Alopecia

Synonym – baldness
Loss of hair is a common complaint that baffles dermatologists b’cos of its cause is often difficult to pin point, its poor response to treatment & the anxious state of the patients mind. Alopecia leads to some amount of stress due to diminished body image satisfaction.

Hair is a cutaneous appendage typical of mammalian skin. Though originally evolved as a protective coat, it has lost this functional value in human except probably over the scalp. Thus man is one of the least hairy mammals with most body hairs being fine and delicate. Although hair has lost its biologic significance its cosmetic & esthetic importance has increased. Too much body hair in women and not enough in men can cause considerable anxiety.

Hair density is maximum at birth & gradually decreases as the age advances. Hair is next to skin color as a marker of racial origins of an individuals.

Classification –

On the basis of damage-
1. Scarring or Cicatrical
2. Non- scarring or non- Cicatrical

Scarring alopecia – Patches of alopecia can not regrow even when the initiating pathology has ceased to be active.
- A loss of more than 100 hairs/ day is considered abnormal.
Alopecia may be Diffuse or Patchy
Diffuse Alopecia - -telogen effluvium
- Post-partum alopecia
- Neonatal alopecia
- Anagen effluvium
- Drug induced
- Associated with Systemic diseases
- Hair shaft defect
- Occasionaly alopecia areata

Patchy Alopecia – Alopecia areata
- Fungal , bacterial , Viral infections of the Scalp
Causes – I. NON – CICATRICAL

1. Localized - Alopecia areata
   - Syphilis
   - Tinea
   - Trichotillomania
   - Traction alopecia

2. Diffuse - a. congenital
   b. premature –
   1. Alopecia steatoidis
   2. Symptomatic – a. post febrile
   b. debility & emaciation
   c. syphilis
   d. During Lactation
   e. endocrine – Simmond’s disease, myxoderma
   f. Stress & Strains
   g. Drugs – Thallium acetate, hydantoin, cytotoxic drugs, hormones

3. Idiopathic - probable causes in such cases -
   - Masculinity
   - Heredity
   - Stress of civilization

   c. Senile

CICATRICAL

1. PRIMARY - Pseudo pelade
   Folluculitis decalvans

2. Secondary - Injury
   Flavus & kerion (tinea)
   Lupus vulgaris
   Lupus erythematous
   Gumma
   Leprosy
Dissecting cellulitis
X – ray burn, scleroderma

GENERAL DIAGNOSIS –
- Ex for atrophy or scarring – shining scalp, diminution or absence of hair pores, loss of skin elasticity & wrinkling

If non- cicatrical – 1. Localized
2. Diffused

Atrophy – Wrinkling, thinning, loss of elasticity & normal shine of integument
Non- cicatrical – skin feels and looks normal but hair is lost it can grow if cause is removed or treated.
Cicatrical cases – hair never grow back.

Androgenic Alopecia
Synonym – Common Baldness, Pattern Alopecia
- Loss of hairs seen with increasing age in genetically predisposed individuals. Since physiological amounts of circulating androgens are needed to express this genetic trait it is called androgenic Alopecia.
- Polygenic pattern of transmission is more likely.
- In advanced Androgenic alopecia, sections from the Scalp resembles those from the face due to the presence of numerous tiny vellus follicles in the upper dermis with prominent sebaceous glands (Sebaceous Follicles)
- There is a gradual & progressive miniaturization of the hair of the affected areas.
- With successive hair cycles, the affected follicles have a progressively shorter anagen phase & become smaller & superficial, producing hairs that are shorter, thinner, lighter in colour.
  o Scalp thickness is reduced due to substantial thinning of the subcutaneous fat.
  o A trichogram reveals an increase in the proportion of follicles in telogen. The rate of growth of hair on the scalp as well as other body areas like axilla is also decreased.

