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Review

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Hair restoration surgery: pre-surgical considerations and pitfalls

Mingjuan Tan[1], Angeline Anning Yong[2], Etienne Cho Ee Wang[1,3]

[1]National Skin Centre, Singapore 308205, Singapore.
[2]Angeline Yong Dermatology, Gleneagles Medical Centre, Singapore 258499, Singapore.
[3]Skin Research Society Singapore, Singapore 138648, Singapore.

Correspondence to: Dr. Mingjuan Tan, National Skin Centre, 1 Mandalay Road, Singapore 308205, Singapore. E-mail: mtdermt@gmail.com

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Abstract

Hair restoration surgery is an increasingly common treatment for androgenetic alopecia and may also address other forms of non-scarring and scarring alopecia. To optimise surgical outcomes, it is crucial to consider key pre-surgical factors and avoid diagnostic pitfalls. This review summarises the diagnostic and differential diagnostic approaches to alopecias, as well as a checklist for holistic patient assessment before hair transplantation.

Keywords: Hair transplantation, hair transplant, hair restoration surgery, androgenetic alopecia, scarring alopecia, non-scarring alopecia, alopecia

INTRODUCTION

Hair restoration surgery or transplantation can be an effective treatment for non-scarring and scarring types of alopecia, particularly androgenetic alopecia (AGA), often complementing medical therapy. However, there are important considerations to evaluate before proceeding with hair transplantation, as these might render the patient unsuitable for the procedure and result in poor subjective or objective outcomes. Such considerations include a precise diagnosis of the type of alopecia and any co-morbidities that could impact the surgical results. In this review, we will discuss these essential diagnostic and differential diagnostic

elements to consider prior to hair transplant, with reference to current peer-reviewed literature.

SUMMARY OF HAIR RESTORATION/TRANSPLANTATION SURGERY

Hair transplantation is an outpatient surgical procedure performed predominantly under local and tumescent anaesthesia. Individual follicular units of one to four hair follicles are transplanted from a donor area, usually the occipital scalp, to the recipient area, typically the frontal hairline, mid-scalp and/or vertex in AGA.

Donor hair harvesting may be done via strip harvesting or follicular unit extraction. Strip harvesting allows for the safe removal of large numbers of hair follicles with minimal transection, but risks include potentially unpredictable wound healing and a larger scar. A trichophytic closure and minimal tension on the closure may help to minimise scarring risk. After removing a strip of hair-bearing scalp skin from the occipital donor site, it undergoes sectioning and microdissection into fine slivers and then further into individual follicular units, which are promptly placed in a holding solution (commonly chilled saline) before transplantation. The donor site is closed with sutures, which will be removed typically between 10-14 days post-op.

In follicular unit extraction or excision, individual single follicular units of 1-4 hair follicles are removed via small punch incisions. While this technique is more laborious, it allows for individual graft selection, and tends to leave only small 'white dot' scars, which are generally less perceptible but may appear more obvious if there is overharvesting. This also results in more rapid healing of the donor site as the individual punch incisions do not require any suturing and will heal within 5-7 days post-op, enabling more rapid recovery.

Post-procedure, a non-adhesive overnight dressing may be applied. A regular saline spray can be used to hydrate the recipient sites, which are examined on the second day post-transplant and adjusted for any dislodged grafts. Gentle hair washing can be resumed after this postoperative check. Avoidance of smoking, alcohol, sun exposure, exercise, and swimming is advised in the immediate post-transplant period of 1-2 weeks.

Transplanted hairs in the catagen phase and hair shafts are normally shed post-procedure, and the new anagen hair follicles begin to regrow after 3-6 months and are fully grown by 9-14 months.

Hair transplantation is most commonly performed for AGA with good results for this type of alopecia [Figure 1]. However, there is a lack of data on success rates, patient satisfaction, and complication rates for other types of non-scarring and scarring alopecia. Future research should focus on gathering real-world data on hair transplantation for these less-studied forms of alopecia.

