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Association between smoking and alopecia areata in the attendants of outpatient clinic in Kerbala

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Abstract

Background : Alopecia areata usually presents with well-defined, localised, non inflammatory, non-scarring patches of hair loss,usually on the scalp. Smoking is a risk factor for many diseases that can be avoided, such as oral mucocutaneous disease, cancer, cerebrovascular disease, and pulmonary disease. Recently, there has been a lot of interest and research into how smoking affects alopecia and the aging of the skin.

Aim of the study : The objective of this research was to evaluate the association between smoking and alopecia.

Materials and Methods : A Case series Study including 50 patients from kerbala city whose was diagnosed clinically as cases of alopecia areata and we asked them about smoking habits and interpret results with spss . our study lasting one year.

Results : Of the 50 participants, most of them are young age , 20 are smoker , 15 of smoker person develop alopecia after smoking , current smokers had a higher risk of incident AA than never smokers.

Conclusion : among smoker patients ,most of cases occur after the initiation of smoking.

Introduction :

Alopecia areata (AA) is a common complaint marked by the rapid start of partial hair loss in a clearly defined, typically circular, area. Any place with hair may be impacted, unclear cause[1] .Worldwide, alopecia areata is a problem. About 0.1% is the estimated prevalence rate. Alopecia areata is a difficult immune-mediated, hereditary condition that affects hair follicles in the anagen period. Both toddlers and adults can get alopecia. and both men and women are affected by the illness at comparable rates. Alopecia, Atopic dermatitis, thyroid illness, allergic rhinitis, asthma, and vitiligo are all often linked to other immune-mediated disorders[2,3].commonly Patients with AA have a family history of the disease. Disease is frequently impacted by stress. One study finds minimal indication that emotional stress significantly contributes to the etiology of AA[1].

Pathogenesis of alopecia areata: The relative immunological privilege of the hair follicle is an important feature. This privilege is primarily created by inhibiting MHC class I, which is required for presenting autoantigens to CD8+ T lymphocytes, and by creating a local signaling environment that is inhibiting[4]. According to some researchers, AA may be significantly influenced by the dissolution of the hair follicle's immunological privilege[5].When immune privilege collapses, inflammatory cells target the hair follicle matrix epithelium that is undergoing early cortical differentiation (anagen hair follicles), which causes the hair follicles to enter the catagen phase too soon. The hair follicle may still cycle and replace itself because the stem cells in it are preserved[6].

Clinical Presentation: There is a wide range of engagement. The majority of alopecic patients describe the abrupt appearance of one to several 1- to 4-cm-wide patches of hair loss on the scalp. The skin is white and either hairless or has short hair stubs. Before the patches develop, some patients report burning, tenderness, and itching. The parietotemporo-occipital region exhibits band-like hair loss as the ophiasis type, The likelihood of spontaneous recovery in the ophiasis pattern is low[1].

In alopecia areata, the hair shaft is poorly developed and breaks before it reaches the skin.The afflicted hairs that have been left in place at the edge of a lesion have a constricted base and a normal upper shaft "exclamation point." Regrowth happens within one to three months, although there is a chance of known hair loss in the same location or elsewhere. The new hair is often the same texture and color as the old hair, although it might also be white and fine. Eyelashes, the beard, and very seldom other body parts may be affected.. [1] A tiny number of people with alopecia areata get alopecia totalis, which is full scalp hair loss[7]. Complete loss of hair on the scalp and body is a symptom of alopecia universalis. It is a more severe form of alopecia areata[8]. Nail Changes; Abnormality in shap and color , texture may be accompanied with AA. The incidence is 10% to 66%. Pitting nail with an irregular pattern, longitudinal or transverse lines may seen in single or all of the nails of some patients with AA . Nail dystrophy may coincides, precedes with, or occurs after resolution of AA[1]. Psychologic Implications; Hair is important to a person's appearance a strange pattern of hair loss is psychologically painful. It limits social freedom and impact the quality of life[1]. Alopecia areata significantly lowers a person's quality of life, according to a few older studies[9]. In one study, as many as 61% of those with alopecia areata experienced depression[10].

Diagnosis: The diagnosis is usually clinical and may be aided by a positive hair pull test or trichoscopy results. Trichoscopy can reveal active disease as broken, yellow, black, or "exclamation mark" shaped hairs. Another indicator of alopecia areata is vellus hair in lesions, which may signify a late-stage or inactive disease[11,12]. In unsure circumstances, a biopsy may be performed.

