Alopecia Areata (AA) is an autoimmune condition that attacks the hair follicles, causing non-scarring hair loss. A systemic review of the epidemiology of AA indicated a similar worldwide lifetime incidence of around 2%. Some smaller studies indicate a slight female-to-male gender bias, but this may be due to higher female concern regarding hair loss and subsequent treatment [1].

AA typically presents as smooth, sharply demarcated, round patches of hair loss without atrophy with “exclamation point hairs” observed on the periphery of the patches [2]. Special designations of the disease include alopecia universalis (AU) (total body hair loss), alopecia totalis (AT) (total scalp hair loss) or alopecia in an ophiasis pattern (band-like hair loss on the temporal and occipital scalp. Less common variants include the diffuse variant with widespread thinning of hair across the scalp or the reticular pattern with recurrent hair loss in one area and spontaneous hair regrowth in another. Ophiasis inversus causes band-like hair loss in the frontoparietotemporal area [3].

Several forms of systemic corticosteroids have been tried for the treatment of AA; however, their efficacy and recommended regimens and doses are controversial [4]. Cyclosporine can reduce peri-follicular lymphocytic infiltrates and also appears to be effective for the treatment of AA. Betamethasone minipulse therapy has been reported to show equally good results and fewer side effects than daily therapy, it is regarded as a relatively safe and effective therapeutic option for the treatment of AA [5]. The present study compared cyclosporine and betamethasone minipulse therapy as treatments for AA.

Materials & Methods
The present study comprised of 56 cases of Alopecia Areata of both genders. All were informed regarding the study and their consent was obtained. Demographic profile of patients such as name, age, gender, occupation etc. was recorded. A thorough clinical examination was performed. Complete blood count, serum electrolytes, glucose, lipid profile, uric acid and creatinine were closely observed.
Type, family history and nail changes was recorded. Patients were divided into 2 groups of 28 each. Group I patients were prescribed oral cyclosporine and group II were given betamethasone minipulse therapy.

Patients’ self-assessments were graded on a 4-point scale as excellent, good, fair, or poor. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

Results

Table 1: Distribution of patients

| Groups | Group I | Group II |
|--------|---------|---------|
| Drug   | Cyclosporine | Betamethasone minipulse therapy. |
| M:F    | 15:13   | 14:14   |

Table I shows that there were 15 males and 13 females in group I and 14 males and 14 females in group II.

Table 2: Assessment of parameters

| Variables                        | Parameters                        | Group I | Group II | P value |
|----------------------------------|-----------------------------------|---------|----------|---------|
| Type                             | Alopecia areata                   | 12      | 8        | 0.02    |
|                                  | Alopecia totalis                  | 10      | 12       |         |
|                                  | Alopecia universalis              | 6       | 8        |         |
| Nail Changes                     | Yes                               | 12      | 7        | 0.01    |
|                                  | No                                | 16      | 21       |         |
| Family history                   | Yes                               | 5       | 6        | 0.05    |
|                                  | No                                | 23      | 22       |         |
| Extent of scalp hair loss        | Mild                              | 15      | 12       | 0.12    |
|                                  | Severe                            | 13      | 16       |         |

Table II, graph I shows that alopecia areata was seen in 12 in group I and 8 in group II, Alopecia totalis 10 in group I and 12 in group II and Alopecia universalis 6 in group I and 8 in group II. Nail changes were seen in 12 in group I and 7 in group II, family history was present in 5 in group I and 6 in group II and extent of hair loss was mild in 15 and 12 in group I and II respectively, severe in 13 and 16 in group I and II respectively. The difference was non- significant (P > 0.05).

