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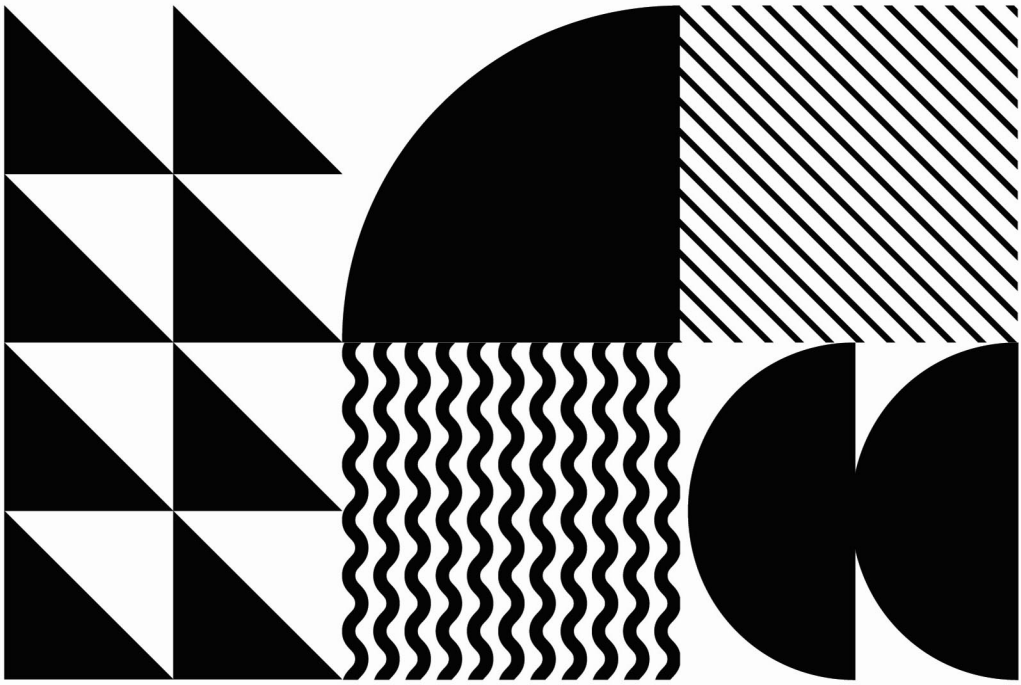

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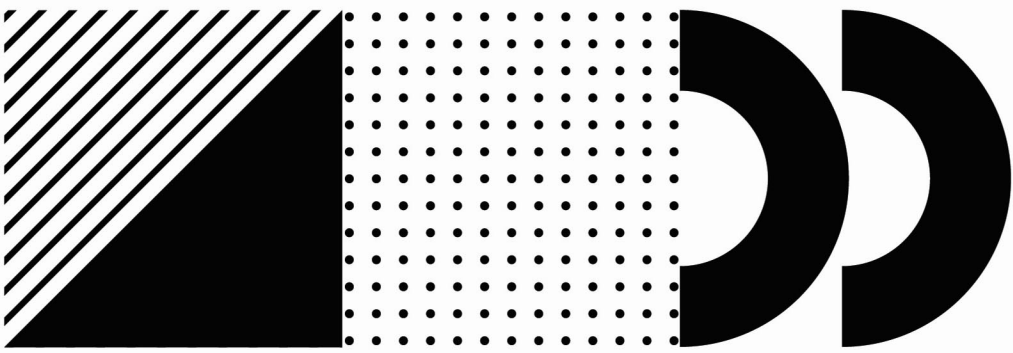

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4 CHAPTER

Stability of Vitiligo: Recommendations and Controversies

Koushik Lahiri, Abhijit Saha

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INTRODUCTION

Concept of stability of vitiligo is of utmost importance as far as treatment planning and prognosis of patient is concerned. In spite of considerable amount of improvement in medical management of vitiligo not all patients respond in a predictable manner. In those medical management failure cases, transplantation techniques are the only viable options to replenish lost melanocytes. A number of surgical modalities have been developed in the last few decades. Success of any surgical modality depends upon proper selection of cases. The specific criteria for selection have been well defined.[1]

Principles for selection are based on one single criterion, i.e., stability of the disease. It is taken as the most important parameter before opting for any transplantation technique to treat vitiligo.[2] But, the most decisive factor still remains elusive on several grounds. Better understanding of vitiligo pathophysiology over the years has prompted researchers to look into ultrastructural, biochemical, and serological parameters in addition to clinical parameters to define stability.