Prognosis: Alopecia areata can develop in a variety of ways. In their lifetimes, some patients may only experience one episode of AA, while others experience many recurrences. During recovery, more variation happens. While some patients may experience complete hair regrowth, others may experience no change or even more hair loss[13].

Treatment : The only current therapies that hair specialists generally agree are beneficial are intralesional corticosteroids and topical immunotherapy. Therefore, widely accepted guidelines suggest them as initial therapies for alopecia areata[14].

Tobacco smoke composition : Solid particle and volatile gas phase can both be found in tobacco smoke. Benzanthracene, benzopyrenes, 2-naphthylamine, catecho, quinolone, aniline, toluidine, nickel, N-nitrosodimethylamine, and nicotine are a few of the main components of the solid phase.Some of the most hazardous compounds in the gas phase are formaldehyde, N-nitrosodimethyamine, and N-nitrosopyrrolidine. Others include carbon monoxide, carbon dioxide, nitrogen oxides, acetone, hydrogen cyanide, acrolein, ammonium, pyridine, and 3-vinyl pyridine[15].Numerous of these factors may have an impact on hair loss, as stated below.

Mechanism of absorption of nicotine and it metabolites : Nicotine and other metabolites enter the body through a variety of routes, including ingesting, inhaling, applying topical cream or patches, and so on. Through blood absorption, nicotine can enter the hair[16] or from exposure to ambient smoke[17]. then Through passive diffusion, blood is absorbed into the developing hair cells through capillaries at the base of the hair follicle.Studies[18,19] showed that nicotine content was

higher in the distal end of hair than in the proximal end. This was because the distal hair had been exposed to ambient smoke for a longer period of time. Because of this, research has suggested that the amount of nicotine in hair may be a useful predictor of extensive smoke exposure[20].

Mechanisms of smoking leading to hair loss : Smoking can hasten hair loss through a variety of processes.

Vasoconstrictor effect : metabolites of nicotine cause a key consequence of both acute and chronic smoking is the constriction of the epidermal microvasculature. Nicotine functions by decreasing acetylcholine-induced endothelium-dependent cutaneous vasodilation and neither increasing the vasoconstrictor impact of nor epinephrine[21].

Effect on free radicals and immunological effect :Both humoral and cell-mediated immune responses are significantly impacted by smoking. As a result, it has been discovered that exposure to cigarette smoke on a regular basis is related to a number of medical disorders. One of the tissues with "immune privilege," which shields them from systemic immunological onslaught, is the hair follicle. AA might develop as a result of the loss of hair follicle immune privilege[3,4]. Additionally, the high concentration of free radicals seen in cigarette smoke has the potential to accumulate in hair follicles, eventually impairing immunity

TNF-, interleukin (IL)-1, and IL-6.2 were among the cytokines that smoking was found to cause to release. By inducing an inflammatory response around the hair follicle, those cytokines may play a significant role in the etiology of AA[2,4,5] . The acute follicular inflammation ends the anagen stage and forces the follicle into catagen.[1]

Hormonal effects : Smoking increased the hydroxylation of estradiol and inhibited the enzyme aromatase, resulting in a hyperestrogenic condition that accelerated androgen-dependent hair thinning[22].Smoking may result in higher amounts of androgens, increasing the chance of androgenic alopecia developing**.**[23]

Smoking and premature hair graying :According to a questionnaire completed by Korean patients aged 12-91, smoking increases the chance of premature graying by 14.9% each year (n = 522 men and 480 women, p 0.001) and by 1.99 times overall compared to nonsmokers[24].

Aim of the study : The objective of this research was to evaluate the association between smoking and alopecia.

Materials and Methods : A case series study conducted on a sample of patients who attended the outpatient clinic of Dermatology and Venereology in al hassan teaching hospital in karbala city during the period between March 2022 to March 2023 , where 50 individuals was enrolled in this study. Each one informed that he or she is a part of scientific study and a verbal consent obtained from each of them . we asked them about duration of smoking and the period of time that that alopecia developed after smoking , also we asked them about family history of smoking , and history of other autoimmune diseases , and interpret results with spss.

Results :

The studied groups included 50 patients. 41 male, 9 female, most of them are young age.

