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Mesenchymal stem cell treatment for psoriasis

Abstract

Psoriasis is a chronic, autoimmune disorder characterized by inflammation and the development of red, scaly patches on the skin called plaques. It is often most recognizable by the dermatological presentation of the disease, characterizedby large or small, red, scaly patches on the skin. These patches are most often located on the joints, trunk, scalp, arms, legs, face, and back. The disease affects millions of people worldwide and manifests in varying degrees of severity, ranging from mild plaque psoriasis to severe erythrodermic involvement. Current treatments for psoriasis-including topical agents, phototherapy, oral immunomodulators, and biologic drugs, target inflammatory pathways but remain limited for individuals with biologic-resistant psoriasis. Mesenchymal stem cell (MSC) therapy has emerged as a therapeutic approach due to the immunomodulatory, anti-inflammatory, and regenerative properties of mesenchymal stem cells. Early clinical studies show promising results, including improved quality of life for psoriasis patients, reductions in Psoriasis Area Severity Index (PASI) scores or Body Surface Area (BSA) scores, and even long-term remission in a few cases. While there is currently no known cure for psoriasis, mesenchymal stem cell therapy has the potential to correct the flaws in the immune system to promote the development of the disease, leading researchers to believe this treatment has to potential to one day become a holistic cure. This paper reviews the pathology of psoriasis, current therapies, and available clinical evidence supporting MSC-based treatments while highlighting future directions, limitations, and potential clinical applications.

Keywords: psoriasis, mesenchymal stem cells, immunotherapy, biologic-resistant psoriasis, pasi score, chronic inflammation

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Abbreviations

MSC, mesenchymal stem cell; HSC, hematopoietic stem cell; PASI, psoriasis area severity index; DLQI, dermatology life quality index; TNF-a, tumor necrosis factor-alpha; IL, interleukin; BSA, body surface area; Th17, T-helper 17 cell; UVB, ultraviolet B; NF-kB, nuclear factor kappa beta; IFN, interferon; NAFLD, non-alcoholic fatty liver disease; MHC, major histocompatibility complex; HLA, human leukocyte antigen; AMP, antimicrobial peptides; PSORS, psoriasis susceptibility locus; JAK, janus kinase; PDE4, phosphodiesterase 4; UC-MSC, umbilical cord mesenchymal stem cell; GMP, Good manufacturing practice

Introduction

Psoriasis is a chronic, immune-mediated dermatological condition that affects approximately 2-3% of the global population.[1] It presents with small, red plaques covered in silvery scales and is associated with comorbidities including psoriatic arthritis, inflammatory bowel disease, non-alcoholic fatty liver disease, obesity, type 2 diabetes, and major depressive disorder. Historically, psoriasis was considered a skin disease. However, it is now understood as a systemic inflammatory disorder involving the dysregulation of both the adaptive and innate immune mechanisms.

Psoriasis has impacts that go beyond the physical presentation of the disease. Many patients endure significant mental distress, including anxiety, depression, and increased risk of suicidal behaviors, embarrassment, and decreased satisfaction with their own bodies. The extreme mental health burden on psoriasis patients also goes beyond the severity score of the disease, as even patients with mild cases of psoriasis experience a psychological burden. Psoriasis patients are also more prone to substance abuse, including alcohol, antidepressants, smoking, and sedatives. Not only does it present an increased risk for patients, but it also healthcare providers. The unpredictable nature of the disease and its impact on many aspects of life make it challenging for physicians to understand the full influence of the disease, which is crucial for developing effective management strategies.[2]

The global challenge presented by psoriasis includes a variety of factors. The economic burden of psoriasis is demonstrated by challenges related to work productivity, with notable reduction in workplace efficiency and activity levels, which can lead to financial instability for affected individuals. Additionally, a lack of public knowledge about the disease contributes to negative stigma surrounding psoriasis. The persistence of misconceptions about psoriasis continues to contribute to preventing improvements in the quality of life of psoriasis patients, as many face judgment or a lack of acceptance.

Psoriasis also significantly increases the risk of other diseases. Patients with psoriasis are at higher risk for stroke and cardiovascular mortality. The risk for myocardial infarction increases by 1.4-fold for patients with severe cases of psoriasis.[3] Psoriasis patients also have higher rates of obesity, insulin resistance, and hypertension.[4] Diabetes risk increases by 65% in patients with psoriasis,[5] and the disease is also strongly associated with NAFLD.[6] Approximately 30% of psoriasis patients develop psoriatic arthritis,[7] and the IL-23 pathway associated with psoriasis has also been linked to Crohn's disease and ulcerative colitis.[8] This goes to show that psoriasis is far more than a topical skin condition and can severely impact the proper functioning of the body.