There are several issues to be considered while defining stability. These include:

- Defining minimum duration of stability
- Is stability area-specific?
- Validity of test graft (TG)
- How long does stability last?
- Any role of dermatoscope in defining stability
- Concept of cellular stability

MINIMUM DURATION OF STABILITY

The basic fundamental requirement "optimal duration" of the concept of stability lacks unanimity among researchers even after four decades of experience in vitiligo surgery. Lack of consensus among workers could be attributed to lack of evidence behind and subjective bias. Different periods of stability have been

mentioned by different authors which range from 4 months to 3 years.[3] Even same author has considered different periods of stability in different articles.[3-5] The Indian Association of Dermatologists, Venereologists and Leprologists (IADVL) task force came forward to solve this issue. In recent past, in their consensus recommendations, the IADVL task force for standard guidelines of care for dermatosurgical procedures attempted to provide a clear definition of stability as "a patient reporting no new lesions, no progression of existing lesions, and absence of Koebner phenomenon during the past 1 year."[6] In the absence of further clarification, this recommendation holds good before opting for surgical intervention. Rao et al. compared successes of suction blister grafting in patient with different periods of stability. Successful repigmentation (>75%) was achieved in 0% of patients in 3 months to 1 year group, 37.5% in 1–2 years group and 77.8% in >2 years group ($p = 0.005$).[7]

A recent cross-sectional study by Taneja et al. further adds to the confusion.[8] They found no association between period of stability (inactivity) and risk of reactivation in case of nonsegmental vitiligo. Longitudinal cohort study using parameters beyond clinical has been suggested in this regard.

IS STABILITY AREA-SPECIFIC?

Results are extremely variable following repigmentation of depigmented area. Simultaneous depigmentation and repigmentation have been observed in different areas of the same patient.[9]

Depigmentation of grafts was documented in herpes labialis-induced lip leukoderma.[10-16] Concurrent donor site repigmentation and depigmentation of grafts at the recipient site or vice versa have also been noticed.[5,9,17] Simultaneous perigraft spread of pigment in one area and spread of vitiligo in another area can be found in the same patient. Parallel concurrence of surgical repigmentation from the grafts and increase in size of an existing lesion in the same anatomical location was also observed. This area-based variable status of stability is neither related to the conventional refractory behavior of vitiligo, typically seen in the so-called "resistant" anatomical sites such as palms, soles, lips, nipples, areola, glans penis, and bony prominences nor it is related to the type of vitiligo such as unilateral (segmental/focal) or bilateral (symmetric, vulgaris, or generalized).

A holistic approach to the assessment of stability was conferred by Falabella[1,5] which comprises the following:

- Lack of progression of old lesions within the past 2 years (in unilateral vitiligo may be shorter, and in bilateral vitiligo, stability establishes after several years)
- No new lesions developing within the same period
- Absence of a recent Koebner phenomenon (either from history or experimentally produced)
- Repigmentation of depigmented areas by medical treatment or sometimes spontaneous repigmentation
- A positive minigrafting test
- Lack of Koebnerization at donor site

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VALIDITY OF TEST GRAFT

Introduction of minigrafting test by Falabella in 1995 added an objective dimension to the concept of stability than just arbitrarily depending upon subjective assumption of duration of stability (inactivity).[5] In the original suggested procedure, a few grafts (1.0–1.2 mm) were placed in the center of the depigmented lesion to be scrutinized. Dressing was done by Micropore® adhesive tape and kept for a couple of weeks. After removal of the tape, the area was exposed to sunlight for 15 minutes daily for a period of 3 months. No treatment was permitted during this test period. All test sites are visualized under Wood's light. The test was considered positive if unequivocal repigmentation takes place beyond 1 mm from the border of the implanted grafts. On the other hand, if <1 mm or no repigmentation was observed, the test was considered as negative. This test was further renamed as test grafting in few successive studies and found to be more realistic than arbitrary period of stability.[14,15,18]

Though TG gained popularity as a reliable method to assess stability over the years, but was not beyond criticism.