DIAGNOSIS OF ALOPECIA

A correct diagnosis of the type of alopecia has implications on pre- and post-transplant care, the choice of transplantation technique, adjunctive pharmacological and non-pharmacological treatment, as well as the very decision to proceed with hair restoration surgery in the first place. In this section, we discuss some diagnostic considerations.

Non-scarring alopecias

Hair transplantation is most commonly performed to treat AGA in which there are usually high graft survival rates (~90%), in comparison to the much lower graft hair transplantation rates of around 40-50% in scarring alopecias[1,2]. Hair transplant is not routinely performed for other non-scarring alopecias. There are

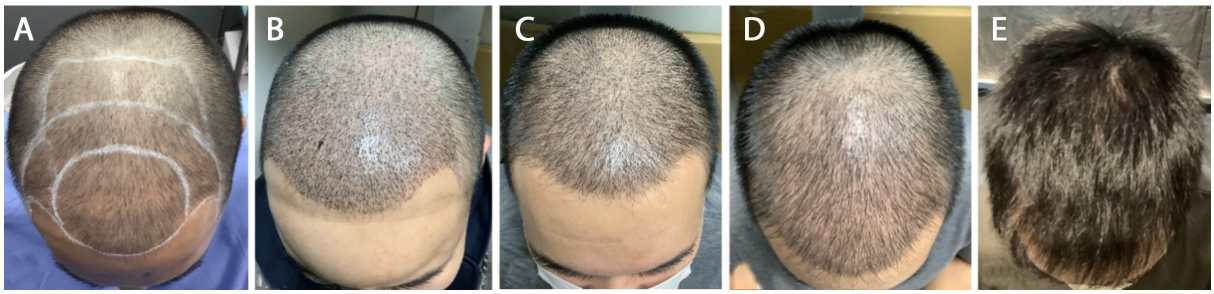

Figure 1. Hair transplantation is most commonly performed for AGA with good results; these photos show the progress of a patient from (A) pre-transplant with preoperative markings, (B) day 1 post-transplant, (C) 1 month post-transplant, (D) 4 months post-transplant, and (E) 6 months post-transplant, showing visible improvement in the frontotemporal hairline and vertex density. AGA: androgenetic alopecia.

a few case reports of successful hair transplant for alopecia areata with the transplanted area remaining quiescent post-transplant, but patients should be counselled on the risk of flare of this chronic condition, which can occur immediately or years after transplant[3]. Other non-scarring alopecias include telogen effluvium, which tends to self-resolve without the need for hair restoration surgery[4], or secondary alopecias, which typically respond to treatment of the causative condition. One should beware of other conditions that may mimic AGA, such as diffuse unpatterned alopecia, which does not respond well to hair transplant and is better treated with medical therapy[5]. The importance of considering such differential diagnoses is further discussed below.

Scarring alopecias

Hair transplantation yields unpredictable and often poor results in scarring alopecias, with no guarantee of good or permanent growth, given the risk of disease flares that may occur even years post-transplant[4]. Several authors suggest hair transplantation only after at least 2-5 years of stable disease[1]. Follicular unit extraction is preferred for scarring alopecias as it enables the selection of individual follicular units and typically results in less scarring. Hair restoration surgery has been utilised in various scarring alopecias including lichen planopilaris and frontal fibrosing alopecia, central centrifugal cicatricial alopecia, discoid lupus erythematous, folliculitis decalvans, and en coup de sabre[1]. A small review of 34 patients[1] reported transplanted hair growth in 76% of patients but did not take into account the graft uptake rate, which may be 20-50% or lower for some patients[4]. Furthermore, actual success rates of hair transplantation in scarring alopecias may be lower due to factors such as publication bias and inconsistent follow-up periods[1]. Given the higher risk of graft failure in scarring alopecias, patient expectations should be moderated, and treatment options such as non-invasive measures (e.g., medical therapy, cosmetic camouflage, wigs) discussed.