Multiple clinical subtypes of psoriasis exist, including plaque psoriasis,which is the most common and accounts for 80-90% of psoriasis cases, nail psoriasis, guttate, inverse, pustular, and erythrodermic psoriasis. The severity of the disease is often quantified using PASI and BSA, with PASI scores of >10 or BSA scores >10% indicating severe disease and probable need for biologic treatment.

The pathophysiology involves overactive T-cell responses, accelerated keratinocyte production, and overactive cytokine release (IL-17, IL-23, TNF-α), leading to epidermal thickening.[9]

While advances in biologic therapy have been made, a subset of patients is not responsive to biologic therapies, loses responsiveness over time, or experiences adverse effects. These limitations highlight the need for new therapeutic strategies. Stem cell therapies, specifically MSCs, have gained positive attention for their ability to modulate immune pathways and potentially lead to long-term remission.

Pathophysiology of psoriasis

Psoriasis arises from a combination of genetic predisposition, environmental factors, and immune dysregulation. Genetic and immune components potentially affecting the development of psoriasis are mutations affecting immune signaling and skin barrier function, overactive infection-fighting immune cells that target healthy cells by mistake, and elevated pro-inflammatory cytokines that promote hyper proliferation of keratinocytes. Some environmental triggers that may contribute to the development of the disease include streptococcal infections (particularly in guttate psoriasis), skin trauma, fungal infections, medications, and stress. In addition to these, cellular mechanisms of psoriasis consist of the proliferation of keratinocytes at drastically accelerated rates, and the activation of Th1 and Th17 cells from dendritic cells, which then amplify cytokines like IL-17, IL-23, and TNF-α- continuing the cycle of plaque formation and inflammation. Symptoms of psoriasis include itching, irritation, red or purple patches of skin, plaques, burning, stinging, pustules or blisters, dry skin that may crack or bleed, changes to nails, and flaking of dead skin.[10]

Genetics play a major contributing role in the development of psoriasis, with an estimated heritability of 60-90%. More than forty regions in the human genome have been associated with psoriasis, including HLA-Cw6, IL12B, IL23R, LCE3A, LCE3D, and STAT3C. The region of the chromosome thought to encode a psoriasis gene is referred to as the psoriasis susceptibility (PSORS) locus, and it is currently understood that it encodes at least fifteen different loci. Many of these loci are involved in the antigen presentation of CD8 T cells, and the active loci may vary depending on geographical location. Simply put, after classifying the genes associated with the disease, different biological mechanisms and environmental factors contribute to the presentation of the disease.[11] These findings illustrate that psoriasis is not only a dermatological condition, but a genetic inflammatory condition that can be set off by environmental factors that may activate underlying immune dysfunction.

Psoriasis is strongly associated with alleles in the MHC region and HLA alleles. Class I antigens HLA-B13, HLA-B17, HLA-B57, and HLA-Cw6 consistently show a positive correlation with psoriasis across various populations. HLA-Cw6 particularly stands out due to its association with more severe and early-onset psoriasis cases. The HLA-Cw6 antigen was also discovered in 100% of patients who had guttate psoriasis. The contribution of the HLA region to psoriasis is still not fully understood (Figure 1).[12]