Clinical feactures
- Also affects to females with equal frequency but in different pattern.
- The onset may be sudden wherein it may begin as telogen effluvium or incidious when the hair fails to regenerate after the telogen.
- Spread of progression and severity of baldness is variable but pattern is uniform in males, greaded by Hamilton into 8 stages.
- In women signs of androgenic alopecia is not easily detected. An early sign is widened central parting.
- Female androgenic Alopecia begins in the 20th but this may occur as early as the teens or as late as 30th.
❖ In the affected women the frontal and parietal hairline remains intact.
❖ There is diffuse loss of hair which may start as telogen effluvium. Later with further progress there is parietal thinning and in elderly diffuse thinning.
❖ Uncommonly in postmenopausal women it may progress to the male pattern alopecia.
❖ hair loss occurs due to end organ hyper reactivity rather than increased levels of androgens.
❖ Occurrence of PCOD (hyperandrogenic state) in women with marked androgenic alopecia is common.

Rx – Reassurance
Men – goal is to prevent further hair thinning and to increase coverage of the scalp by reserving the miniaturization.
Minoxidil 2% or 5%
Finasteride 1 mg/day
In severe graded rather than medical, surgical line of Rx is recommended.
Women - Minoxidil 2% or 5%
Finasteride 1 mg/day
Surgery - >30 yrs
Hair transplantation – based on the principle of donor dominance.
Scalp reduction – Hairy skin is expanded.
Other – Alternative hair patterning plans
   Temporoparieto – occipital flap
   Strip grafts

Diffuse alopecia –
Telogen effluvium –
❖ Loss of telogen hair that occurs 2-4 months after an acute systemic stressful episode.
❖ Typical reaction pattern of the follicle is to ‘go into a shell’ by reverting back to their inactive stage i.e. telogen. The situation under which this phenomenon occurs were collectively termed as ‘Telogen effluvium’
❖ In an iron deficiency anaemia follicles fails to regenerate following a normal telogen, leading to diffuse hair thinning in absence of any obvious hair shedding.

Stress body conditions – febrile states - malaria, typhoid, TB, >39\(^\circ\)c and recurent
   o difficult labour
   o major accidents
   o surgical trauma
   o haemorrhage
   o severe emotional stress
   o starvation
- crash dieting
- leukemia
- lymphoma
- severe liver or kidney dysfunction
  - Normal scalp has 13% of its follicle in telogen. In telogen effluvium, this population rises to 20% but occasionally >25% and rarely >50%.
  - TE never results in total alopecia and is always diffuse.
  - The hair growth within 6 months but in severe cases regrowth may be incomplete.
  - TE may occur from - Early anagen release
  - Delayed anagen release
  - Short anagen cycle

**Postpartum Alopecia** –
Loss of hairs occurs 1–3 months after parturition. There are the hairs that were retained during pregnancy due to the circulating high oestrogen loads that are withdrawn after delivery.

**Neonatal alopecia**
Hairs present at birth are lost within a few weeks. Probably due to a sudden fall of circulating estrogens leading to loss of Telogen hairs.

**Alopecia due to endocrine disorders** –
- hypothyroidism
- hyperthyroidism
- hypoparathyroidism
- Psedohypoparathyroidism
- Hypopituitarism
- Sheehan’s syndrome

**Drug induced alopecia**
- Cytotoxic agents – used for malignancy and immunosuppresision
- Hair loss after 4-6 days of drug administration
- Cyclophosphamide
- Methotrexate
- Actinomycin D
- Doxorubicin
- Vincristine
- Azathioprine and prednisonolone combination
Anticoagulants -
- heparin,
- heparinoids,
- caumarin,
- dextran
- warfarin

Antimitotic –
- Arsenic
- Gold
- Bismuth
- Borax
- Colchicines
- Levodopa
  - antithyroid drugs
  - oral contraceptive
  - cimetidine
  - danazol
  - bromocryptine
  - tamoxifen
  - octreotide
  - Anticholesterol agents -
    - antipsychotics
  - Acute vitaminosis A
  - chronic vitamine A overdose
  - Retinoids
  - Antihypertensives – beta blockers
  - AEC inhibitors
  - Antiepileptics
  - Antiretroviral drugs
  - NSAIDS
  - Immune modifiers
  - Environmental Chemicals