Inaccurate diagnosis or multiple pathologies

Several conditions can present similarly and mimic AGA, such as frontal fibrosing alopecia and diffuse unpatterned alopecia[5]. Misdiagnosed or undiagnosed alopecias may, unfortunately, be worsened by hair transplantation and other surgical procedures. This includes both non-scarring alopecias, as has been noted in patients with recurrence of alopecia areata triggered by hair restoration surgery[5], as well as scarring alopecias, such as patients who developed the first signs of lichen planopilaris or frontal fibrosing alopecia post hair transplant for presumed AGA[6]. In scarring alopecias, this may be at least partly due to koebnerization, the risk of which may potentially be reduced by treatments such as antimalarial therapy for discoid lupus, hence underscoring the importance of the correct diagnosis.

Furthermore, patients can have more than one condition, such as concomitant AGA and alopecia related to nutritional deficiencies, all conditions of which should be identified and stabilised pre-surgery.

DIFFERENTIAL DIAGNOSIS OF ALOPECIAS

Alopecias can be subdivided into scarring and non-scarring alopecias. Clinical features such as the pattern of alopecia and trichoscopy findings can point toward the diagnosis[7] and are summarised in Table 1. However, if the diagnosis is in doubt, one may consider seeking dermatology input, as well as performing diagnostic investigations such as a biopsy for histology (and direct immunofluorescence if conditions such as discoid lupus or lichen planopilaris are suspected) and laboratory investigations to exclude systemic diseases and infections.

Trichoscopy can often reveal the diagnosis in both scarring and non-scarring alopecias. In AGA, there is hair shaft diameter variability, affecting more than 20% of the hairs; brown halos around hair shafts (peripilar sign) may also be seen [Figure 2]. In alopecia areata, patients also have non-scarring alopecia on physical examination, but trichoscopy reveals black dots, exclamation point hairs, dystrophic hairs, and vellus-like hair shafts [Figure 3]. In lichen planopilaris, there are discrete white dots over scarred fibrotic tracts and perifollicular fibrosis, as well as peripilar casts and scaling [Figure 4]. Further trichoscopic findings of other alopecias are summarized in Table 1.

A scalp biopsy should be considered if conditions other than AGA are suspected, particularly mimics such as lichen planopilaris and frontal fibrosing alopecia, to exclude these diagnoses prior to consideration of hair transplant. Clues pointing to a need for biopsy and other investigations include unusual patterns of hair loss, hair loss without the hallmark pattern miniaturization of AGA, signs of scarring alopecia on physical and/or trichoscopic examination [Table 1], symptoms such as prominent scalp itch (which may suggest occult lichen planopilaris), or other signs of systemic illness, local infection and secondary causes of alopecia[5]. Biopsies should be taken from an active area to elicit specific findings that could pinpoint the diagnosis, particularly in scarring alopecias. Common biopsy findings in scarring alopecia include loss of follicular unit architecture, sebaceous glands and even hair follicle units, as well as perifollicular fibrosis, with other findings varying according to the condition [Table 1].

HOLISTIC ASSESSMENT OF PATIENT SUITABILITY FOR HAIR RESTORATION SURGERY

After making the correct diagnosis of the patient's hair loss condition, a comprehensive systems assessment of the patient should be performed, including relevant aspects of the patient's medical history, such as other medical conditions and drug allergies. A checklist for pre-transplant assessment is included in Annex 1 [Supplementary Material].

Medical conditions that may affect the decision for hair transplantation include:

Disease control

Both scarring and non-scarring alopecias should be well-controlled and stabilised prior to hair restoration surgery, including with pharmacological treatment. In particular, patients with AGA should already be on medical therapy (e.g., oral dihydrotestosterone blockers, topical/oral minoxidil) for an adequate duration before undergoing a hair transplant, although more evidence is needed on the optimal combination and duration of medical therapy pre-transplant, given that medications such as finasteride may take up to 1-2 years to reach peak efficacy[9,10]. Medical therapy is important for several reasons; firstly, it can potentially stabilise or stop the progression of hair loss, potentially allowing for deferment or even avoidance of transplant. Secondly, medical therapy can help to maintain patients' existing non-transplanted hair,

