Am Acad Dermatol 2008; 59:64-7.
24. Wang E, Santos L, Li X, Tran A, Kim S, Woo K. Alopecia areata is associated with increased expression of heart disease biomarker cardiac troponin I. Acta Derm Venereol 2018; 98:776-782.
25. Wolk M, Bailey S, Doherty J. Multimodality Appropriate use criteria for the detection and risk assessment of stable ischemic heart disease. JACC 2013; 63:380-406.