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DISEASES OF THE ESOPHAGUS

Case report

Lichen planus esophagitis: report of three patients treated with oral tacrolimus or intraesophageal corticosteroid injections or both*

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SUMMARY. **Clinically significant involvement of the esophagus is uncommon in patients who have lichen planus, a common disorder of squamous epithelium. In three patients who had oral, cutaneous, and esophageal lichen planus, endoscopic intralesional esophageal injection of corticosteroids (in all three patients) and oral tacrolimus (FH506) (in two patients) resulted in improvement in dysphagia, a less frequent need for dilation, and improvement in esophageal inflammation.**

INTRODUCTION

Lichen planus is a common disorder of squamous epithelium. Clinically significant involvement of the esophagus is uncommon. We describe three patients with oral, cutaneous, and esophageal lichen planus. Endoscopic intralesional esophageal injection of corticosteroids in all three patients and oral tacrolimus (FK506) in two patients resulted in considerable improvement in dysphagia, a less frequent need for esophageal dilation, and improvement in esophageal inflammation.

REPORTS

Case 1

A 69-year-old woman was referred to one of the authors (R.F.K.) in 1992 with progressive dysphagia. Esophagoscopy showed a proximal, band-like stenosis with white exudate (Fig. 1). Brushings demonstrated fungal elements; biopsies showed inflammation. The patient was managed with periodic dilations; the fungal infection was treated with fluconazole and nystatin. At 1 year after initial evaluation, a rash (Fig. 2) and oral lesions, consistent with lichen planus, were noted. Histologic examination of a cutaneous lesion on the neck was consistent with lichen planus (Fig. 3). Esophageal biopsies demonstrated changes suggestive of esophageal lichen planus (Fig. 4). Treatment with etretinate, adrenocorticotropic hormone, griseofulvin, and prednisone did not produce long-lasting improvement in symptoms.

In September 1995, we initiated therapy with endoscopically directed intralesional injections of 2–5 mg of triamcinolone acetate. All injections were performed by one of the authors (R.F.K.), under direct vision, using a sclerotherapy needle and a solution of triamcinolone diluted to a concentration of 2.5 mg/mL. Increments of 0.4 mL were injected. This low dose was chosen to minimize risk and because it had been shown to be effective in pediatric patients with esophageal stenoses.[34] Subsequent to this intervention, there was an improvement in swallowing and a longer interval between esophageal dilation sessions. During the 3 years before corticosteroid injection, the esophagus was dilated on 18 separate occasions (six dilations per year); in the 5 years after the first injection (October 1995), there were five injections and five dilations. Despite active cutaneous disease, the patient has not required esophageal dilation or injection for 2 years. Her stricture is more widely patent and surrounded by less inflammation (Fig. 5). Intralesional corticosteroid

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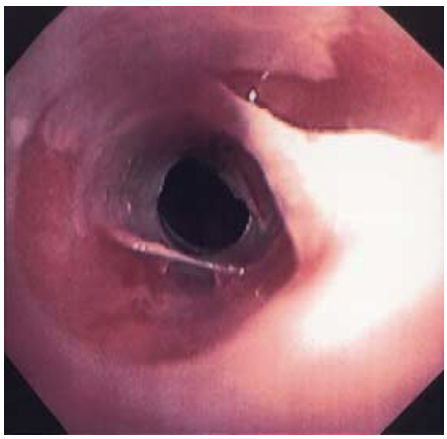

Fig. 1 (Case 1). Endophotograph taken at presentation to our institution in 1992, demonstrating a proximal esophageal stenosis with a pseudomembrane. The membrane easily pulled off the surface. Figure 4 shows the findings on subsequent biopsy of this area.