Nutritional Alopecia
- In protein energy malnutrition the hair growth slows, shaft diameter reduces & the hair lightens in color. In severity it becomes brittle and causes alopecia.
  - Essential fatty acid deficiency developing on long term total parenteral alimentation.
  - Malabsorption syndromes and pancreatic disease.
Diffuse non scarring alopecia of other causes

Secondary and tertiary syphilis
SLE
Dermatomyositis
Systemic scelorosis
mixed connective tissue disorders
Sjogren’s syndrome
Cirrhosis of liver
Renal failure
Malignancies
Myotonic dystrophy

ALOPESIA AREATA
It is a very common reversible condition characterised by patchy loss of hair without atrophy in practice of dermatology & incidence is on increase.

Clinical features
1. It affects selectively the scalp & beard region though it may also affect other hairy regions also.
2. Patch – smooth, well defined, round/oval, slightly depressed below the surface. occasionally paraesthesia or mild erythema.
3. Can occur at any age but usually in young adults & children
4. The disorder is characterized by round or oval circumscribed patches of alopecia without any signs of inflammation, scarring, or atrophy.
5. The integument is shiny, slightly thin & depressed.
6. The hair pores are visible.
7. The onset is usually sudden, one or more patches may develop at the same time, by peripheral extension & confluence, irregular areas may be formed.
8. Hair is loose at periphery in a spreading, active patch but it is firmly attached in stationary one.
9. Severity, course and prognosis is unpredictable.
10. In a patch where the hair is tending to grow back it is typically in a shape of an exclamation mark. An exclamation mark hair has a thin lower portion and a thick stump. This is because newly growing hair is thin and portion of old hair which has broken off is thick. There are no subjective symptoms. Free ends are splayed giving ‘frayed rope’ appearance.
11. It may become widespread & lead to total loss of hair on the scalp ( alopecia totalis ). The initial patch may regrow in few months may enlarge slowly or new patches may appear after 3-6 wks.
12. In alopecia universalis case even hair from eyebrows, eyelashes, beard, region and all the hairy portions of the body may fall.
13. The course is slow & variable. The hair usually grows back in two months to two years. The new hair is usually first downy & then grey. It takes time to come back to normal.
14. In a bad case hair may not grow back & eventually atrophy of the skin may ensure.
15. In a moderately case new hair may stay grey.
16. Alopecia areata may be accompanied by shedding or atrophy of nails.

**ETIOLOGY** –
unknown cause
1. result of acute physical or emotional stress in a sensitive individual (trophoneurosis) – majority of cases
2. No infective micro organism or viruses.
   Toxins from sore throat or any septic focus may be the cause in some cases.
3. Auto immune mechanism as by chemicals, drugs, infections.
4. heredity
5. psychologic factors

Classification – based
1. atopic type – begins during childhood or adolescence and progresses slowly over many years with individual patches lasting > 1 yr.
   - ophtiasic and reticular pattern are common.
   - chances of developing total alopecia is very high.
2. Autoimmune type – affects middle aged, runs a prolonged course and leads to alopecia totalis.
3. Prehypertensive type – occurs in young adults whose one or both parents are hypertensive. Progresses faster and leads to total alopecia.
   Free of associated diseases.
   Individual patches last for less than 6 months with spontaneous regrowth occurring within 3 yrs.

Diff. diagnosis –
1. Androgenic alopecia
2. early Psedopelade
3. Traumatic alopecia
   1. Tinea captis –
   2. Syphilis –
   3. Trichotillomania
Pathogenesis & pathodynamics –
- severity is in the center
- periphery – impaired keratinization & shaft breakage. ‘Exclamation mark hair’

Prognosis –
1. Age – outlook is better in young patients. Worst in children when associated with atrophy or mongolism.
2. duration – shorter duration – better prognosis
   Old standing – hairs may not grow back.
   Repeated attacks & recurrence - bad prognosis.
3. size – smaller patch – better
4. exclamation mark hair - signifies recovery
   Firmly implanted hair at periphery – stationary
   Loose hair – at periphery – still active
5. General health of pt & other tropic / auto immune diseases.
6. poor prognosis – AT and AU
   ->50% of scalp involvement
   - Ophiastic or reticular pattern.
   - B/L – loss of eyebrows & eyelashes
   - Multiple patches.

