EVALUATION OF CASES OF ALOPECIA AREATA IN BOTH GENDERS

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Abstract

Background: To evaluate cases of Alopecia areata in both genders. Materials and Methods: A sum total of ninety- two cases of Alopecia Areata (AA) of either gender was enrolled. The site was selected and lesion was observed through the dermascope. Grading was performed and hair loss was recorded. Results: Out of 92 patients, 42 (45.6%) were males and 50 (54.4%) were females. Dermascopic findings showed black dots in 24, vallus hair in 23, yellow dots in 21, broken hair in 14 and exclamation mark in 10 patients. A non- significant difference was observed (P > 0.05). Hair loss grade S1 was seen in 4%, S2 in 8%, S3 in 14%, S4 in 20% and S5 in 54% patients. The difference was significant (P < 0.05). Conclusion: Most common dermascopic findings in Alopecia areata patients was vallus hair and yellow dots.

INTRODUCTION

An autoimmune disease called AA targets the hair follicles and results in non-scarring hair loss. A comprehensive assessment of the epidemiology of AA revealed a similar lifetime prevalence of 2% across the globe.[1] Some smaller studies have found a tiny gender difference between men and women, although this could be because women are more concerned about hair loss and its treatment.[2] Studies demonstrating a 3-42% proportion of family history in patients with AA and reports of a link between AA and human leucocyte antigen (HLA) class II antigens are two examples of the evidence supporting a genetic component to AA. Additionally, AA and families of persons with AA have been linked to a number of autoimmune disorders, including as thyroid disease and vitiligo, as well as hereditary problems (such as Down's syndrome).[3,4]

Alopecia areata (AA) includes three primary subtypes: patchy AA (localised hairless patches), alopecia totalis (the entire scalp is afflicted), and alopecia universalis (AA), which affects the entire body surface.[5] Other AA subtypes include diffuse form, sisaiph (central hair loss preserving the marginal hair line), and ophiasis (band-like alopecia in the occipital and temporal scalp), moderate (3 patches without alopecia totalis or universalis), and severe (alopecia totalis, universalis, and ophiasis) are the three categories under which the severity of AA can be classified. Dermoscopy is a non-invasive diagnostic method that is being used more frequently in dermatology practise to assess pigmented skin lesions as well as hair abnormalities.[6] We performed this study to assess cases of Alopecia areata in both genders.

MATERIALS AND METHODS

After considering the utility of the study and obtaining approval from ethical review committee, we selected ninety- two cases of Alopecia areata of both genders. Patients’ consent was obtained before starting the study. Data such as name, age, gender etc. was recorded. A thorough general physical examination, systemic examination and mucocutaneous examination was done. The site was selected and lesion was observed through the dermascope. Grading was performed and hair loss was recorded in all patients. The results were compiled and subjected for statistical analysis using Mann Whitney U test. P value less than 0.05 was set significant.

RESULTS

Table 1: Patients distribution

<table>
<thead>
<tr>
<th>Total</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>42 (45.6%)</td>
<td>50 (54.4%)</td>
</tr>
</tbody>
</table>
Out of 92 patients, 42 (45.6%) were males and 50 (54.4%) were females [Table 1].

Table 2: Assessment of dermascopic findings

<table>
<thead>
<tr>
<th>Dermascopic findings</th>
<th>Number</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black dots</td>
<td>24</td>
<td>0.72</td>
</tr>
<tr>
<td>Vallus hair</td>
<td>23</td>
<td></td>
</tr>
<tr>
<td>Yellow dots</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>Broken hair</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Exclamation mark</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>

Dermascopic findings showed black dots in 24, vallus hair in 23, yellow dots in 21, broken hair in 14 and exclamation mark in 10 patients. A non-significant difference was observed (P > 0.05) [Table 2].

Table 3: Grading of hair loss

<table>
<thead>
<tr>
<th>Grading</th>
<th>Percentage</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>4%</td>
<td></td>
</tr>
<tr>
<td>S2</td>
<td>8%</td>
<td></td>
</tr>
<tr>
<td>S3</td>
<td>14%</td>
<td></td>
</tr>
<tr>
<td>S4</td>
<td>20%</td>
<td></td>
</tr>
<tr>
<td>S5</td>
<td>54%</td>
<td>0.05</td>
</tr>
</tbody>
</table>

Hair loss grade S1 was seen in 4%, S2 in 8%, S3 in 14%, S4 in 20% and S5 in 54% patients. The difference was significant (P < 0.05) (Table II).

DISCUSSION

Regarding the pathophysiology of AA, various theories exist. The pathophysiology of AA has been linked to genetic, endocrine, autoimmune, psychiatric, and viral and bacterial infection. Family history has been shown to be one of the most significant risk factors for getting AA.[7] Since AA seems to be more common among those with a family history of the disease, it appears to have a hereditary component. Numerous genes have been linked to AA, and some gene variations may make people more susceptible to the disease.[8] Investigations are still being conducted to determine the precise genetic components and their underlying mechanisms. There is proof of an unbalanced immunological response in people with AA.[9] When the immune system's tolerance for hair follicles is compromised, an attack on these tissues results. T cells, natural killer cells, and dendritic cells are a few immune cells that are implicated in the inflammatory response within the damaged hair follicles.[10] Signalling chemicals called cytokines control immunological reactions. A change in the production and activity of certain cytokines is present in AA.[11] In particular, pro-inflammatory cytokines including tumour necrosis factor-alpha (TNF-alpha), interleukin-1 (IL-1), and interferon-gamma (IFN-gamma) are thought to contribute to inflammation and hair follicle destruction. Hair follicles go through a process known as miniaturisation as a result of the immunological response.[12] As a result, the afflicted follicles shrink in size, create hair that is thinner and shorter, and finally go dormant. The ability of the miniaturised follicles to renew and produce hair explains the possibility of hair development in AA.[13,14] We performed this study to assess cases of Alopecia areata in both genders.

Our results revealed that out of 92 patients, 42 (45.6%) were males and 50 (54.4%) were females. Dermascopic findings showed black dots in 24, vallus hair in 23, yellow dots in 21, broken hair in 14 and exclamation mark in 10 patients. We observed that hair loss grade S1 was seen in 4%, S2 in 8%, S3 in 14%, S4 in 20% and S5 in 54% patients. The difference was significant (P < 0.05) (Table II).
hypothyroidism are examples of frequent comorbid illnesses.

A standardised psychiatric assessment was given to 31 alopecia areata patients. A lifetime diagnosis of mental illness was present in 74% of people. The high lifetime prevalence rates of major depression (39%) and generalised anxiety disorder (39%), in particular, are remarkable. Additionally, patients reported higher prevalence of anxiety disorders (58%), affective disorders (35%), and drug use disorders (35%), all of which are mental diseases, in first-degree relatives. Generalised anxiety disorder diagnoses were more prevalent in patients with patchy alopecia areata. Major depression and any factor identifying the history of alopecia areata were not linked. Alopecia areata and potential connections between psychiatric diseases are examined.[18]

CONCLUSION

Most common dermoscopic findings in Alopecia areata patients was vallus hair and yellow dots.

REFERENCES