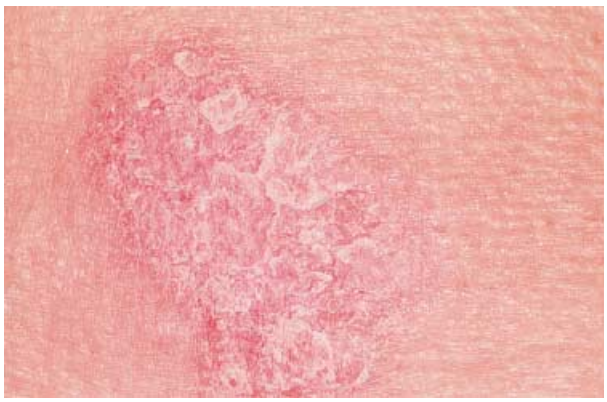

Fig. 2 (Case 1). Cutaneous lesion, demonstrating an irregular pink papule. Figure 3 shows the findings on biopsy of a similar lesion.

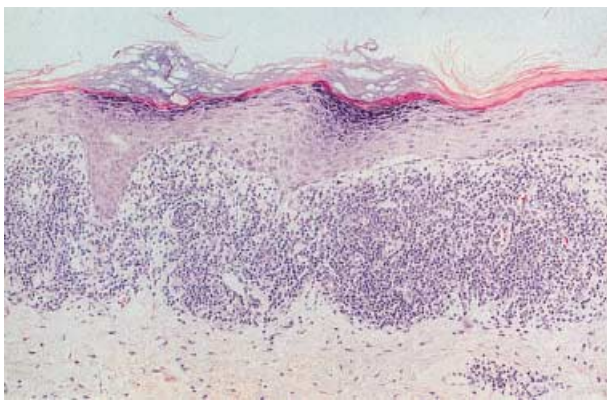

Fig. 3 (Case 1). Skin biopsy specimen, demonstrating irregular acanthosis, hyperkeratosis, focal hypergranulosis, damage to the basal layer, and a band-like dermal infiltrate. Vacuolar degeneration of the basal epithelium is the classic finding in cutaneous lichen planus. (Hematoxylin-eosin; ×40)

injections were more successful than any other treatment. A summary of the patient's treatment is given in Table 1.

Case 2

A 66-year-old woman presented to the dermatology clinic with a 2-year history of an erythematous rash and mucosal erythema accompanied by redness and swelling and abnormal sensation of the mouth and vaginal area. Examination showed erythematous lichenoid papules on the dorsal forearms; the tongue was slightly hyperkeratotic with deep fissures. The vaginal mucosa was beefy red and tender. Punch biopsies showed lichenoid changes, and special stains were negative for fungal organisms. Immunofluorescence was consistent with a lichenoid reaction. Hepatitis C antibody testing was negative. Serum antibodies were positive for extractable nuclear antigen. She was treated with griseofulvin and fluocinonide gel; 6 months later, scalp and more severe cutaneous involvement occurred. Further skin biopsies demonstrated lichenoid changes. Direct immunofluorescence demonstrated granular but patchy deposition of C3 at the basement membrane; there were insufficient changes to suggest that this was a lupus band. The final interpretation was suggestive of lichenoid reaction.

One year later, dysphagia occurred. A videoesophagram demonstrated a proximal esophageal stenosis at the level of the fifth and sixth cervical vertebrae. A pill easily lodged at this stenosis. Endoscopy demonstrated moderate distal esophagitis, pearly white exudate in the mid-esophagus, and a pseudomembrane with stenosis at the proximal esophagus. Biopsies showed necroinflammatory ulcer debris and entrapped glandular mucosa; special stains for fungus were negative. Immunodermatopathologic examination was suggestive of lichen planus: direct fluorescence showed a weak discontinuous granular basement membrane zone deposition of C3 and fibrinogen. Only superficial tissue was present.

Treatment was begun with endoscopically administered intralesional triamcinolone acetate. The patient received six endoscopic injections of triamcinolone acetate in doses from 4 to 20 mg per session and six wire-guided bougienage sessions. Initially, a solution concentration of 2.5 mg/mL was used. Because of persistent symptoms, the concentration was increased to 5.0 mg/mL for the most recent three injection sessions.

