Alopecia areata: causes, types and symptoms

Alopecia areata can affect any hair-bearing area and can also involve nails. A peribulbar lymphocytic infiltrate in a ‘ swarm of bees’ pattern is characteristic of the acute stage of the disease leading to a dystrophic anagen phase. There may also be increased psychiatric morbidity in patients with alopecia areata. Ikeda (2) classified alopecia areata into four types including the common type (81%), the atopic type (10%), the autoimmune type (5%) and the prehypertensive type (4%)

The course of the disease is unpredictable and the response to treatment is variable. The various treatment modalities used can be classified into topical and systemic therapies. The topical therapy includes intralesional corticosteroid, topical corticosteroids, minoxidil, anthralin and topical immunotherapy in the form of diphenylcyclopropenone (DPCP) and squaric acid dibutylester (SADBE). The systemic therapy includes systemic corticosteroids and photochemotherapy. Cyclosporine, methotrexate, sulphasalazine and biologics like etanercept, efalizumab, adalimumab and infliximab have been used with limited success. Intralesional corticosteroids are the treatment of choice for adults with less than 50% of scalp area involvement.

The sensitivity of picking up clinical response to treatment by a clinical examination is very variable and has interindividual variation. Dermoscopy is a noninvasive diagnostic tool which visualizes subtle patterns of skin lesions not normally visible to the unaided eye. It is performed by a hand held or a video dermoscope equipped with lenses that currently allow magnifications ranging from 10 to 1000, the images obtained can be visualized on a monitor

and stored using specific software on a personal computer, to identify and compare changes over time.(3)

The characteristic features of alopecia areata on dermoscopy (4) are yellow dots, black dots, broken hair, vellus hair and tapering or exclamation mark hair. After therapy there is a decrease in the number of these characteristic findings. On the other hand, the presence of thin and unpigmented ‘ vellus hair’ within the patch, and evidence of transformation of vellus hair into terminal hair, appearing as increased proximal shaft thickness and pigmentation, are characteristic of remitting disease and indicative of a response to treatment.

The present study is being undertaken to evaluate the efficacy of intralesional triamcinolone acetonide in the treatment of alopecia areata and to assess its local and systemic side effects. Dermoscopy has been used to identify signs of early clinical response to the chosen therapeutic regimen. The useful markers to assess the severity of alopecia areata on dermoscopy are the black dots, yellow dots, broken hairs or dystrophic hair, tapering or exclamation mark hair and short vellus hairs. Previous reports have suggested that the severity of alopecia areata is an important prognostic factor. Therefore, dermoscopic examination of patches of alopecia areata may provide predictors of the response to therapy and can also be utilized for monitoring response to any prescribed regimen.

Definition

Alopecia areata is an autoimmune disease of uncertain etiology that involves the hair follicle and sometimes the nail and is usually reversible. Although

autoimmune, genetic and environmental factors have been implicated but the exact pathogenesis is yet to be elucidated.

History

Hippocrates first used the term alopecia which literally means ‘ fox’s disease’. The characteristics of alopecia areata were first described by Cornelius Celsus in 30 A. D., who described two forms of alopecia. The first he described as complete baldness occurring in people of all ages. The second he called ‘ ophiasis’, which literally means ‘ snake’, due to the pattern in which the hair loss spreads across the scalp and also suggested that ophiasis was only seen in children. Alopecia areata is sometimes also referred to as ‘ area celsi’ in tribute to Cornelius Celsus. Alopecia areata has been given many different names throughout history. However, the actual term ‘ alopecia areata’ was first used by Sauvages in his “ Nosologica Medica”, published in 1760 in Lyons, France.

From the beginning of 19th century there was considerable debate about the cause of alopecia areata. Two main hypotheses were put forward, one based on parasitic infection by Gruby in 1843 and Radcliffe-Crocker in 1903 and the other based on a nervous disorder by Von Barensrung in 1858. The parasitic hypothesis drew support from the pattern in which alopecia developed - expanding slowly in size just as a local infection would. Even more significant were the apparent epidemics of alopecia areata reported to occur in institutions such as orphanages and schools. However, many attempts to isolate an infective organism and to transfer alopecia areata by inoculation failed.

