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Managing Cyclosporine-Induced Sebaceous Hyperplasia in a Renal Transplant Patient: Efficacy of Oral Isotretinoin

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Abstract

Sebaceous hyperplasia (SH) is a benign proliferation of sebaceous glands that can pose aesthetic concerns and treatment challenges, especially in renal transplant recipients, where lesions may be larger and more diffuse. This report presents a 33-year-old male, 18 years post-kidney transplant, with a two-year history of facial papules on an immunosuppressive regimen of cyclosporine, prednisone, and mycophenolate. Treatment with isotretinoin 10 mg daily resulted in significant improvement. Isotretinoin, known for its antiproliferative and sebostatic effects, has demonstrated safety and efficacy without impacting renal function. This case supports low-dose isotretinoin as a potential treatment for SH in renal transplant patients.

Keywords: Isotretinoin; Renal transplant; Immunosuppression; Sebaceous hyperplasia; Sebaceous hyperplasia treatment; Sebaceous hyperplasia treatment in immunosuppressed patients

Introduction

Sebaceous hyperplasia (SH) is a benign proliferation of the sebaceous glands which can cause significant aesthetic concerns for patients and can prove difficult to eradicate. SH may occur more frequently after kidney transplantation, and SH lesions may also be larger in size and more diffuse as compared to SH in the general population [1]. Oral isotretinoin has been reported as an effective treatment for SH in both immunosuppressed and immunocompetent patients [2-5]. Herein we report the successful treatment of SH with isotretinoin in a renal transplant recipient with a review of the literature.

Case Presentation

A 33-year-old male with a history of kidney transplant eighteen years prior was referred to dermatology for evaluation of a two-year history of facial papules, over which he experienced significant emotional distress. He was prescribed benzoyl peroxide wash without improvement. Treatment with cosmetic laser was not feasible for this patient due to high costs. His immunosuppressive regimen included cyclosporine, prednisone, and mycophenolate.

Physical exam showed numerous 1-2 mm yellow- to skin-colored lobulated papules with central dell on the face, consistent with SH (Figure 1A). He was started on isotretinoin 10 mg daily, with monthly laboratory monitoring given his history of chronic hypertriglyceridemia secondary to cyclosporine. By the second month of treatment, he had noted 50% improvement in his SH (Figure 1B) without any side effects. He remained on isotretinoin for a total of fourteen months with continued clinical improvement, with few brief interruptions in treatment for temporary hypertriglyceridemia attributed to cyclosporine. At that point, transplant rejection secondary to cyclosporine toxicity was suspected, and the patient was switched to tacrolimus as part of his immunosuppressive regimen. Isotretinoin was then discontinued due to adequate regression of the lesions and termination of cyclosporine, the offending agent. After discontinuation of both agents, the patient developed a few new SH lesions, but these subsequently plateaued and had not developed further by dermatological follow up four months later.

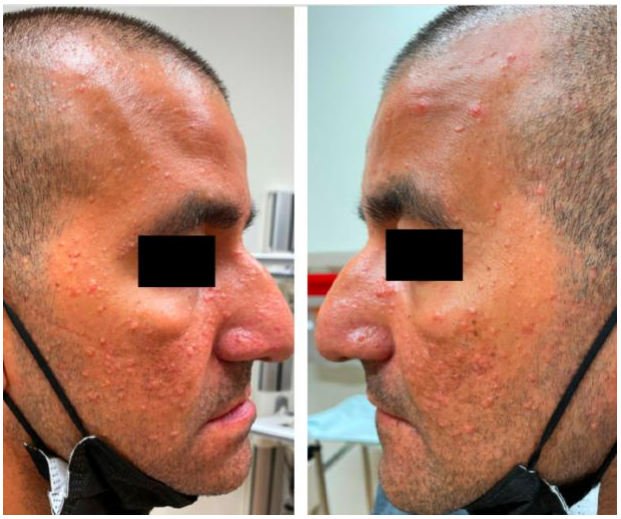

Figure 1A: Numerous sebaceous hyperplasia lesions on the patient's scalp, forehead, temples, nose, cheeks, and chin prior to isotretinoin treatment.

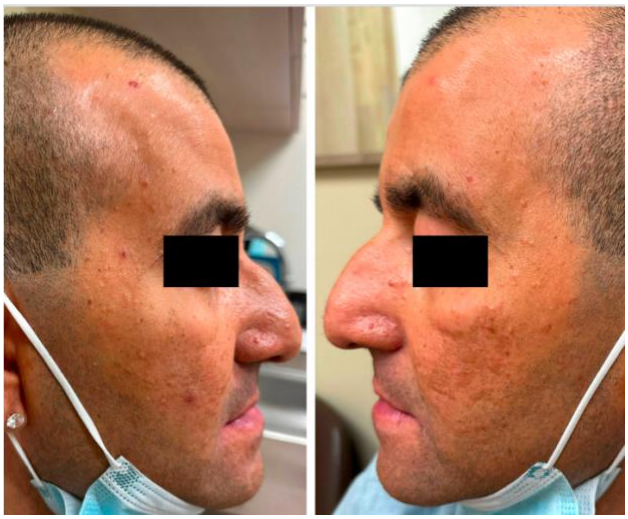

Figure 1B: Patient with a significantly reduced number of sebaceous hyperplasia lesions after two months of isotretinoin treatment.