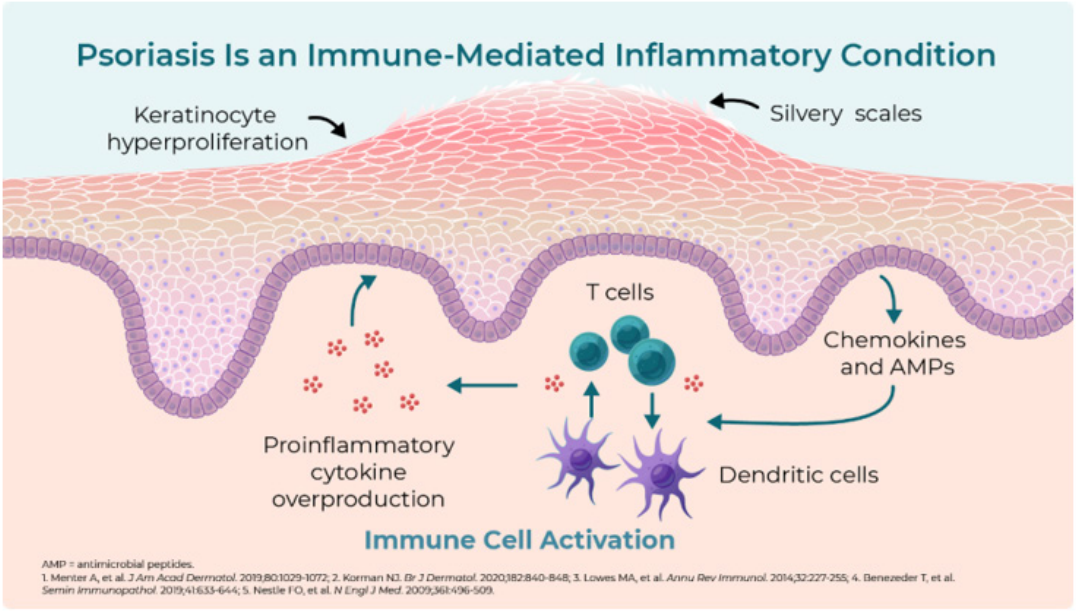

Figure 1 Pathway formation of psoriatic plaques.[13]

Immune cells are the major contributors to psoriasis plaques. These immune cells are dysfunctional and consist of dendritic cells, T cells, and pro-inflammatory cytokines. The dendritic cells become overactive and stimulate T cells that promote chronic inflammation. The T cells then signal keratinocytes to work faster and produce inflammatory molecules. This leads to the overproduction of pro-inflammatory cytokines, which are responsible for the inflammation, redness, swelling, and thickening of skin associated with psoriasis. The keratinocytes also produce chemokines, which attract more immune cells to the skin, ultimately worsening the inflammation. The AMPs are natural immune molecules that become over expressed in psoriasis and can activate dendritic cells, also increasing inflammation. Overall, the cycle of psoriasis begins with an environmental trigger activating dendritic cells, the dendritic cells activate T cells through cytokines, the T cells release pro-inflammatory cytokines, and then keratinocytes begin proliferating and release their own inflammatory mediators, followed by chemokines recruiting even more immune cells, which send the cycle back to the beginning.

Clinical manifestations and classification

Plaque psoriasis is characterized by raised plaques with silvery scales, often located on the scalp, elbows, knees, and trunk. This is the most predominant subtype of psoriasis, affecting 80-90% of patients with psoriasis.

Guttate psoriasis presents as small, scattered plaques around the body and is frequently triggered by an infection such as streptococcal infection. It is commonly found on the midsection of the body, including the trunk, arms, legs, and back. It can also be found on the face and the scalp.

Inverse psoriasis occurs in areas where skin overlaps. This is often in areas of skin folds, such as the armpit, genitals, under the breasts, and behind the knees, and consists of shiny lesions lacking scales.

Pustular psoriasis is similar to plaque psoriasis but features pustules located on top of plaques and can be localized or generalized.

Nail psoriasis includes the pitting and discoloration of fingernails on the fingers and toes, and can accompany other types of psoriasis as well.

Erythrodermic psoriasis is the most severe type of psoriasis, characterized by a BSA of 90% or higher or a PASI score of 90 or higher. It is a rare, severe variant and may be life-threatening.

Current treatments

The current treatment protocol for patients with psoriasis has a variety of potential options, including topical treatments, systemic and biologic therapies, phototherapies, and oral immunomodulators. Options for topical treatments include steroid and non-steroid treatment options. Steroid options include corticosteroids ranging from superpotent to least potent and can occasionally be used in combination with retinoids. Non-steroid treatment options include anthralin, vitamin D, and vitamin A derivatives. Over-the-counter therapies are also considered topical, non-steroid treatments and can include coal tar, moisturizers, and medicated shampoo. These are typically used for mild cases of psoriasis and are not sufficient for moderate to severe cases. These types of psoriasis therapies are often hard to control due to varying dosages and skin sensitivity. This is because topical treatments are usually prescribed in the form of a cream or gel and given to patients for self-administration. Though physicians do give guidelines on how much and how often to apply the topical treatment, it is nearly impossible to monitor the exact amount patients are administering. This also becomes challenging when patients do not perfectly stick to an administration schedule, as over applying can lead to complications like skin thinning, and under applying can lead to inhibited results on dermatological plaques. Phototherapies for psoriasis include narrowband UVB, natural sunlight, and excimer lasers.

Oral targeted agents include PDE4 inhibitors like Otezla and JAK inhibitors such as Rinvoq.[14] These medications are frequently used to treat psoriatic arthritis in conjunction with other treatments for the dermatological presentation of the disease.