Diagnosis –
1. Trichogram
2. Presence of lymphocytes, eosinophils in peribulbar location.

Treatment –
Large number of treatment modalities for AA speaks of their lack of adequate efficacy.
1. reassurance
2. Improving general health with diet, tonics etc.
3. removing active causes like nervous or physical strain, septic focus etc
   - avoiding use of chemicals & junk foods
   - Quiet holiday in congenial environment is beneficial.
4. Symptomatic - a. internal - sedatives in nervous patients with active disease
   - Stimulants like orabolin (p), dianabol (p)
   - ACTH / steroids in resistant widespread cases.
   - levamisole 150 mg once a week for 6- 8 wks
   - Andropogon muricatus & Withania somnifera herbs are benificial.
   B. external - steroid cream like flucort (p) in active phase.
   - Only after becoming stationary – local stimulants should be used like
    -10% cantharidine solution
- tincture K5 (p)
- Alopen (p)
- Ammi majus oil
- Tincture iodi mitis
- U.V.R.

- Local infiltration with hydrocortisone, triamcinolone useful in resistant Cases.

5. steroids - e.g. – Triamicinolone acetonide.
   Systemic steroids to check spread.

6. Topical irritants – e.g.- croton oil
   - 10% sodium lauryl sulfate & liquid
   - Anthralin

7. Contact sensitizers - 2% dinitrochlorobenzene

8. Minoxidil

9. PUVA therapy

10. Immunomodulators – Isoprinosine 1 gm q.i.d.
    Cyclosporine

11. poor prognosis – wig can be used.

12. hair weaving as an potion to those with stable extensive but not total AA

Although it is comforting to know that a large majority of cases regrow even after assurance it is equally frustrating to note that most therapies seems to only hasten what ultimately would be a spontaneous remission.

Traction alopecia
- A characteristic pattern of hair loss is seen in Rajasthani females & Skikh males who tie their hairs in traditional knots & apply excessive traction in the process.
- The frontal hair line which is normally sharp becomes unsightly, irregular & receding.
- Stumps of broken hair with mild erythema & inflammation around the follicular opening can be seen.
- Hair population is thin in there areas.
- Skin is not atrophic but if traction continues then cicatrisation may follow.

Rx – avoid traction

CONGENITAL ALOPESIA

- It is very rare
- Hair may not be present at birth & will not grow at all later.
- In some cases, hair may be poorly formed or downy, growing sparsely.
- In others, a good growth may be present at birth but later may fall & will not grow back.
- Congenital alopecia may occur alone or be accompanied by other congenital ectodermal defects of skin, nails, teeth etc.

ALOPESIA STEATOIDES
- affects young adults
- Characterized by – seborrhoeic diathesis
  - Constant dandruff like scaling from scalps A/W moderate itching.
  - Short, poorly formed hair from vertex & temples.
- There may or may not be frank seborrhoeic dermatitis.
- Condition is progressive rather than resistant to treatment.
- Latter consists of therapy for pityriasis capitis & mild stimulant topical preparation as mentioned for alopecia areata.
- Scalp should be washed with selsun suspension (p) or cetavlon lotion (p) followed by rubbing in flucort (p) solution.
- For females - tincture K5 (p) applied twice a week or acid salicylic, sulphur, prednisolone cream.