In the original article, no mention was there about the donor site. Inclusion of the outcome of the donor site will provide much comprehensive idea about stability.[19] Many researchers raised question about reliability and validity of TG as a predictor of successful repigmentation in each and every case being grafted.[18,20] Some researchers expressed their faith on availability of method of identification and isolation of skin-homing melanocyte-specific cytotoxic T-cell.[21]

None other than Falabella himself expressed his doubts about the comprehensiveness of minigraft test. He observed that even when the minigrafting test is positive, "depigmenting activity" may still be present and prevent satisfactory repigmentation.[4]

Few years later, Njoo et al. came up with their work to establish relationship between experimentally induced Koebner phenomenon (KP-e) and Koebner phenomenon by history (KP-h) and the responsiveness to ultraviolet light therapy [ultraviolet A (UVA) plus fluticasone propionate or narrowband ultraviolet B (NB-UVB)] in 61 patients who were off any topical or systemic therapies for at least 6 months. KP-e was assessed by inducing an injury by taking a 2-mm punch biopsy specimen from clinically uninvolved skin scar or depigmentation of the biopsy site after 3 months was considered as KP-e positive. A substantial difference was noted between KP-h and KP-e and the positive and negative predictive value, respectively, of KP-h was found to be 89 and 52%, respectively. Surprisingly, the responsiveness to UVB (311 nm) therapy among KP-e-positive or KP-e-negative patients did not show any statistically significant difference ($p = 0.66$). However, in the fluticasone propionate plus UVA group, KP-e-positive patients showed a better response than did KP-e-negative patients ($p = 0.01$).[22]

Vitiligo disease activity (VIDA) score was described by the same worker to grade the level of activity of disease in individual vitiligo patients who were without any therapies for at least 6 months. Scores were given from +4 to –1 depending upon decrease in disease activity. It was observed that patients with VIDA scores

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of +1 and +2 did not necessarily show a positive KP-e. However, all the patients with VIDA scores of +3 and +4 showed a positive KP-e. They concluded:

- The VIDA score did not always predict a positive KP-e.
- The KP-e may function well as a clinical factor to assess present disease activity and may also predict the responsiveness to fluticasone propionate plus UVA therapy but not to UVB (311 nm) therapy.

It this context, it can be concluded that KP or TG should not be considered as full proof of disease activity. Because what these two reveal is the apparent clinical stability only and that may not be the true reflection of stability status of the disease at the cellular level.[18]

HOW LONG DOES STABILITY LAST?

It is often hard to predict how long the disease will remain stable or on the other way round when it will start to become unstable. Repigmentation in previous graft failure cases with NB-UVB (311 nm) phototherapy has been documented.[16] Proposed mechanism of action of NB-UVB might stabilize the disease by promoting apoptosis of the perilesional and circulating melanocyte-specific cytotoxic CD8 cells.[23] The observation of spontaneous repigmentation points toward a possible release of fresh cytokines from the donor skin not only stimulate the vitiliginous patches and hair follicles of the grafted sites but also repigment distant sites by local absorption.[24-26] Another theory was the immunogenic mechanism which was originally responsible for the development of vitiligo may have lost its antigenicity due to the autologous grafts.[23] Successful split-thickness skin graft (STSG) in PG failure cases further complicates the picture.[27] Period of stability of repigmentation has been proposed as 1 year. That means, to qualify as stable, the repigmentated status must be maintained for a period of not <1 year.[28]

ROLE OF DERMATOSCOPE IN DEFINING STABILITY

Recently, dermatoscope has emerged as a powerful tool to assess stability of vitiligo. Perifollicular and perilesional hyperpigmentation point toward stable vitiligo whereas trichome border, satellite lesions, and micro-Koebner phenomenon is strongly associated with instability.[29]