Systemic and biologic therapies are the most holistic and intense psoriasis therapies. These treatments typically come in the form of a self-administered injection in varying doses. They work by targeting specific immune pathways. Examples of this include TNF-a inhibitors, including Enbrel and Humira, IL-17 inhibitors, including Cosentyx, Taltz, and Bimzelx, IL-23 inhibitors like Skyrizi and Tremfya, and IL-12/23 inhibitors like Stelara. While biologic therapies are effective for many, they are expensive and often not fully covered by insurance. They are also not recommended during pregnancy and may fail in a subset of patients with biologic-resistant psoriasis. There are assistance programs in place to help psoriasis patients pay for injections.

Biologics work by targeting immune components and are currently the most effective treatment administered for severe psoriasis. There are a variety of pathways targeted by biologics, including TNF-α inhibitors, IL-17 inhibitors, IL-23 inhibitors, and IL-12/23 inhibitors.

TNF-α inhibitors work by neutralizing TNF-α, which is a cytokine that drives the activation and maturation of dendritic cells, keratinocyte proliferation, Th1 and Th17 differentiation, and neutrophil recruitment. It is an early contributor to psoriasis inflammation, and blocking or inhibiting it can suppress multiple inflammatory pathways.

IL-17 inhibitors neutralize IL-17, which is a cytokine that stimulates keratinocyte proliferation, chemokine release, and antimicrobial peptide production. IL-17 is farther downstream than TNF-α, and inhibiting it primarily prevents plaque formation.

IL-23 inhibitors neutralize IL-23, which is a cytokine that aids in maintaining Th17 cells, sustaining chronic keratinocyte inflammation, and initiating IL-17 and IL-22 production (cytokines that also contribute to inflammation). IL-23 is also a downstream pathway in psoriasis, but inhibition has proven to be highly effective.

IL-12/23 inhibitors bind the p40 subunits shared by IL-23 and IL-12. This prevents Th1 differentiation driven by IL-12 and Th17 differentiation and survival driven by IL-23. Overall, this reduces the production of IL-22 and IFN-γ, which are major contributing cytokines and psoriatic plaque formation.[15]

Rationale for mesenchymal stem cell treatment

MSCs offer a unique therapeutic profile due to their immunomodulatory effects. They can modify both innate and adaptive immune responses by secreting anti-inflammatory cytokines, including IL-10 and TGF-β, suppressing pathogenic T cell activity. MSCs also inhibit Th17 differentiation, dendritic cell maturation, and NK cell activity. They also promote regulatory T cell expansion.[16] MSCs are capable of tissue repair and regeneration through the secretion of growth factors that reduce epidermal thickening and restore the natural skin profile.

MSCs in psoriasis patients are dysfunctional relative to stem cells in non-psoriasis patients. In psoriasis patients MSCs have been discovered to have a low differentiation capacity and low immunomodulatory function. They also have high HLA-I expression levels, abnormal cytokine secretion, and impaired inhibition of lymphocytes. MSCs from psoriatic plaques have been shown to stimulate the growth of keratinocytes, leading to epidermal thickening. In addition to this, there are reports of acquired psoriasis in patients who underwent bone marrow transplants from donors who had psoriasis.[16] HSCs play a large role in acquired psoriasis from bone marrow transplants, though it demonstrates the concept of dysfunctional stem cells in psoriasis patients, exaggerating the need for allogeneic MSC therapies as opposed to autologous MSC therapies.

Current clinical trials with MSCs have shown promising results, suggesting that MSCs are tolerated well and are safe for the duration of follow-up (>2 years).

Clinical trials and case studies

Early clinical trials show promising results for the treatment of psoriasis with MSC therapies. Results currently vary, and study samples are small and limited to biologic-resistant psoriasis populations. Administered dosage of MSCs also varies by individual. The clinical trial presented consists of three women with long-standing psoriasis who underwent treatment with multiple biologics and had no success. All three women underwent therapy with umbilical cord mesenchymal stem cells and were observed for more than a year post-infusion. Administered dose of UC-MSCs varies by individual due to body weight, and because therapies are still in early stages and have not yet been narrowed down to a specific dosage.[17]

Patient 1 is a 46-year-old white European woman with plaque psoriasis for 25 years. She had associated comorbidities, including psoriatic arthritis, type 2 diabetes, non-alcoholic fatty liver disease (NAFLD), anxiety, and depression. Her baseline severity scores consisted of a PASI score of 37.6 and a DLQI of 27, indicating a significantly obstructed quality of life and extremely severe psoriasis coverage. Patient 1 had previously undergone treatment with six biologics, all of which had failed. These biologics include Cosentyx, Skyrizi, Tremfya, Humira, Remicade, and Enbrel. This patient was given a concentration of $2.57x10^6$ cells/kg/infusion ($229x10^6$ total). Her initial infusion occurred at week 0, and she received a second infusion at week 1. The patient continued treatment with biologics (Enbrel) for approximately 30 weeks post-infusion (Figure 2).