Discussion

Cyclosporine is commonly used after renal transplantation to promote transplant survival, but has also been associated with mucocutaneous side effects including SH [1]. In one study of 117 renal transplant patients, 91 (77.8%) received cyclosporine and 35 (29.9%) developed SH [1]. Cyclosporine-induced SH may be due to stimulation of undifferentiated sebocytes, increasing the size of the sebaceous gland, sebum secretion, and 5-a reductase activity [3,5]. In most cases SH develops years after renal transplantation, with an average time to onset of 11.25 ± 4.5 years (Table 1). Treatments for SH include photodynamic therapy, cryotherapy, carbon dioxide laser, electrodessication, and excision [3,5]. However, possible complications of these methods include atrophic scarring and dyspigmentation, as well as high associated costs [3]. Isotretinoin represents a potential treatment alternative for SH in renal transplant recipients.

Isotretinoin's efficacy against SH is theorized to be due to its antiproliferative and sebostatic effects, inducing apoptosis and cell cycle arrest in sebocytes [3,4]. In all reported cases, isotretinoin effectively treated SH in renal transplant patients with no reported side effects except for mild cheilitis in 2 out of 5 cases (Table 1) [2-5]. Isotretinoin may be associated with proteinuria, acute rhabdomyolysis, myoglobinuria, and isolated elevation of serum creatine kinase, however, these effects did not appear to impact renal function in a recent systematic review [6]. In our case, the patient experienced brief episodes of worsening hypertriglyceridemia, but this was attributed to the cyclosporine itself rather than to the isotretinoin. Interestingly, two studies have proposed renoprotective effects of isotretinoin against both acute and chronic allograft nephropathy in animal models, thought to be due to isotretinoin's anti-proliferative, anti-fibrotic, immunosuppressive, and anti-inflammatory actions [6]. Notably, in the accumulated literature and in our patient, no adverse effects on renal graft function secondary to isotretinoin use were reported, further supporting isotretinoin as a treatment option for SH in renal transplant recipients [2-5].

Table 1: Review of Reported Cases of Sebaceous Hyperplasia (SH) in Renal Transplant Patients Treated with Systemic Isotretinoin.

Author	**Patient Demographics**	**Time to onset of SH (from date of transplant)**	**Immuno-suppressive Regimen**	**Isotretinoin Regimen**	**Other attempted treatments for SH**	**Adverse Effects**	**Recurrence**
Burton et al.[2*]	39 M	Unknown	Prednisone 10 to 15 mg daily and 100 mg azathioprine daily	Isotretinoin 40 mg twice daily for two weeks, then two-week taper, then 40 mg every other day indefinitely	Astringent cleansers, benzoyl peroxide, topical trans-retinoic acid, systemic antibiotics, electrodesiccation, cryosurgery, and topical 50% trichloroacetic acid	None	SH recurrence after taper, none after reinstitution of isotretinoin

McDonald et al.[3]	56 M	15 years	Cyclosporine 60 mg twice daily, azathioprine 75 mg daily and prednisolone 5 mg daily	Isotretinoin 10 mg daily	None	Mild cheilitis	No recurrence, maintenance dose of isotretinoin 10 mg daily
	45 M	9 years	Cyclosporine (dosage unknown) for 9 years after transplant, discontinued Prednisolone 5 mg daily, tacrolimus 3 mg twice daily and mycophenolate mofetil 1.5 g daily	Isotretinoin 20 mg daily	None	None	No recurrence, maintenance dose of isotretinoin 20 mg daily
Jung et al. [4]	40 M	Unknown**	Cyclosporine 350 mg and prednisolone 7.5 mg for 11 years after transplant, discontinued	Isotretinoin 20 mg daily for two months, two-month	None	None	SH recurrence two months after initial treatment, no recurrence for 9 months after

			Tacrolimus 6 mg daily and prednisolone 5 mg daily for 4 years	break, then second treatment with isotretinoin 20 mg daily for two months			second treatment
Caytemel et al. [5]	36 F	5 years	Cyclosporine 75 mg daily, azathioprine 75 mg daily, and methylprednisolone 6 mg daily	Isotretinoin 40 mg daily for two months, then 20 mg daily for 4 months	None	Mild cheilitis	No recurrence one year after discontinuation of therapy
This case	33 M	16 years	Cyclosporine 100 mg twice daily, mycophenolate 1000 mg twice daily, prednisone 5 mg daily	Isotretinoin 10 mg daily for 14 months	Benzoyl peroxide wash	Temporary elevation in triglycerides	Mild SH recurrence after discontinuation of therapy

*This patient was not treated with cyclosporine, instead was treated with 10-15 mg prednisone and 100 mg azathioprine daily. Onset of SH also unknown, reported as patient gradual onset following renal transplant 13 years prior to initiation of isotretinoin treatment.

**SH onset during 11-year period of cyclosporine and prednisolone treatment following renal transplant.

Our patient underwent an extended course of isotretinoin, which is unique amongst the reported cases to date. Though recurrence of SH after stopping isotretinoin treatment has been reported in treatment regimens of less than two months, each case was effectively treated upon restarting the drug [2,4]. Notably, our patient experienced rapid improvement in his lesions after only a few months on low-dose isotretinoin, which he repeatedly expressed had significantly enhanced his quality of life. Despite his complex medical history, including a kidney transplant, the isotretinoin regimen proved to be a worthwhile endeavor for him.

Conclusion

This case highlights SH as a complication of immunosuppressive therapy in renal transplant recipients and demonstrates the potential of low-dose isotretinoin as a safe, effective, and viable treatment option for managing this stubborn condition.

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