CONCEPT OF CELLULAR STABILITY

The exact pathomechanism of stability is still an enigma. The incompletely understood mechanism involves many theories and hypothesis. The only definite and certain factor is the absence of melanocytes in the lesion. So, it can be concluded that destruction of melanocytes is the key factor for unfolding of the disease process. Classically, there have been three hypotheses to elucidate vitiligo: the neural hypothesis, the self-destruct hypothesis, and the immune hypothesis of which the last one is gaining grounds. Aberrations of both humoral and cell-mediated immunity have been noticed. Most of the patients with generalized vitiligo were found to have circulating antibodies to cell surface

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antigens on normal human melanocytes; these antibodies were cytotoxic to normal melanocytes and to melanoma cells in tissue culture. Humoral immunity does not appear to be fundamental for the pathogenesis of vitiligo, as serum titers of melanocyte-reactive antibodies do not correlate with disease activity,[30] and the uniform distribution of circulating antibodies cannot explain the patchy appearance of vitiligo lesions.[31] Perilesional and circulating T-cells strongly suggest that melanocyte-specific cytotoxic T cells are involved. And now it is known that UVB therapy promotes this T-cell apoptosis. Repigmentation has been successfully induced in previous graft failure cases under NB-UVB (311 nm) phototherapy.[32]

Recent updates on pathogenesis in vitiligo focus on few important points:

- Resident memory T cells (T_{RM}) play a crucial role in the persistence and relapse of vitiligo. They (a type of CD8+ T cells expressing CD69, CD103, and CD49a) have the ability to remain in tissues and induce early immune responses.[33] A large population of T_{RM} cells reside in vitiligo skin and are involved in relapse promoting recruitment and proliferation of circulating $CB8^+$ T cells through the release of interferon (IFN) pathway cytokines.[34] Interleukin-15 (IL-15) has a central role in the maintenance and function of T_{RM} cells. CD8+ T cells secrete IFN-γ, which induces the production of chemokines CXCL9 and CXCL10 by keratinocytes.[35] These chemokines bind to the T-cell receptor CXCR3, increasing T-cell recruitment, thereby leading to the initiation, progression, and maintenance of vitiligo lesions.[36] Tregs play a pivotal role in maintaining self-tolerance by suppressing the activity of autoreactive effector T cells. IL-2 is essential for Tregs survival and functionality in peripheral tissues.
- In one study, significant increases in CD3+, CD4+, and CD8+ T cells were found in margin of vitiligo lesions and these cells were mostly activated CD45RO+ cells of the memory subset. Most intense infiltration was present within 0.6 mm of the edge of the lesion.[37]
- The detection of melanocyte-reactive cytotoxic T cells in the peripheral blood of vitiligo patients and the observed correlation between perilesional T-cell infiltration and melanocyte loss in situ suggest the important role of cellular autoimmunity in the pathogenesis of this disease. T cells have been isolated from both perilesional and nonlesional skin biopsies of vitiligo patients, then cloned and their profile of cytokine production analyzed. Perilesional T-cell clones (TCC) derived from patients with vitiligo revealed a predominant type-1-like cytokine secretion profile, but the degree of type-1 polarization in uninvolved skin-derived TCC correlated with the process of microscopically observed melanocyte destruction in situ. Furthermore, CD8+ TCC derived from two patients also were analyzed for reactivity against autologous melanocytes. The antimelanocyte cytotoxic reactivity was observed among CD8+ TCC isolated from perilesional biopsies of patients with vitiligo.[38]
- Expression of CCL22, a skin-homing chemokine for Treg (regulatory T cells), has been found to be deficient in vitiligo skin on immunohistochemistry. This was hypothesized to explain the failure of circulating functional Treg to

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home to the skin in vitiligo. Absence of Treg cells at the place of occurrence perpetuate antimelanocyte reactivity in progressive disease.[39]