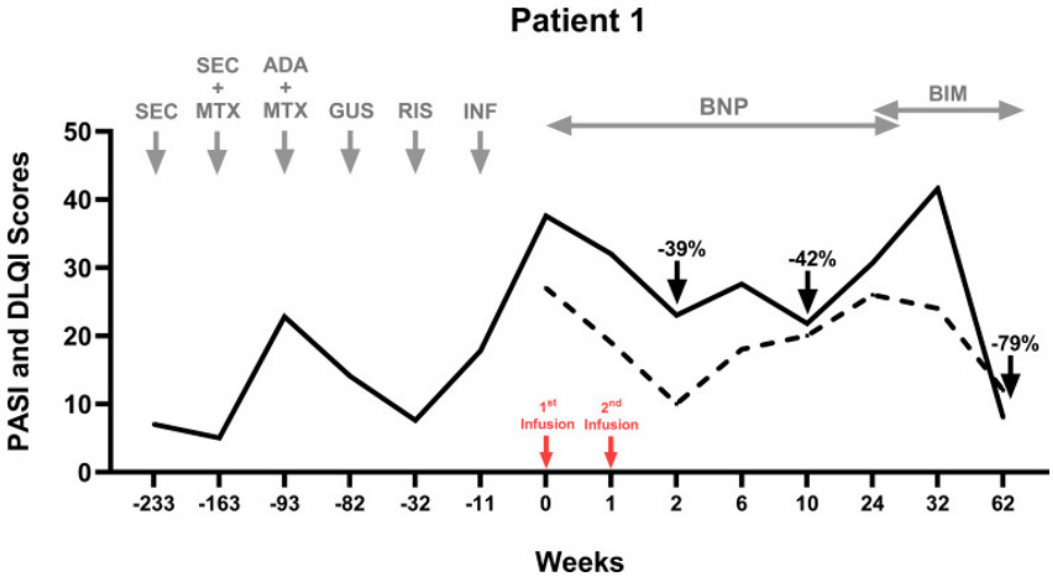

Figure 2 Progress of patient 1 before and while undergoing treatment with MSCs.[17]

At week 2, patient 1 had experienced a 39% improvement from her baseline PASI score of 37.6. At week 10, she had achieved a 42% improvement from her baseline PASI score. At week 24, her PASI score was 30.7, and continued to increase until week 32, when she had surpassed her baseline score. At this point, treatment with a new biologic (Bimzelx) was initiated. At week 79 62 the patient had achieved a 79% improvement from her baseline PASI score.

The treatment administered to this patient was unique. This is because patient 1 is the only patient who remained on a biologic treatment for the duration of her treatment with UC-MSCs. This presents a potentially confounding variable in the study because the effects of MSCs are still not fully understood, and researchers may be unaware of potential interactions between the biologic and the initial infusion dose of MSCs. However, it is still reasonable to say the UC-MSCs had an impact on patient 1, as she had previously undergone treatment with a biologic in the same category as the biologic she was using during her stem cell treatment. After her infusion, she had increased sensitivity to a biologic known as Bimzelx. Before her infusion, she had undergone treatment with Cosentyx. Both biologics fall under the category of an IL-17 inhibitor. Because she had no significant improvement with an IL-17 inhibitor before her MSC infusion, it can be inferred that the increased sensitivity to the IL-17 inhibitor after her infusion was likely due to the immunomodulatory effects of the UC-MSCs administered.

Patient 2 is a 45-year-old South Asian woman with small plaque psoriasis for 25 years. No comorbidities were listed for this patient. Her baseline severity scores included a PASI score of 13.8 and a DLQI score of 15, indicating a low quality of life and severe psoriasis coverage. Though her PASI score and DLQI score are lower than the scores of patient 1, her case is still considered severe, and her psoriasis is also biologically resistant. Patient 2 previously underwent treatment with four biologics, none of which were successful. These biologics include Enbrel, Cosentyx, Stelara, and Tremfya. This patient was given a concentration of MSCs of 3.00x10^6 cells/kg/infusion ($229x10^6$ total). Her initial infusion occurred at week 0, and she received a second infusion at week 1. The patient was not being treated with biologics during her initial infusions and for several weeks during her treatment (Figure 3).