The initiation of alopecia areata by a nervous disorder, known as the trophoneurotic, neurotrophic or neuropathic hypothesis, eventually gained the support of most dermatologists of the time. This vague hypothesis could be supported by the apparently frequent clinical observations of emotional or physical stress and trauma that were associated with the onset of alopecia areata and often reported in the medical journals of that time. Emotional stress and physical damage were believed to adversely affect hair follicles via the nervous system and Joseph in 1886 showed that patchy hair loss could apparently be induced by cutting nerves in the necks of cats (it was later suggested that the hair loss was actually due to the cats scratching themselves). The idea circulated among dermatologists for many years because it was very difficult to fundamentally prove or disprove that alopecia areata was a nervous disorder. The hypothesis is still supported by some dermatologists today.

One of the more unusual variations on the neuropathic origin of alopecia areata was put forward by Jacquet in 1902 who suggested that alopecia areata was initiated by sources of nerve irritation such as defective and diseased teeth. Jacquet’s hypothesis was apparently confirmed by Decelle 1909, although Baily in 1910 showed dental disease to be equally frequent in people without alopecia areata. Eye strain was another suggested cause of alopecia areata by Kinnear 1939. With the start of the twentieth century, alopecia areata was known to be associated with disorders of the endocrine glands, particularly the thyroid. As such, some believed the underlying cause of alopecia areata was due to a hormone dysfunction. By the 1920’s most dermatologists had abandoned the parasitic theory of alopecia areata and

favoured variations on the trophoneurotic and endocrine theories – often combining the two.

Sufferers of alopecia areata were under extensive mental stress from fear that they would be suspected of having syphilis. Until the advent of antibiotics, syphilis was a widespread, contagious disease and it also often manifests itself by sudden, rapid loss of hair in well-defined patches, just like alopecia areata. Syphilis in the secondary stage can also affect finger nails. To further complicate the matter, some dermatologists suggested that alopecia areata could be found in increased association with syphilis – as distinct from the direct action of syphilis on hair follicles. Syphilis was believed to induce alopecia areata by the mental distress it created and its possible upset of the endocrine system. These clearly visible symptoms of syphilis were often confused with alopecia areata by the general population and resulted in social ostracism for the sufferer.

The early 20th century saw the development of another hypothesis of alopecia areata induction based on toxic agents. An unknown poison was believed to be introduced to the hair follicle via the blood system inducing hair loss. The sudden remission and relapse of alopecia areata and its action simultaneously over the body was believed to support the idea. Also in support, injection of thallium acetate (rat poison) was shown to induce alopecia areata like hair loss in some patients, with expression of exclamation mark hairs – a diagnostic feature of alopecia areata. However, the toxic origin of alopecia areata never gained widespread popularity against the neuropathic and endocrine hypothesis.

It is now widely believed that alopecia areata is an autoimmune disease. Even though studies more than 100 years old showed that alopecia areata affected hair follicles were invaded by inflammatory cells by Giovannini in 1891, the inflammatory autoimmune disease hypothesis did not become popular until the 1960’s. The idea was first proposed by Rothman in a discussion of a paper by Van Scott in 1958.

Treatment of alopecia areata by intradermal corticosteroid injections has been practised for many years. Kalkoff and Macher in 1958 were the first to have reported a series using hydrocortisone. Thereafter, Orentreich et al in1960 and Gombinger and Malkinson in 1961 reported the use of prednisolone and triamcinolone, and Porter and Burton in 1971 used triamcinolone acetonide and hexacetonide. Moynahan and Bowyer in 1965 and Verbov and Abell in 1970 reported the initial use of jet injection apparatuses in a number of conditions including alopecia areata.(6)

Epidemiology

Alopecia areata occurs worldwide and there is no racial or sex prelidiction. It is a common disease forming 0. 7% to 3. 8% of patients seen by dermatologists. (7) In the United States, alopecia areata was estimated to occur in 0. 1% to 0. 2% of the general population, with a lifetime risk of 1. 7%.(1) Sixty percent patients present with their first patch below 20 years of age.(8) One study suggests that 85. 5% of Asian patients with alopecia areata have disease onset before the age of 40 years.(9) The disease prevalence peaks between the second and the fourth decade of life. A family history is found in 5%-25% of patients.(10)

Natural History

Natural history that includes the severity, course and prognosis is highly unpredictable and it can be said that ‘ the only thing predictable about its course and prognosis is that it is unpredictable’. With the available information at present the spontaneous remission rates have ranged from 34% to 80% within one year and 15% to 25% patients progress to total loss of scalp hair (alopecia totalis) or loss of the entire scalp and body hair (alopecia universalis), of which only 10% eventually recover. (11, 12) It is a non-scarring alopecia and is reversible but it can be recurrent and abrupt and in long standing cases scarring can occur.