DIOPATHIC PREMATURE ALOPESIA
Synonym – Masculine alopecia
- Common in young male adults.
- Gradual onset starts from 20-40 yrs of age.
- starts with a receding hair margin & with widening of forehead thinning of hair which becomes atrophic & lusterless later from vertex, frontal region, temples & even the whole of the scalp may fall completely. The distribution of alopecia is symmetrical. A few lanugo hairs may keep on growing but these lack quality for full growth & vigor.
- Exact cause is unknown. In most cases there is heredo- familial predisposition to alopecia.
- Usually male, intelligent & sedentary type.
- Eunuchs do not get bald proves that androgens are greatly responsible.
- acc. To some – disease of civilization
- artificial menopause produces similar alopecia.
Rx – unsatisfactory
- Oestrogen, prednisolone ointment rubbed locally
- Minoxide lotion 2%
- Hair transplantation

SENILE ALOPESIA
Characterized by symmetrical thinning or loss of hair in old age giving the pt. a dignified look. The vertex, frontal region, temples, or even the occipital region may be involved.
- Atrophic changes that take places with age are also present in the integument.
- Causes responsible for idiopathic, premature alopecia may be contributory factor.
- Senile alopecia – after 45 – 50 yrs.
Rx – not satisfactory
Alopecia neoplastica
- Benign tumours
- Primary malignancies of scalp.

PRIMARY CICATRICAL ALOPESIA

Synonym – Pseudo – pelade
- Rare
- Characterised by ill defined round or oval areas of alopecia.
- Skin appears white, Shinning, atrophic or scarred without any sign of inflammation or subjective symptoms.
- Cicatrical alopecia patches appear like footprints in the snow across the scalp.
- Onset is insidious & the course variably fluctuating. The disease affects mainly young adults.
- M > F
- Idiopathic but - syphilis, focal sepsis, and local infection should be excluded.

Cicatrical Alopecia
- whenever the hair follicle is seriously damaged it is permanently lost and replaced by scar tissue. The resulting are grouped as scarring or Cicatricial alopecias.. medical therapy is futile except probably for stopping the progress.
- the scarring are differentiated from non scarring by the absence of follicular openings, incased wrinking & thin shiny , dry , depressed skin covered with telangictasis.
- Such alopesias occur due to defective development of follicles or scalp skin as also to some inherited diseases.
- Thses can be diagnosed on the basis of onset in early life, typical clinical feactures & other associated findings.
- Inflammatory are best divided into those with follicular inflammation & others with inflammation of interfollicular dermis affecting follicles.
- Conditions with primary foliculitis – with pustules
  - without pustules
  - follicular inflammation.

Investigation – punch biopsy
- relevant systemic examination.

Rx – Smaller patches may be excised & closed primarily.
  For large patches auto graffeting & scalp expansion technique is useful.
  Creative hair styling ., altering the curling or colour of hair, wig.
AYURVEDIC POINT OF VIEW

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  धावनं कफसम्भूते चैत्तुपुरे प्रस्यते।
  
  पैतिके च हितं दुःखं नवनीतान्तिं तथा।
  
  शिष्नाशिवालं यथी पैतिके धावनं मतम्।
  
  श्रृंगेणासेन श्रुतं श्रुतं वैवर्षसं तथा।
  
  सौरीरकसेनाधीर तिलानु शिष्वा प्रलेपनम्।
  
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प्रारम्भिक चेलनदलज्ञ खृतमेव निवारणम्
पैतीकानि च लिङ्गानि दृष्ट्वा दुर्धेन धावनम्
शीतलानि प्रदेयायति पैतिके विषयवे
धतुप्रश्नाणि च माणधीनां निशाविशालागृहधृतमकृष्ठम्
घूर्तेन युक्तेन जलने चिं शिरः प्रलेपे श्रति वाणम यथात्
पितकृते दोषाये च श्रेणे पतोलाक्रमकृत वा
तथा मलवचनः फलमेव पिष्ठवा घूर्तेन खण्डने प्रलेपनवच
प्रति संहिता ५३/३

शिरोयोग निदान

d्रुमातपनुषांकुंकीक्रियास्वप्नप्रजागः
उद्वेदिष्कृत्वामानिनिग्रहरोदनाः
अतथामुद्धाराते खृतमिभिवागः
उपश्रायुजाबाणः प्रतत्तेकार:
असात्मयस्तृधामभाष्यवेश शिरोगातः
जनवन्यामायय दोषः