At week 1, patient 2 had already achieved a 41% improvement from her baseline PASI score of 13.8. At week 4, she had reached an 87% improvement from her baseline PASI score. At week 9, she experienced a temporary rebound of symptoms, and her PASI score exceeded her baseline score by above a 15. At this point, treatment with a biologic (Tremfya) was resumed. At week 30, patient 2 had achieved about a 90% improvement from her baseline, and this was maintained for the remainder of her observed treatment window.

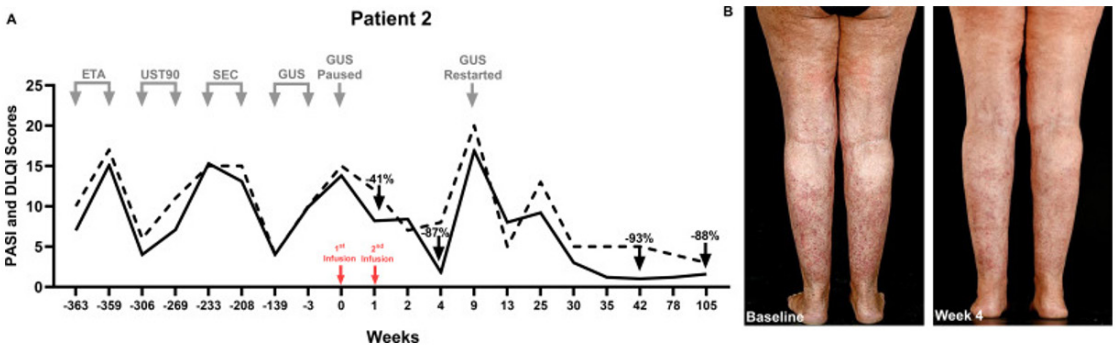

Figure 3 Progress of patient 2 before and while undergoing treatment with MSCs.[17]

Patient 3 is a 47-year-old Chinese woman with long-standing large-plaque psoriasis. Her comorbidities include NAFLD and a positive test for the Hepatitis B core antibody. Her baseline severity scores include a PASI score of 22.8 and a DLQI score of 20, indicating a significantly low quality of life and very severe psoriasis coverage. Her psoriasis is also biologic-resistant, as she previously underwent treatment with five biologics, and none were successful at treating her condition. These five biologics include Humira, Stelara, Cosentyx, Skyrizi, Siliq, and Bimzelx. The patient was given a concentration of MSCs of $1.96x10^6$ cells/kg/infusion ($151x10^6$ total). Her initial infusion occurred at week 0, and she received a second infusion at week 1. The patient was not being treated with biologics at the onset of her MSC therapy and continued to abstain from biologic treatment for several weeks post-infusion (Figure 4).