Table 1. Differential diagnosis of alopecias

	History and examination	Trichoscopic findings	Histopathology	Special considerations
Non-scarring alopecias				
Androgenetic alopecia (AGA)	• Gradual onset of progressive hair thinning in a pattern distribution • Patterns include male pattern (frontotemporal recession and vertex thinning) and female pattern (crown sparsity) • Occipital hair density typically preserved Patients may have family history of hair loss	• Hair shaft variability (anisotrichia) that usually spares the occipital region • Miniaturisation of hair follicles	• Normal total follicle number and no significant inflammation • Increased numbers and percentage of miniaturised and vellus hairs • Slightly increased telogen count	• Exclude and treat other causes of alopecia, which can occur concurrently • Be wary of fibrosing alopecia in a pattern distribution (FAPD) (if the temporal, parietal, and occipital areas are affected), the latter of which is better managed with medical therapy given the high risk of transplant failure and frequent lack of a good donor site[5]. Ensure stabilised on medications pre-transplant • Avoid transplanting patients who are too young or in the early stages of hair loss (e.g., before Norwood stage 3-4)[5] • Examine patients for miniaturisation in the donor area, which may limit harvesting and/or necessitate consideration of body hair harvesting[5] • Exclude endocrine abnormalities such as hyperandrogenism in women, particularly those with male pattern hair loss (features include recalcitrant acne, hirsutism, and infertility) • May potentially be associated with cardiovascular risk factors that may affect surgery risk, such as ischemic heart disease
Telogen effluvium (TE)	• Abrupt hair shedding • Preceded by physiological or emotional stress such as febrile illnesses, hospitalisations, rapid weight loss or childbirth • Typically self-resolves, although can evolve into chronic form Hair pull test shows telogen hairs	• No/minimal vellus hairs • Empty follicles	• Normal total number of hairs as well as terminal hairs • Telogen count >15-20%, but rarely >50% • No inflammation	• Careful history-taking to identify possible precipitants • Chronic telogen effluvium is a diagnosis of exclusion, and other causes of alopecia should be ruled out • Hair transplantation (HT) should be used rarely, with careful discussion with patient on expectations and outcomes • May mimic, unmask or occur concurrently with AGA • Telogen effluvium may occur post hair transplantation but tends to self-resolve
Alopecia areata (AA)	• Hair loss can be localised, patchy or diffuse • May have alopecia of other hair-bearing sites such as beard, eyebrows • May have nail pitting	• Exclamation mark hairs, black dots and broken hairs suggest active disease • Fine vellus hairs, usually depigmentated may be seen in chronic disease	• Peribulbar mononuclear cell infiltrate around terminal, anagen and catagen hair bulbs ("swarm of bees") • In chronic AA, most hairs are in catagen or telogen phases, and miniaturised, rapidly cycling nanogen hairs may be seen	• Diffuse AA may mimic AGA • Active AA is a contraindication to HT as the transplanted hair may be affected by AA • Surgery may precipitate recurrence of AA, the risk of which is reduced but not eliminated in quiescent disease[5]
Secondary non-scarring alopecias	• Differs according to condition • In traction alopecia, there is focal alopecia along the frontotemporal hairline • In trichotillomania, there are alopecic patches in bizarre configurations and distribution • In tinea capitis, there may be patchy alopecia with short broken hairs • In secondary syphilis, there is classically patchy 'moth-eaten' alopecia, but it may also mimic diffuse AA	• Differs according to the condition, such as short broken hairs growing at different lengths in trichotillomania	• Differs according to the condition, such as incomplete and distorted follicular anatomy in trichotillomania	• Exclude secondary causes such as medications (e.g., chemotherapy causing anagen effluvium), nutritional (e.g., iron deficiency) and endocrine factors (e.g., thyroid disorders), hair care practices (e.g., traction alopecia), cutaneous malignancies (e.g., basal cell carcinoma), and psychiatric diagnoses (e.g., trichotillomania) • Alopecia contributed to by chronic illness is common in women and a screening full blood count, thyroid panel and iron studies should be considered in women presenting with apparent AGA • Consider infective causes such as