Etiology

Alopecia areata is a chronic, autoimmune, organ specific disease, probably mediated by autoreactive T cells, which affect hair follicles and sometimes the nails. The increased frequency of other autoimmune diseases favours the above postulation. Hair follicle autoantibodies are also found although it is unlikely that these are involved in the pathogenesis of the disease.

Genetic factors: Most reports describe the prevalence of positive family history to be in the range of 10 to 20% but it is believed that some mild cases may be overlooked or concealed and hence the actual figure may be greater. Price and Colombe (13) found a family history of alopecia areata was more common in those who had a disease onset before the age of 30 years (37% compared with 7. 1% in those with onset after 30 years). A study amongst monozygotic and dizygotic pairs found a concordance rate of 55% for monozygotic twins and no concordance amongst dizygotic twins.(14) The

genetic basis of inheritance appears to be multifactorial and polygenic and not a simple Mendelian pattern.

The strongest associations have been with major histocompatibility complex (MHC), particularly the Class II alleles HLA-DQB1*0301 and HLA-DRB1*1104 and the association is linked to chromosome 6p and few susceptibility loci on chromosomes 10, 16 and 18. (15, 16)

Atopy: Several studies have reported an association and also suggested an earlier age of onset and more severe disease in atopic individuals.(17, 18)

Autoimmunity: A statistically significant association between alopecia areata and Hashimoto’s thyroiditis, Addison’s disease and pernicious anemia has been reported. It is also associated with other autoimmune diseases like vitiligo, lichen planus, Sjogren’s syndrome, systemic lupus erythematosus, morphea, lichen sclerosus, pemphigus foliaceus, ulcerative colitis, myasthenia gravis, autoimmune haemolytic anemia, diabetes mellitus, autoimmune testicular and ovarian disease, Down’s syndrome (in which other autoimmune disorders are common) and autoimmune polyendocrinopathy-candidiasis-ectodermal dysplasia syndrome (also called as autoimmune polyglandular syndrome-1) which is an autosomal recessive disorder in which there are mutations in autoimmune regulator gene.(19)

There is also evidence of circulating organ specific antibodies against thyroid, gastric parietal cell, adrenal tissue, smooth muscle, testis and the ovaries.

The cells in the lower part of hair follicle have low or absent expression of MHC proteins and the loss of this ‘ immune privilege’ leading to induction of CD8+ T cell-mediated immunity against follicular melanocytes is hypothesized to be causative of alopecia areata. This explains the peribulbar lymphocytic infiltrate and also the sparing of white hair in the patch and hence leads to what is commonly called as the phenomena of “ overnight greying.”

Environmental factors

Infection: Prior notion of alopecia areata being due to infection directly or due to a remote focus of infection has a long history and was very popular till the middle of 20th century. Skinner et al (20) reported finding mRNA for cytomegalovirus in alopecia areata lesions. Few reports of coexistence in husband and wife but many have refuted this and likened it to be a mere coincidence.

Stress: Is suggested to be an important precipitating factor and this also explains cures by sleep therapy, reassurance and suggestion therapy. Such patients may also have stress associated depression and the finding of elevated serotonin levels in such patients lends support to the theory.

It has been seen that there is aberrant expression of estrogen receptor -1 in the hair follicles of diseased mouse with alopecia areata. Corticotrophin releasing hormone (CRH) can induce mast cell differentiation from hair follicle mesenchyme and the CRH/receptor activity is seen to be high in alopecia areata skin.(21)

Diet: Iron deficiency has been postulated to modulate the hair loss in alopecia areata. The mechanism is by inhibiting the rate-limiting enzyme for DNA synthesis and hence it diminishes the proliferative capacity of hair follicle matrix cells.(22) It has also been seen that dietary soy intake increases the resistance to the development of alopecia areata. A study in a Japanese population living in Hawaii, where a Westernized non soy diet predominates, showed disproportionally higher alopecia areata incidence. (23)

Other factors that have been implicated include hormones, drugs, and vaccinations. These factors may increase or decrease susceptibility to the disease onset, pattern, severity, duration and response to treatment by modifying the physical and biochemical status of the immune system and hair follicles.(24)

Pathogenesis and pathology:

There are four key phases in the normal hair cycle which includes the anagen (growth) phase, the catagen (regression) phase, the telogen (resting) phase, and the exogen (controlled shedding phase). When the new hair cycle begins the old hair fiber is shed from the hair follicle in the exogen phase and hence this maintains the overall hair density of the scalp. If the exogen occurs before the anagen is renewed or there is a dystrophic anagen then this leads to a state called kenogen in which there is no hair fiber in the hair follicle.(24) Thus the patch of alopecia areata can be said to be in a state of kenogen. When the amount of inflammatory infiltrate around the hair follicle increases this can lead to miniaturization of the hair follicles and shortening of the hair cycle with rapid changes from anagen to telogen leading to the

formation of ‘ nanogen’ hair follicles which is an intermediate stage between terminal and vellus anagen.(25)

In the acute stage of the disease there is a “ swarm of bees” infiltration of CD4+ and CD8+ lymphocytes into the peribulbar space of anagen stage hair follicles and some penetration of lymphocytes to intrafollicular locations which leads to a state of ‘ dystrophic anagen’. This disrupts the ability of the hair follicle to produce hair fibres of sufficient length and integrity and the expelled hair fiber is not replaced by a fiber that can produce adequate scalp coverage, hence leading to alopecia.(25) Hair follicles are smaller than normal and do not develop beyond the Anagen 3-4 stage, where the actual hair shaft begins to form and return prematurely to telogen.

As more and more hair follicle move to telogen phase the amount of inflammation decreases. At this stage most of the inflammation is localized to the papillary dermis around the miniaturized hair follicles. In all stages of the disease, there can also be a diffuse infiltration of eosinophils and mast cells into the disease affected skin. There is no inflammatory infiltrate which is seen around the isthmus of the hair follicle which is the site for the stem cells. Thus the pathological location of the disease process saves the stem cells from destruction and makes it a reversible and non-scarring alopecia. Trichocytes in the in the hair bulb matrix undergoing early cortical differentiation show vacuolar degeneration and are also the predominant cell types showing aberrant class I and II MHC expression.(10)

Classification:

Ikeda’s classification (2)

The atopic type (10%) begins during childhood or adolescence and progresses slowly over many years with individual patches lasting more than one year. Ophiasis and reticular patterns are common and the chances of developing total alopecia are very high (30-75%).

The autoimmune type (5%), affects the middle aged, runs a prolonged and led to alopecia totalis in 10%-50%.

The prehypertensive type (4%) occurred in young adults whose one or both parents were hypertensive progressed faster and led to total alopecia in 40%. Reticular pattern is common.

The common type (81%) is the prototype of the fast progressive form of disease that affects adults aged between 20-40 years. No associated conditions and individual patches last less than 6 months and there is spontaneous regrowth occuring within 3 years. Alopecia totalis may develop in 5%-15%.

Based on the pattern of alopecia:

Restricted to the scalp

- Patchy
- Ophiasis
- Sisaphio
- Reticulate
- Diffuse
- Subtotal
- Alopecia totalis

Generalized

Alopecia universalis

Clinical features:

Alopecia areata may begin at any age but the disease incidence peaks between 20-40 years of age and has an equal sex incidence. The characteristic initial lesion is a well circumscribed, totally bald smooth patch in which the skin appears slightly reddened. The disease is asymptomatic but few patients may complain of itching and burning prior to the onset of the lesions. During the active phase of the disease short easily extractable broken hairs are seen at the margins of the bald patches which are known as exclamation mark hairs and hair pull test is positive.(26)

Subsequent course is highly unpredictable. The initial patch may regrow hair or it may increase in size and new patches may appear after a variable interval. The succeeding patches may become confluent. In some cases the initial hair loss is diffuse and total scalp denudation has been reported in 48 hours. Regrowth is initially of fine vellus unpigmented hair and later these assume their normal thickness and pigmentation. It is possible that regrowth may occur in one region while alopecia is extending in another region.(10)

Alopecia areata may affect any hair bearing skin but the scalp is involved in 90% of patients. The eyebrows and eyelashes may be associated with hair loss elsewhere or may be the only site affected. The term alopecia totalis (AT) is used when complete loss of all scalp hair occurs and alopecia universalis (AU) when there is loss of all body hair. About 5% of patients progress to AT/AU. A new variant has been described by Sato-Kawamura et

al (27) called as diffuse and total alopecia which has a favourable prognosis but has rapid progression and extensive involvement.

The disease process preferentially affects the pigmented hair and spares the white hair thus leading to the phenomena popularly known as the overnight greying of hair but this is a relative process as white hairs are also lost albeit less as compared to pigmented ones. Hair regrowth may be initially nonpigmented but later complete pigmentation occurs.