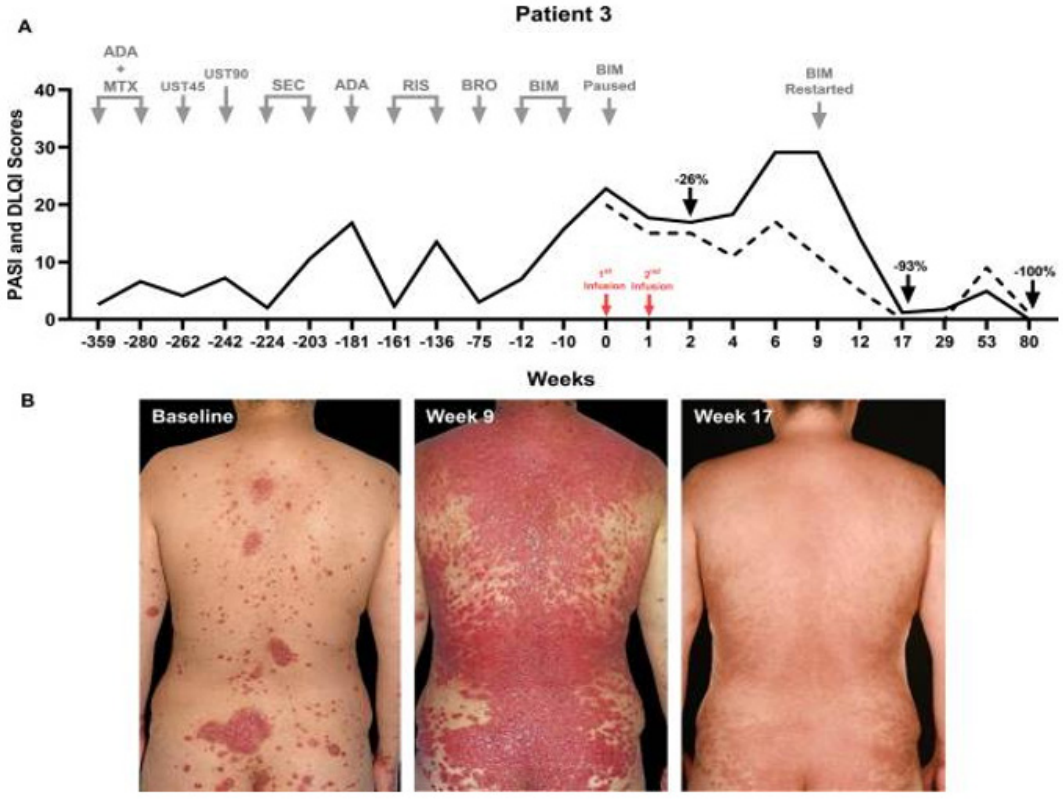

Figure 4 Progress of patient 3 before and while undergoing treatment with MSCs.[17]

At week 2, patient 3 had achieved a 26% improvement from her baseline PASI score of 22.8. At week 6, she experienced a temporary rebound in her symptoms, with her PASI score exceeding her baseline at about 28. Without seeing improvement after a week, treatment with a biologic (Bimzelx) was resumed at week 9. At week 17, the patient experienced a 93% improvement from her baseline. At week 80, she had 100% improvement from her baseline and was in remission.

Discussion

Results from these studies show promise regarding the future of MSC therapies for psoriasis. The most significant take away from this study is the increased sensitivity to the biologic treatments after infusion with the mesenchymal stem cells. All three patients showed noticeable improvement after their first infusion, with one patient showing improvement after only her first infusion. Additionally, another significant finding from this study is the complete remission noted in patient 3. This finding indicates that mesenchymal stem cells have the potential to be curative for psoriasis patients.

It is important to note that all three patients remained on biologics during their stem cell therapy. This presents a potentially confounding variable in all cases, as remission cannot be proven to have been reached through UC-MSCs infusion alone. It was not stated in the study how long these patients remained on biologics after the observation window, or if they remained on biologics at all. The combination of the UC-MSC infusion and the biologic treatment proved to be effective at reducing the PASI scores of each of these patients, suggesting that the MSC therapy was effective in these three patients who had previously been biologic resistant.

Though it was not explicitly stated why patients received two injections, one week apart, it is likely that this occurred with the intention of allowing the first infusion to begin to work in the body, and then the second infusion would act as a "booster" and amplify the effects of the MSCs. More infusions likely did not occur as progress was seen very quickly after two infusions of MSCs, and in some cases, after one infusion. While only one patient reached remission, all three patients showed significant improvement from their baseline scores during treatment. A key takeaway from these studies is the increased sensitivity to biologic treatment after undergoing MSC therapy. While each patient had previously failed treatment with biologics before treatment with MSCs, upon the resumption of biologic treatment after their infusions, their PASI scores significantly improved when the biologic was added back into their treatment plan.

It is unclear exactly why all three patients experienced a rebound in their PASI scores after infusion with MSCs. Speculation suggests that this may be due to there being a gradual shift from an initial inflammatory response to long-term immune regulation. Because MSCs primarily exert their effects through paracrine signaling, improvement would occur after regulatory pathways are established in the body. In addition to this, MSCs may trigger innate immune activity directly after administration, which may lead to a temporary inflammatory flare before they can exert their immunomodulatory effects.