				tinea capitis or syphilis, particularly in patients with risk factors Rarer causes include malignancy or haematologic disorder-associated alopecias such as follicular mucinosis ● Non-scarring alopecias may eventually lead to scarring alopecia as the final common end-point
Scarring alopecias				
Discoid lupus erythematosus (DLE)	● Well-demarcated, scaly, erythematous, plaques with follicular plugging on sun-exposed sites, which are most active centrally ● Late lesions are atrophic and dyspigmented with peripheral hyperpigmentation, telangiectasias, and loss of follicular ostia ● Symptoms include hair fall, itch or burning, tenderness, photosensitivity	● Large yellow dots (follicular keratotic plugging) ● Prominent arborizing blood vessels	● Vacuolar interface changes at follicular epithelium +/- epidermis ● Increased dermal mucin ● Direct immunofluorescence (DIF) shows granular IgG, C3 and/or IgM/IgA deposits at the dermal-epidermal junction or follicular epithelial-dermal junction	● Exclude systemic lupus erythematosus through history, systems examination and laboratory tests (e.g., lupus antibodies, full blood count, renal function) ● HT may be performed on chronic, burnt-out, and quiescent patches of alopecia that have been stable for at least 2 years
Lichen planopilaris (LPP)	● Symptoms include itch, burning, tenderness, hair shedding ● Active patches show an expanding rim of activity with erythematous perifollicular papules with hyperkeratotic follicular spines, may appear violaceous ● Ill-defined atrophic patches with decreased follicular orifices ● Variants include: - Classic form: patches may be anywhere on the scalp - Frontal fibrosing alopecia (FFA): progressive band-like frontotemporal recession with atrophic, ill-defined scarring alopecia; loss of eyebrows can be an early sign - Graham-Little Syndrome: Triad of progressive scarring scalp alopecia, nonscarring alopecia of axillary and pubic hair, and widespread keratosis pilaris-like follicular papules - Lichen planopilaris diffuse pattern: diffusely distributed scarring alopecia patches	● Perifollicular scaling corresponds to disease activity, with tubular perifollicular scaling ● Elongated linear blood vessels, violaceous inter or perifollicular areas ● End-stage LPP may exhibit fibrotic white dots with a lack of follicular openings ● In FFA, "lonely hairs" may be seen	● Lichenoid mononuclear cell infiltrate at follicular epithelial-dermal junction, with vacuolar interface alteration and hypergranulosis within affected infundibula ● Interface changes may include colloid or civatte bodies ● Inflammation predominantly at upper portion of follicle May have perifollicular fibrosis and an artefactual cleft between epithelium and stroma ● DIF shows grouped globular immunofluorescence (usually IgM) adjacent to follicular epithelium	● Exclude and address secondary drug causes, infective associations (e.g., Hepatitis B and C) and associated hypothyroidism which may further contribute to active disease- May mimic AGA, especially lichen planopilaris diffuse pattern ● An itchy or burning scalp may be a sign of occult LPP, and clinicians should consider scalp biopsy from both the active edge of the balding area and the donor site (this may be performed blind if there are no specific findings on trichoscopy) to exclude this condition
Fibrosing alopecia in a pattern distribution (FAPD)	● Alopecia in an androgenetic pattern distribution, but with lichen planopilaris features such as follicular hyperkeratosis ● Usually spares androgen-independent areas of the scalp, eyebrows, eyelashes, and body hair ● Unlike AGA, may have symptoms such as scalp dysaesthesia or pain, itch	● Peripilar casts/ perifollicular hyperkeratosis ● Perifollicular erythema ● Hair shaft diameter variability ● Loss of follicular ostia ● Predominance of single hair follicles	● Overlapping histologic features of lichen planopilaris and AGA	● HT performed in active disease is likely to have a poor prognosis
Central centrifugal cicatricial	● Slowly progressive, symmetric cicatricial alopecia centered on the	● Few isolated hairs, some with polytrichia (tufting) in an	● Premature desquamation of inner root sheath ● Eccentric epithelial atrophy	● Elicit history of and counsel patients to stop potentially damaging hair care practices