Table 1: The table show the percentage of smoker and non smoker among the participant patients who suffer from alopecia areata

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	smoker	20	40.0	40.0	40.0

	not smoker	30	60.0	60.0	100.0
	Total	50	100.0	100.0	

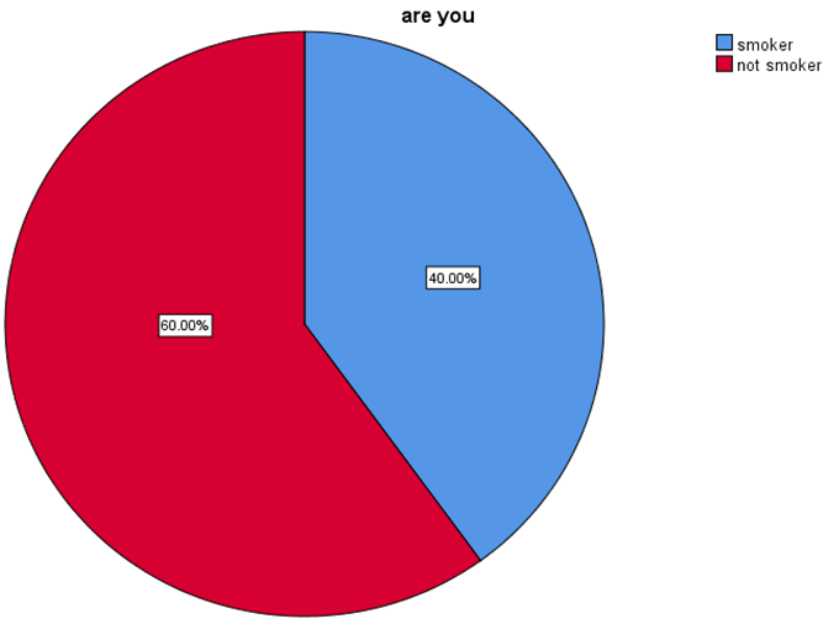

Figure 1: percentage of smoker among participant

Table 2: Percentage of smoker that develop alopecia areata after smoking

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	yes	15	30.0	75.0	75.0
	No	5	10.0	25.0	100.0
	Total	20	40.0	100.0	
Non smoker		30	60.0		
Total		50	100.0		

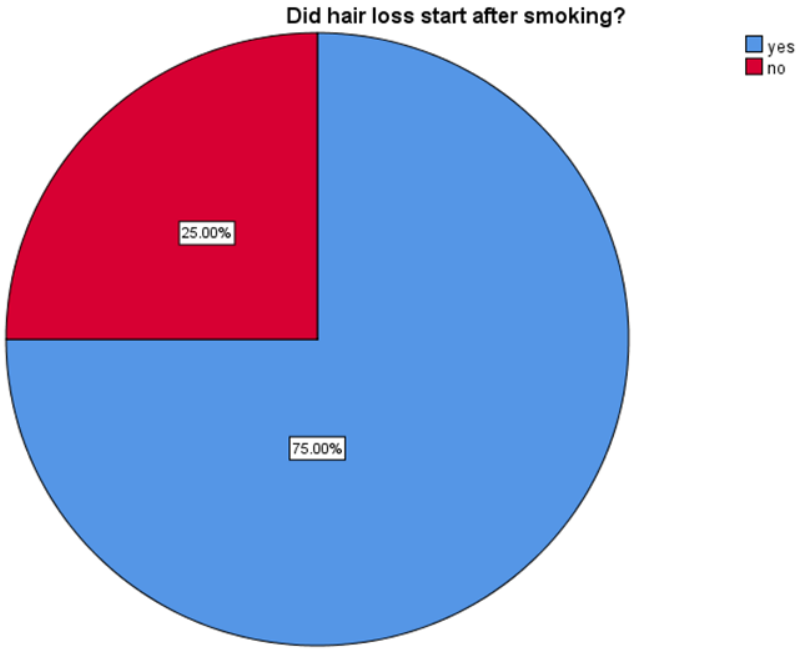

Figure 2 : Percentage of smoker that develop alopecia areata after smoking

Table 3 : This table show the age of participant.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	less than 20	14	28.0	28.0	28.0
	20-30	23	46.0	46.0	74.0
	30-40	6	12.0	12.0	86.0
	More than 40	7	14.0	14.0	100.0
	Total	50	100.0	100.0	

Table 4 : The table show the gender of participant

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	male	41	82.0	82.0	82.0
	female	9	18.0	18.0	100.0
	Total	50	100.0	100.0	