- Rao et al. studied the correlation of results of repigmentation following suction blister grafting in vitiligo with serum catalase levels and lesional immunohistochemistry for CD4, CD8, CD45RO, CD45RA, and FoxP3. No correlation of serum catalase levels with either the period of stability or the results of surgical repigmentation was found. Similarly, CD45RA, CD4, and Fox P3 were also not found to correlate with response to surgical treatment. However, CD8 T-cell counts were found to be lower in responders [median 1% (range 0–4%)] as compared to the nonresponders [median 3% (range 1–7%)] ($p = 0.04$). Higher percentage of CD8+ cells is associated with lower percentage of repigmentation. CD45RO cells were found to be completely absent in responders while some nonresponders showed their presence. Along with CD8 cells, CD45RO cells contribute significantly for propagation and persistence of cytokine milieu which are responsible for instability.[7]
- A recent study evaluated potential biomarkers to assess stability of vitiligo and success of surgical transplantation. CXCL9 and CXCL10 levels in blister fluid sample of stable and active vitiligo were measured. Outcome of the cultured pure melanocyte transplantation (CMT) was evaluated after 6 months. CXCL9 and CXCL10 levels were found to be sensitive and specific markers of active vitiligo. CXCL9 and CXCL10 levels were significantly lower in stable vitiligo. Effectiveness of CMT was determined by CXCL9 level.[40]

CONCLUSION

It is obvious that before any attempt to predict the outcome of any vitiligo surgery, along with the clinical stability, assessment of "cellular" stability is of utmost importance. Before embarking upon any surgical alternative, no preconceived, biased, and arbitrary formulae should guide us to assess stability of vitiligo. Till those unexplored areas are charted and the cellular parameters come into real practice, we should not step back from surgical options rather we should continue surgical interventions based on logical application of our clinical knowledge. Also, so far these tests are only research tools available in very few centers and extensive work needs to be done to make these tests sufficiently reliable, cheap, and widely available. Lesional stability as a concept needs to be investigated in depth. In addition, there is a need to set uniform clinical criteria for defining stability before embarking on biochemical and other investigative findings so as to be able to compare results between studies and draw definitive conclusions.

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Clinical Management of VITILIGO

This comprehensive book on vitiligo provides an all-in-one resource for dermatologists, covering clinical, medical, and surgical management, along with phototherapy and its evolving role. It incorporates the latest innovations and research in vitiligo treatment, such as cellular grafting techniques, immunomodulators, Janus kinase (JAK) inhibitors, laser-assisted therapies, and combination approaches, while offering step-by-step procedural guides for surgical techniques such as melanocyte transplantation and suction blister grafting. Featuring real-life case studies, high-resolution of clinical images, and comparative treatment visuals, it ensures practical insights and enhanced understanding. The book emphasizes the psychosocial aspects of vitiligo, offering counseling techniques tailored to diverse patient's needs, particularly for ethnic and darker skin tones. With detailed protocols for phototherapy modalities such as narrowband ultraviolet B (NB-UVB), psoralen ultraviolet A (PUVA), and excimer laser, it serves as a vital resource for optimizing results and minimizing side effects. Additionally, it provides a future-forward perspective on emerging therapies such as biologics and cellular reprogramming, making it a forward-thinking and invaluable reference. Dedicated as a tribute to a visionary teacher, this book has been written by leading dermatologists, blending evidence-based science with mentorship and inspiration. It combines global best practices with localized insights, making it a must-have for dermatologists, postgraduate students, and researchers worldwide.



Madhulika Mhatre is a renowned dermatologist with over a decade of expertise in the diagnosis and treatment of vitiligo. She has dedicated her career to advancing knowledge and improving patient's outcomes in vitiligo care. Dr Mhatre has conducted numerous workshops and delivered talks on vitiligo at both national and international platforms, establishing herself as a leading voice in the field. Her contributions to dermatology are reflected in her various publications and chapters in prominent medical books. Over the years, she has been honored with several scholarships recognizing her outstanding work and commitment to vitiligo research and treatment. Driven by a deep desire to enhance public understanding and patient's support, Dr Mhatre coauthored this book to provide a comprehensive resource that empowers individuals affected by vitiligo and educates the wider medical community.



Aseem Sharma is a distinguished physician with a strong academic background in dermatology and a particular focus on vitiligo. His collaborative work with Dr Mhatre in the field of vitiligo has led to groundbreaking insights, and together they have worked to raise awareness and improve therapies for this challenging condition. Dr Sharma's dedication to advancing vitiligo research and patient's care has made him a respected figure in the dermatological community. Motivated by the need for more accessible, reliable information on vitiligo, he coauthored this book with Dr Mhatre to fill the gap in patient's education and provides a holistic understanding of the disease, its treatment options, and its psychological impact.

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