Table 3: Assessment of treatment response

| Groups | Response | Excellent | Good | Fair | Poor |
|--------|----------|-----------|------|------|------|
|        | Mild     | 3         | 8    | 2    | 2    |
|        | Severe   | 5         | 4    | 3    | 1    |
|        | Total    | 8         | 12   | 5    | 3    |

|        | Mild     | 1         | 3    | 5    | 2    |
|        | Severe   | 3         | 3    | 7    | 4    |
|        | Total    | 4         | 6    | 12   | 6    |

Table III, graph II shows that 8 cases in group I and 4 in group II had excellent response, 12 in group I and 6 in group II had good, 5 in group I and 12 in group III had fair and 3 in group I and 6 in group II had poor response.
Alopecia areata (AA) is a complex autoimmune condition that causes nonscarring hair loss. It typically presents with sharply demarcated round patches of hair loss and may present at any age. In this article, we review the epidemiology, clinical features, pathogenesis, and new treatment options of AA, with a focus on the immunologic mechanism underlying the treatment. Nail abnormalities are associated with the disease with an incidence estimated between 7% and 66%. Most frequently, nail pitting is observed, although AA is also associated with trachyonychia, Beau’s lines, onychorrhexis, nail thinning or thickening, onychomadesis, punctate or transverse leukonychia, red spot lunulae, and koilonychia.

Other commonly associated conditions are thyroid disease (8%–28%), vitiligo (1.8%–16%) and atopy (1%–52%). Other skin conditions that may be confused with AA include traction alopecia, temporal triangular alopecia, androgenic alopecia, trichotillomania, lichen planus, tinea capitis, secondary syphilis, pressure-induced alopecia, aplasia cutis, chemotherapy-induced alopecia, telogen effluvium, and the many forms of cicatricial alopecia.

A variety of treatment modalities for alopecia areata (AA) are available, including topical, intralesional, and systemic steroids; topical immunotherapy; anthralin; minoxidil; photochemotherapy; and systemic agents such as cyclosporine, methotrexate, sulfasalazine, and biologics. However, no definitive therapy, and particularly no definite systemic treatment, currently exists for AA. The present study compared cyclosporine and betamethasone minipulse therapy as treatments for AA.

In present study, there were 15 males and 13 females in group I and 14 males and 14 females in group II. Jang et al. assessed and compared cyclosporine and betamethasone minipulse therapy as treatments for AA in 88 patients who received at least 3 months of oral cyclosporine (n=51) or betamethasone minipulse therapy (n=37) for AA. Patients with ≥50% of terminal hair regrowth in the alopecic area were considered responders. The responder of the cyclosporine group was 54.9% and that of the betamethasone minipulse group was 37.8%. In the cyclosporine group, patients with mild AA were found to respond better to the treatment. Based on the patient self-assessments, 70.6% of patients in the cyclosporine group and 43.2% of patients in the betamethasone minipulse group rated their hair regrowth as excellent or good. Side effects were less frequent in the cyclosporine group.

We found that Alopecia areata was seen in 12 in group I and 8 in group II. Alopecia totalis 10 in group I and 12 in group II and Alopecia universalis 6 in group I and 8 in group II. Nail changes were seen in 12 in group I and 7 in group II, family history was present in 5 in group I and 6 in group II and extent of hair loss was mild in 15 and 12 in group I and II respectively, severe in 13 and 16 in group I and II respectively. Cyclosporine is an immunosuppressive agent that inhibits helper T-cell activation and suppresses interferon gamma production. For these reasons, cyclosporine has been used alone or in conjunction with corticosteroids to treat AA. Even though it has various adverse effects (especially nephrotoxicity, immune suppression, and hypertension) and a high relapse rate, the reported efficacy of oral cyclosporine for the treatment of AA has ranged from 25% in some trials to 76.7% in others, when combined with methylprednisolone.

The prognosis of the disease is unpredictable. Current data suggest 34%–50% of patients recover within 1 year, while 14%–25% of patients will progress to AT or AU, at which point patients rarely fully recover.

Conclusion

In present study it has been concluded that oral cyclosporine found to be better than betamethasone minipulse therapy in treatment of cases of AA.

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