alopecia (CCCA)	crown or vertex, with areas of sparing ● Tends to affect black women of African heritage ● May be aggravated by caustic hair care products and/or hair styles causing traction ● May show features of folliculitis decalvans (pustules, erythema, crusting, secondary bacterial superinfection)	otherwise denuded central zone (white grey halo) ● Pinpoint white dots ● Pigmented asterisk-like macules with sparse terminal and vellus-like hairs	with hair shafts close to dermis ● Concentric lamellar fibroplasia of follicles ● Dense lymphocytic perifollicular inflammation at upper isthmus and lower infundibulum ● Fusion of infundibula resulting in polytrichia ● Eventual total follicular epithelium destruction with retained hair shaft fragments and granulomatous inflammation	● May be associated with vitamin D deficiency and diabetes mellitus, which should be addressed pre-surgery
Folliculitis decalvans	● Painful erythematous and crusted follicular papulopustules on vertex, occiput ● Multifocal, centrally indurated zones of scarring alopecia Perifollicular erythema and tufting at advancing margin	● Hair tufts with 5-20 hairs surrounded by yellow tubular scaling, sometimes in a starburst pattern	● Dense perifollicular inflammation of the upper follicle with epithelial destruction ● Both acute and chronic inflammation are usually present, but may have lymphocytic, neutrophilic or granulomatous predominant inflammation	● Culture for and treat active bacterial infection ● Be vigilant for squamous cell carcinoma in longstanding sites ● HT may be considered in burnt-out patches in quiescent disease
Dissecting cellulitis of the scalp	● Follicular pustules on vertex or occiput that develop into deep-seated, boggy noduloplaques with purulent sinus tracts ● Can involve entire scalp, resulting in atrophic, hypertrophic, or keloidal scars ● May have cervical or occipital lymphadenopathy	● Early findings include empty follicular openings, yellow dots, and black dots that can mimic AA ● Late findings include yellow structureless areas and 3-dimensional yellow dots over dystrophic shafts	● Dense, perifollicular inflammation of lower dermis extending into subcutaneous fat ● In early disease, many apparently undamaged follicles with intact sebaceous glands surrounded by an acute and chronic lymphocytic infiltrate ● Inflammatory infiltrate is neutrophil-predominant in longstanding lesions, with vascular proliferation and granulomatous change ● Chronic abscesses become lined with squamous epithelium, forming sinus tracts ● Eventual dense dermal and superficial fat fibrosis with loss of follicles	● Part of the follicular occlusion tetrad including acne keloidalis and hidradenitis suppurativa, which may affect wound healing ● Culture for and treat active bacterial infection
Folliculitis (or acne) keloidalis nuchae	● Follicular erythematous papules and pustules that progress to keloid-like scarred nodules along posterior hairline ● May have discharging abscesses/sinuses as well as polytrichia ● Symptoms include pruritus and burning	● Perifollicular pustules and dotted vessels[8] ● May have a white halo surrounding hair follicules[8] ● Tufted hairs can be seen	● Perifollicular, chronic lymphocytic and plasmacytic inflammation, densest at isthmus and lower infundibulum ● Lamellar fibroplasia, especially at the isthmus ● Loss of sebaceous glands ● Can progress to total follicular destruction	● Optimise non-pharmacological measures such as avoidance of high/stiff collared shirts and close shaved haircuts, and encourage the use of mild keratolytic cleansers and antimicrobial washes ● HT is unlikely to be successful in scarred keloidal skin

reducing the need for additional transplant procedures. Medical therapy may also reduce the risk of hair transplant shock loss of non-transplanted hairs in the donor area[13]. Lastly, combining medical therapy with hair transplantation can enhance overall results and maintain long-term hair health by promoting thicker hair growth, allowing the results of the transplant to last longer and look more natural over time.

Unfortunately, some forms of alopecia (e.g. alopecia areata, lichen planopilaris) may be triggered by hair restoration surgery even if well-controlled prior to the procedure, and patients should be informed of this risk[5].