निदानपूर्वकसम्प्राप्ति
रेमकुपानुगां पितं वातेन सह मूर्चितम्
प्रच्यवायति रोमाणि ततः श्रेष्ठा सश्रोणितः
रूपाणि रेमकृपासुतः ततो नवेयासबसवः
नधिदुल्लम खालितव रहेति च विभवाये

रेमकुपानुगां पितं वातेन सह मूर्चितम्
प्रच्यवायति रोमाणि ततः श्रेष्ठा सश्रोणितः
रेमकृपान् रूपाण्यवस्तु तेनायेयाससम्भवः
नधिदुल्लम रूपः च प्राहार्य च चापरे

सु.भ. १३, भाष. ६१/६

- रूक्ष सपाणुं वालापिताद्रकं सदहकम्
कफातिन्त भवेत दिनसंदेह तक्तात भांकं ब्रजिन्त तत्त
सतिशातेन सदृश जायथे सर्वस्वाक्षरणम् ॥

इन्द्रतुष्प - इन्द्र: + लुप्त
इन्द्र: - इन्द्रीति। इदि पारमेष्ट्रोऽयम् तस्मात् रन् प्रत्ययः। देवराजः। स सु अदिनिपुः।
लुप्त: - लुप + कः
चौर्यन्धनम्। लोत इति यवात्।
- इन्द्राण्य इन्द्रीलवणकेशानां लुप्त लोपो यस्मात्।
- श्रम्भुकेशञ्जम्यः।
- शिशीरार्धं।

पार्श्व - इन्द्रुत्तकः। केशान: इन्द्रुत्पः। (रा.)
इन्द्रुत्नधेः। (भू)

बालन्त रूप्येतु तत्सः पर्यायं कथानाम् ॥
- भोजः।
- तदन्तूलखमित्राः। खलसर्वयाच्य केषान।
इन्द्रुत्पः श्रम्भुणि भवति बालन्तिः शिशीराः।
- अदिनिपुः।
- इन्द्रुत्पः श्रम्भुणि भवति।
- कालिकः।

- खलित्वं पुष्पावेत न तु योषिताम्। तथा च विदेहः। "अत्यन्त्सुकमाण्यो रजो दृष्टं स्ववितं।
अव्यायंसता यस्मात्समात्। खलिति: स्त्रिया:। इति ॥
- तल्लण, सू.प्र. २३/३३-३४।

- रोमकपारण: पितामसिद्धिना हंदुलुप्तं। खलित्वं रूप्येतु तत्सः संजानात्मक।
विदेहः। "अव्यायसता यस्मात्समात्। खलिति: स्त्रिया। इति। अप्यामसिद्धिः।
अव्यायपरेण वातपिते न प्रकुप्येते, ततो न लोमप्रधव:। रजो विमुक्त्वा।
स्त्रोतीवर्षोधाभावावच्चूजुनानामपि पुनिर्विहो इति।
- न्या.च., सू.प्र. २३/३३-३४।

खलित्वः

खलित्व निदान

तेजोिनिलान्तः। सह केशभूमि दश्वाणु कुर्यात्। खलिति नस्य।
-च.चि.२६/१३२।

खलित्वपी जनपी शार्तं तत्र तु अद्भूत ॥
阿. ह. उ. २३/ २६

टिका

एवं एततुल्क्यम्। खलितरूपमिति। किंतु खलेत्रो,व्रमण केशानां शालो न त्विन्दुल्प इव सहसा ॥
- सा वातानिदिग्धाभा, पितास्वित्राग्निसिद्धृताम्
  कफाधनन्तरणापि यथास्वं निर्दिष्टेऽत् तवचि।
  दोषे: सर्वाङ्गूः सर्वरसाध्या सा नखप्रभा।
  दस्यानिनेव निलोमा सदाहा या च जावते। — अ. ह. उ. २३/ अ.
  खालित्याचिकित्सा —
  छागकृष्णसाहजनपुटदग्धं गजेन्द्र दत्तमसीलिप्तः।
  जावते सप्तग्रामत्वं खल्ल्यामपि कुल्लिताधिकारः। — च. द. कृष्ण. चि. /१०२

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