Table 5 : This table show the onset of hair loss after smoking

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	non smoker	35	70.0	70.0	70.0
	1 years	5	10.0	10.0	80.0
	10 years	4	8.0	8.0	88.0
	30 years	1	2.0	2.0	90.0
	5 years	5	10.0	10.0	100.0
	Total	50	100.0	100.0	

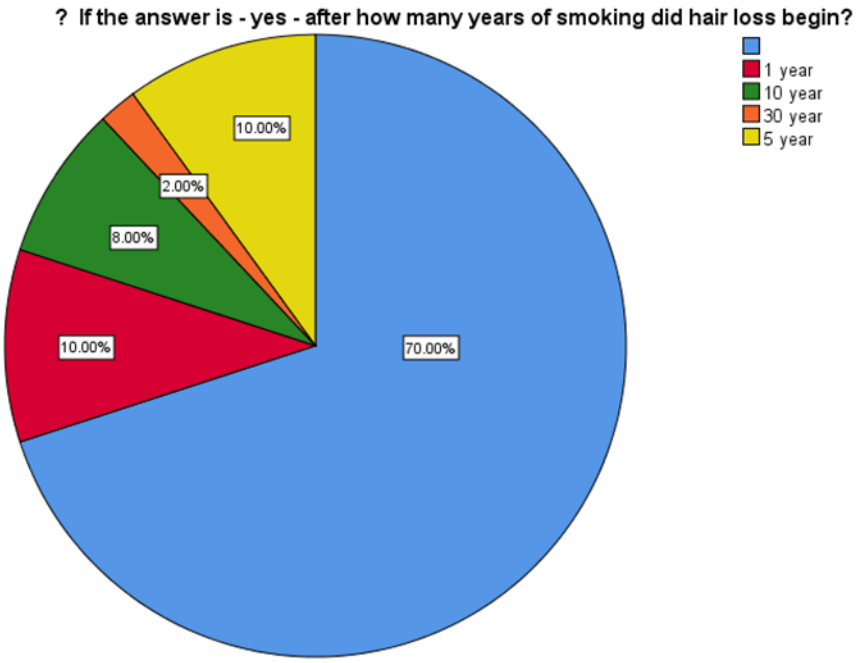

Figure 3 : This figure show the onset of hair loss after smoking

Table 6 : Is there any member of your family or relatives suffering from (alopecia)

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	no	38	76.0	76.0	76.0
	yes	12	24.0	24.0	100.0
	Total	50	100.0	100.0	

Table 7 : Association of other autoimmune disease with alopecia

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Asthma	1	2.0	2.0	2.0
	Vitiligo	1	2.0	2.0	4.1
	Other	2	4.0	4.1	8.2
	Nothing	45	90.0	91.8	100.0
	Total	49	98.0	100.0	
Missing	System	1	2.0		
Total		50	100.0		

Discussion :

While genetics play a significant part in alopecia areata , environmental variables, like as smoking and nicotine exposure, should also be taken into account.

"Our study was done on 50 patients with alopecia areata to assess if there is a relationship between disease and smoking

We found in this study that 40% from participants are smoker and 75% of these patients develop alopecia after smoking , as seen in [table 1&2.]

Recently, Dai et al. looked into the relationship between smoking and AA.3 They discovered that there was a trend toward higher risk with more years of smoking and total pack-years, and that current smokers had a higher risk of incident AA than nonsmokers (adjusted hazard ratio 1.88, 95% CI 1.22-2.88)[25].

In this study we found that Most of the patient are male in young age group as see in [table 3&4]

The average age at which alopecia areata was diagnosed in men was 32 years old and in women it was 36 years old, according to the clinical data gathered from the population of Olmsted County, Minnesota between 1990 and 2009[26].

And about Cumulative smoking effect 10% , 8%, 10%, 2% from participants develop alopecia after 1 years , 5 years , 10 years , 30 years respectively , as seen in [table 5]

Studies on the hair loss-prone C57BL/6 mouse strain may provide the explanation for why people who smoke experience more hair loss. The mice showed symptoms of premature hair graying and hair loss after three months of exposure to ambient tobacco smoke (ETS), including circular alopecic patches and graying on their backs[27].

And 26% of participants patients have family history of alopecia , as seen in [table 6].

And 6.16% of participants patients have other autoimmune disease , as seen in [table 7].

Conclusion :

among smoker patients ,most of cases occur after the initiation of smoking.

Recommendations:

1.Further studies include controls to estimate the significant association between smoking and alopecia areata.

2.As we observed that cases develop after initiation of smoking ,so educational programs about the risk of smoking should be increases especially among young age group.

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