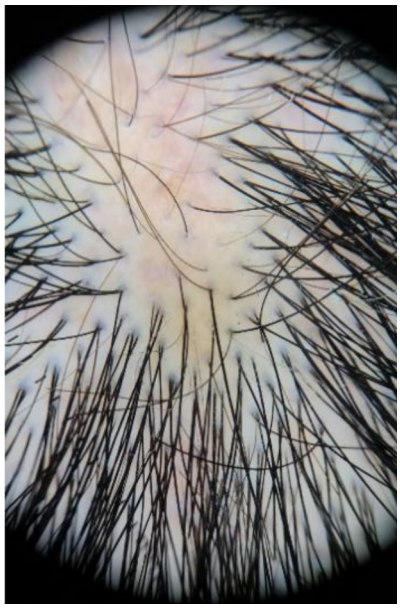

Figure 2. Trichoscopy of androgenetic alopecia showing variability of hair shaft diameters, affecting more than 20% of the hairs.

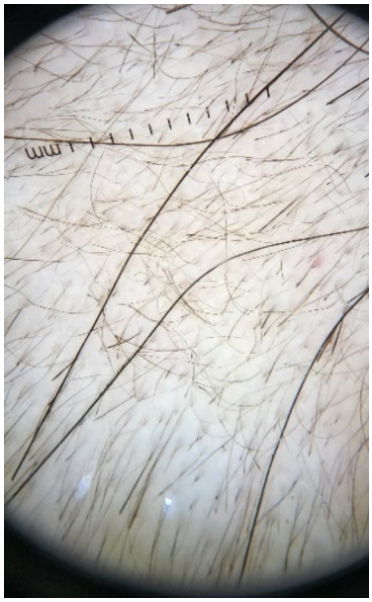

Figure 3. In active alopecia areata, trichoscopy reveals black dots, exclamation point hairs, dystrophic hairs, and some vellus-like hair shafts.

Stage of progression and patient age

In conditions such as AGA, the degree of hair loss is expected to continue to progress with time and even after surgery, thus negating the results of the hair transplant in patients who undergo the procedure too early or who do not receive adequate medical management. The Norwood scale, along with measurements of the degree of miniaturization (e.g., via folliscope), may be used to assess the patient's stage of AGA, the course of which often progresses inexorably along the 7-point scale. Some authors suggest waiting until patients are at least 25-30 years of age and/or Norwood stage 3-4, before the first hair transplant[5,11], with discussion of the potential requirement of second or third procedures down the line. Conversely, patients

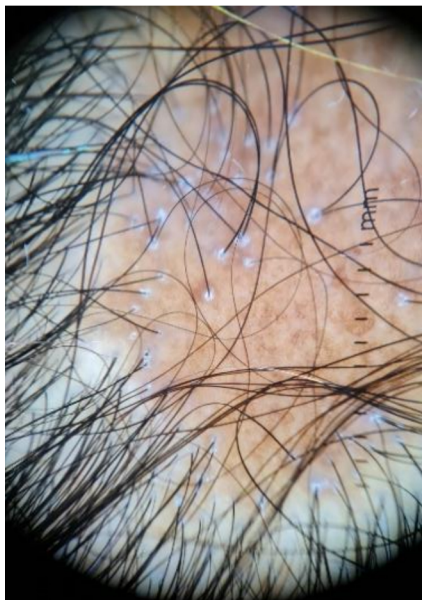

Figure 4. In lichen planopilaris, there are discrete white dots over scarred fibrotic tracts, perifollicular fibrosis, and peripilar casts.

with advanced AGA (e.g., Norwood stage 6-7) that has affected potential donor sites may not have sufficient reserves for hair restoration surgery; low density may be defined as less than 60 follicular units (FUs)/cm 2 in the donor region[5].

Donor area

The patient's donor area should be evaluated for adequate hair density and qualitative characteristics of the hair, which may vary depending on the stage of AGA, patient ethnicity, and individual differences. The qualitative features of the donor hair, such as individual donor hair thickness and characters, rather than just the number of hairs transplanted, will affect the results of hair transplantation. While the occipital scalp is the most commonly used donor area, other areas of the body such as the beard have been successfully used as donor sites in patients lacking sufficient occipital hair due to such factors as advanced AGA or previous hair transplants[12].