Nail involvement occurs in 10%-15% of patients in which the most characteristic feature is fine stippled pitting but sometimes there may also be trachyonychia, red or mottled lunulae, nail thinning and ridging, discoloration that includes longitudinally arranged punctate leuconychia, splitting, onychodystrophy and onycholysis may be seen.(26) Some studies have reported psychiatric diseases like mood disturbances and anxiety and ophthalmological findings like asymptomatic lens opacities and fundus changes.(28, 29)

Poor prognostic indicators:(10)

Early age of onset

Extensive scalp involvement (> 50% scalp)

Loss of eyebrows and eyelashes

Alopecia totalis or universalis

Recurrent episode

Patterns – ophiasis, sisaphio, reticular

Nail changes: Pits, onychodystrophy, onycholysis, anonychia

Associated systemic disorders: atopy, hypertension and connective tissue disease.

Associated genetic disorder: Down syndrome

Family history of alopecia areata

Macrophage migration inhibitory factor (MIF) -173*C gene

INVESTIGATIONS:

1. Trichogram/ hair pluck test:(30)

To perform the pluck test, hairs are taken from the specified sites on the fifth day after the last shampoo. The surrounding hairs are fixed with clips and 60-80 hairs are grasped with a hemostat covered with rubber. The hairs are plucked, twisting and lifting the hair shafts rapidly in the direction of immergence from the scalp. Hair shafts are then cut off 1cm above the root sheaths and roots are arranged side by side on a slide and then taped.

The anagen hair bulbs are seen as darkly pigmented triangular or delta-shaped bulbs with an angle to the hair shaft and there is presence of inner root sheath. The telogen hair is seen as less pigmented hair with club-shaped hair bulb and there is absence of inner root sheath. Anagen hairs are distinguished from the telogen hairs and anagen to telogen ratio is calculated.

Trichogram in alopecia areata reveals a mixed telogen-dystrophic pattern. Telogen hairs predominate in the slowly growing patches, whereas dystrophic anagen hair forms the majority in rapidly progressing disease.

2. Scalp biopsy:(10)

A peribulbar lymphocytic infiltrate in a “ swarm of bees” pattern is characteristic of the acute stage of the disease, in which the number of follicles is normal and many are in catagen or telogen. In the later stages, only a few lymphocytes or eosinophils are present in fibrous tracts and in a peribulbar location. Many follicles in early anagen stage are observed in this late stage and the actual number of hair follicles may be reduced.

3. Dermoscopy:

A dermoscope is a non-invasive diagnostic modality which can be used to visualize fine details of skin lesions and even subsurface skin lesions which are not visible to the naked eye. It is also called as skin surface microscope, epiluminescence microscope or an episcope. An advantage of their use is the storage of the results and their reproducibility. (3)

The history of dermoscopy:(31)

Skin surface microscopy began in Europe when in the year 1663, Kolhaus used a microscope for examining the small vessels in the nail fold. In 1878, Abbe described the use of immersion oil in light microscopy and this principle was transferred to skin surface microscopy by the German dermatologist, Unna, in 1893. He introduced the term ” diascopy” and described the use of immersion oil and a glass spatula for the interpretation

of lichen planus and for the evaluation of the infiltrate in lupus erythematosus.

The term “ dermatoscopy” was introduced in 1920 by the German dermatologist Johann Saphier, when he used a used a new diagnostic tool which resembled a binocular microscope with a built-in light source. The term “ dermoscopy” was introduced by Goldman from the United States when he used this new technique for the evaluation of pigmented lesions of the skin. In 1971, Rona MacKie had identified the advantage of surface microscopy for the improvement of preoperative diagnosis of pigmented skin lesions and for the differential diagnosis of benign versus malignant lesions. Dermoscopic patterns of pigmented skin lesions including melanoma were established and standardized in consensus conferences that were held in 1989 in Hamburg and 2001 in Rome.

Principle of dermoscopy: (3)

The basic principle is to transilluminate a lesion and then to study the same under a high magnification to visualize its subtle features. When light is incident on a skin surface it undergoes reflection, refraction, diffraction and absorption and the magnitude of each of these phenomena is influenced by physical properties of the skin. When light is reflected on a dry, scaly skin surface most of it is reflected back but when the same falls on a smooth, oily skin most of the light passes through it and reaches the deep dermis. Thus certain fluids are used to improve the translucency of the skin that includes oils (olive and mineral oil), liquid paraffin, glycerin and water. Hand-held dermoscope have the basic principles:

The refractive index of glass is almost similar to skin and when it is in contact with oil-applied skin, it further enhances the transillumination and hence visualization.