All three patients had severe psoriasis cases, with patient 1 having the most severe case with a PASI score of 37.6. Patient 2 had the least severe case with a PASI score of 13.8. Patient 3 was in the middle with a PASI score of 22.8. Both patient 2 and patient 3 reached remission or got very close to remission, while patient 1 had significant improvement but was not close to remission at the end of the observation period. The reason for this is not explicitly outlined, though it can be speculated that this is related to the severity of their psoriasis at baseline. Patient 1had a significantly higher baseline score than both patient 2 and patient 3, even though all three cases were considered severe. Patient 1 may have needed a greater dosage of UC-MSCs during her infusions, though it would be challenging to come to that conclusion due to their being lack of confirmation in dosages at this point in clinical studies. Additionally, patient 1 remained on a biologic for the entirety of her observation period, including during her first and second infusions. There is a chance that the interaction between the UC-MSCs and the biologic during infusion may have confused the results of the study and impacted the PASI score of patient 1. It is also possible that patient 1 may have continued to improve, but her observation window was too short. Patient 1 was observed for 62 weeks post-infusion, while patient 2 was observed for 105 weeks post-infusion, and patient 3 was observed for 80 weeks post-infusion. Due to the observation window being shorter for patient 1, it is unclear whether she would have continued to improve and reach remission in the following weeks, as observed in patients 2 and 3.

Going forward

For future studies, the intention for MSC therapies going forward includes finding a way to narrow down the concentration of administered MSCs across patients. Additionally, it is currently being investigated whether GMP can be the universal cell source for clinical immunomodulation therapy, as they are working on developing pluripotent MSCs with the capacity for mass production that will be more accessible for treatments. The development of a manufactured stem cell could revolutionize stem cell therapies. Doing this would increase the availability of stem cells for treatment and ensure more people receive the care needed to undergo therapy for their ailments. Additionally, without being limited in the number of stem cells, more research in stem cell therapies could take place, and treatments could be studied more extensively and made more effective so that those undergoing stem cell therapies would hopefully be able to achieve better results.

In addition to this, another future goal of stem cell therapy includes the effort to remove biologic treatments altogether. Biologic injections are expensive and only effective for certain populations of people with psoriasis. Current stem cell treatments still use biologics to lower PASI scores, meaning patients are paying for injections in addition to paying for the administration of stem cells. The major takeaway from current clinical trials is the increased sensitivity to biologics after receiving stem cell infusions. In the future, researchers are hopeful that stem cell therapy will become a cure for the condition, rather than a booster for an already administered treatment.[18]

Finally, the current sample populations undergoing stem cell therapies in clinical trials are small and limited to biologically resistant psoriasis patients. Additionally, the samples are also limited to those with very severe cases of psoriasis for long periods of time. As demonstrated by the study with the three women, this sample included three women, all above the age of 40, who have struggled with psoriasis for many years. This is not necessarily representative of the entire population of people with psoriasis. Going forward, the intention is to increase sample populations to other patients struggling with the disease. Examples of this would be younger patients, patients undergoing biologic treatment with success, patients with moderate psoriasis, etc. The reason for this is that the disease is currently without a cure, and current treatments target symptoms rather than curing the disease. Psoriasis affects millions of people and severely affects the quality of life for those afflicted. Researchers hope to be able to reach more people with stem cell treatment in the future.[19]

Summary and conclusions

Psoriasis remains difficult to treat due to the chronic systemic inflammation it causes, increased numbers of biologic-resistant psoriasis cases, and due to the high cost of current treatments, and limited control of the illness.

Treatments of psoriasis with mesenchymal stem cells provide many advantages for those with psoriasis, including their immunomodulatory characteristics, as they secrete anti-inflammatory cytokines and suppress pathogenic T cell activity. They are also capable of tissue regeneration and repair through the secretion of growth factors. Based on the results from the clinical trial, it is evident that MSCs can increase sensitivity to biologics in biologic-resistant psoriasis patients, which demonstrates promising results for treating the disease going forward. MSCs have also been shown to be safe for the duration of treatments, as no major adverse effects were reported in this clinical study or many others post-infusion.

The clinical study presents encouraging results, as all three patients showed significant improvement following infusion with MSCs. Key takeaways from this study include that all patients showed improvement immediately after their infusion, with one patient showing improvement after only her first infusion. Additionally, the increased sensitivity to biologics is also a key component of this study. All three patients in the study showed improvement after the combination of treatment with MSCs and biologics, which previously did not improve their PASI scores. This shows that biologic-resistant psoriasis patients who previously had no effective treatments now have a promising future to treat their disease.

Treatment of psoriasis with mesenchymal stem cells is still very early in clinical trials and still has a long way to go. Clinical trials in the future will need to expand sample sizes, narrow down the dosage of administered MSCs, examine the role of GMP in psoriasis treatment, and aim to reduce or replace biologic treatments altogether. Although treatment of psoriasis with MSCs is still in its early stages, the evidence that is accumulating suggests that it may be the first simple disease-modifying therapy for psoriasis as opposed to another symptom-targeting treatment.

Acknowledgements

None

Conflicts of interest

The authors declare that there are no conflicts of interest.

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