Complete medical and surgical hair-related treatments to date

This includes non-surgical treatments such as medications and surgical treatments such as microneedling and platelet-rich plasma (PRP), or previous hair transplants. Some therapies can optimise hair transplantation outcomes, such as medical therapies pre- and post-transplant[10]. However, other treatments such as microneedling-associated scarring or previous hair transplants may potentially adversely affect donor site reserves for future hair restoration surgeries[11]. Some patients may have poor quality or limited donor hair, limiting transplant coverage, which may not necessarily preclude transplant, but limitations of which should be made known to the patient before surgery[5].

Optimisation of non-pharmacological measures

These can help optimise outcomes peri-transplant. For instance, in discoid lupus, patients should be counselled on photo-avoidance and protection, smoking cessation, and avoidance of trauma to avoid further disease progression.

Condition of the patient's scalp

Conditions such as seborrheic dermatitis and scalp psoriasis may worsen hair loss or limit treatment and thus should be controlled prior to hair transplant. Active infection or inflammation in conditions such as folliculitis decalvans should be treated pre-transplant.

Medical conditions that may adversely affect wound healing

These should be controlled where possible, and patients informed of potential adverse effects on wound healing and graft survival. Such conditions include diabetes mellitus, smoking, corticosteroid usage, immunosuppression, and the use of dietary supplements or traditional medications that could affect bleeding risk and wound healing[5,11]. A tendency to post-inflammatory dyspigmentation (which may be more common in persons of colour), keloid or hypertrophic scar formation may also affect cosmesis post-procedure, although the risk of scarring may be reduced with follicular unit extraction[9].

Diagnoses that may affect the surgery

For instance, patients with an allergy to local anaesthetics would require the usage of alternative anesthetics (e.g., esther or amide local anesthetics) and may require allergy testing to ascertain anesthetic safety[11]. Medications used during the surgery may need to be used with caution in patients with underlying conditions such as hypertension, and liver and kidney disease.

Psychiatric or psychological diagnoses that may render patient unsuitable for hair transplant

Patient satisfaction post-procedure may be poor for patients with diagnoses such as body dysmorphic disorder[5]. Other patients may have unrealistic expectations regarding improvements in hairline and hair density post-surgery. Careful attention should be paid to pre-procedure assessment and detailed counselling of these patients, including discussion and even visual representation of projected outcomes. Such patients may require the development of physician-patient rapport and assessment of transplant suitability over several sessions. Conditions such as body dysmorphic disorder or trichotillomania may require consultation with a psychologist or psychiatrist.

Other patient factors that may contribute to a poor outcome

Non-compliance to medications, such as poor compliance to immediate post-surgical care regimens or maintenance medications such as oral dihydrotestosterone blockers in AGA, may result in poor graft survival and increased attrition of transplanted hairs post procedure[11]. Although hair transplantation is a relatively safe procedure, patients should also be counselled preoperatively on other potential unpredictable complications such as sterile folliculitis, numbness or hypersensitivity in the donor and/or recipient areas[13], and postoperative oedema/swelling. Some patients may also be at risk of subsequent infection that could compromise outcomes, such as those with MRSA colonization or who are immunosuppressed.

CONCLUSION

Careful patient selection is important to optimise outcomes in hair restoration surgery, which often involves significant time and cost, and draws on finite donor reserves. Clinicians should ascertain the diagnosis of alopecia, consider differentials/mimics and multiple pathologies, as well as address other relevant conditions prior to proceeding with hair transplant. 'No-go' features such as unstable disease and unrealistic patient expectations should be identified. As one author[5] put it, 'Not every balding person is a candidate for hair restoration'.

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Authors' contributions

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All authors declare that there are no conflicts of interest.

Ethical approval and consent to participate
As this was a review and did not involve patient studies, the study did not require ethical review and approval. The clinical photographs were deidentified as far as possible, and the patients had consented to have their photographs included for publication.

Consent for publication
Not applicable.

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