The application of a glass plate flattens the skin surface and provides an even surface for better examination.

Videodermoscopy represents evolution of dermoscopy and it is performed with video camera equipped with optic fibers and lenses that currently allow magnification ranging from 10X to 1000X, and images are visualized on a monitor and stored using specific software on personal computer.

Basic design of a dermoscope:(3)

The essential components include:

Achromatic lens: To achieve the desired magnification which ranges from 10X to 100X.

Inbuilt illuminating system: Various illuminating systems are used that include:

Halogen lamps emit yellow light which can alter the colour contrast of the lesions.

Light emitting diodes (LED): Used in Delta 20©, Dermlite©, provide high intensity and consume 70% less energy than the halogen lamps. The illumination provided can be adjusted by turning off a set of LEDs. Can also be designed to emit lights of different colors and hence wavelengths and this

can help in better visualization of skin as the penetration of skin is directly proportional to its wavelength.

Power supply: By batteries eg. Lithium ion battery or using rechargeable handles.

The types of dermoscopy instruments that can be used include:

Instuments without image capturing facility.

Instuments with image capturing facility.

Instuments with image capturing facility and analytical ability.

Technique: (3)

It can be done either by the non-contact or the contact technique.

In the contact technique the glass plate of the dermoscope comes in contact with the fluid applied on the lesion whereas in the non-contact technique, there is no contact of the lens with the skin. The cross-polarized lens absorbs all scattered light and hence allows only light in one plane to pass through. The advantage of a non-contact technique is that there is no nosocomial infection but this is eclipsed by poor resolution and decreased illumination.

The contact plates used are made mostly of silicon glass and can be graduated for measuring the size of the lesion. These contact plates should be sterilized by using either 2% glutaraldehyde or methylated spirit. It can be used for the diagnosis of melanocytic nevi, melanoma, lichen planus, dermatofibroma, cicatricial alopecia, seborrheic keratosis and to calculate

the follicular density in the donor area before follicular unit hair transplantation.

Dermoscopy of normal scalp:(32)

Dermoscopy of the scalp can be performed with or without interface solution, which is referred to as “ dry dermoscopy”. Dry dermoscopy is useful for observing tertiary structures of the skin, such as hairs, scaling and follicular hyperkeratosis. An interface solution (thermal water) is used to analyse follicular and interfollicular (vascular) patterns.

Dermoscopy of the normal scalp shows interfollicular simple red loops, and arborizing red lines, which represents the normal vascular patterns, and honeycomb pigmentation in sun exposed areas and in subjects with darker skin. Follicular units are easily identified and usually contain 1 to 4 hairs. In children, dermoscopy often shows ‘ dirty dots’ corresponding to dust particles retained in the scalp. This feature is not observed in adolescents or adults as sebaceous secretions prevent particle deposition.

Dermoscopic findings in alopecia areata:

The characteristic findings are yellow dots, black dots, broken hair, tapering hairs corresponding to exclamation mark hair and regrowing vellus hair.

Yellow dots:

They are due to dilatation of the affected follicular infundibulum with keratinous material or sebum. (4) They vary in size, shape and colour. They may be round or polycyclic, yellow to pink.(33) They may be devoid of hair or contain miniaturized regrowing hair. They represent active and progressive

disease. Although yellow dots are seen in androgenetic alopecia, female pattern of androgenetic alopecia, trichotillomania and discoid lupus erythematosus, the number of yellow dots is limited in these conditions as compared to alopecia areata, which shows numerous yellow dots and is its characteristic feature.(34)

The incidence of yellow dots reported in the study by Inui et al (4) was 191 of 300 patients and in the study by Mane et al it was 81. 8%.(35) It is speculated that this may be the result of yellowish skin colour of Asian patients. Another possible reason may be the different devices used: a handheld dermoscope (DermLite® II pro) in the study by Inui et al vs. videodermoscopy and a handheld dermoscope by Ross et al (33) and only hand held dermoscope by Mane et al.(35)

Black dots:

They are remnants of exclamation hair and broken hair. They represent pigmented hairs broken or destroyed at the scalp level. They provide a sensitive marker of disease activity and disease severity.(4) The black dots of alopecia areata are characteristic of black haired individuals, including Asians, and these findings have not been used for the diagnosis of alopecia areata in white population. This feature may be attributed not only to hair colour but also to cuticle resistance. Takahashi et al (36) reported that Asian hair cuticles fall as large pieces while keeping their original shape under extension stress, whereas hair cuticles of white populations tend to collapse to form small fragments. They are also observed in dissecti