Approximately 6 months later, oral tacrolimus in Aquaphor 0.1% was added to the patient's regimen. She applies it to oral areas of active inflammation and then is encouraged to swallow the remainder. With the combination of intralesional triamcinolone and oral tacrolimus, her dosage of oral prednisone has been tapered to 20 mg every other day. She continues to have active inflammation and an esophageal stenosis, but she has improved symptomatically. Her clinical course is summarized in Table 2.

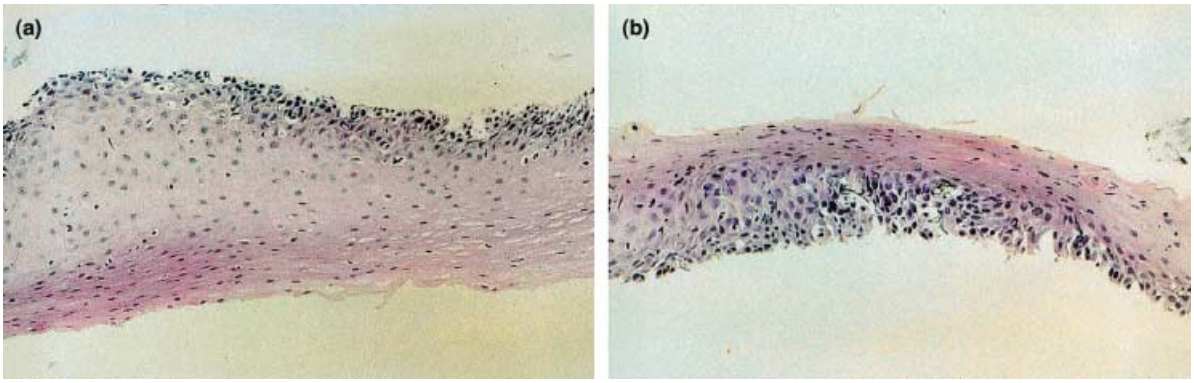

Fig. 4 (Case 1). (a and b) Photomicrographs of an esophageal biopsy specimen. In esophageal lichen planus, the mucosa can be thickened (acanthosis) or thinned. (a) Mild acanthosis. (b) Thinned. Both figures show parakeratosis (nuclei in upper layers) but no granular layer or hyperkeratosis, as is typical of this disease in oral or esophageal mucosa. The underlying stroma is not present, but damage and an infiltrate along the basal layer are seen. (Hematoxylin-eosin; ×100).

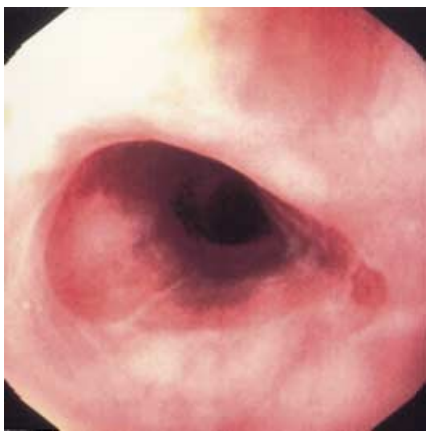

Fig. 5 (Case 1). Endophotograph of the esophagus obtained 7 years after presentation shows improvement in the diameter of the esophageal lumen and less severe inflammatory changes.

Table 1. Summary of treatments for cutaneous and esophageal lichen planus in Case 1

	Treatment	
Year	Dilation no.	Other
1992	6	Nystatin, fluconazole, etretinate, omeprazole
1993	5	Adrenocorticotropic hormone
1994	4	Griseofulvin, H_2 blockers
1995	5	Prednisone, 40 mg/day and tapered
1996	1	One esophageal injection, one intramuscular injection of betamethasone
1997	1	One esophageal injection
1998	2	Two esophageal injections
1999	0	None
2000	0	None
2001	0	None

Case 3

A 77-year-old woman was examined by one of the authors (R.F.K.) in March 2001. She had previously been evaluated at our clinic by a colleague in 1988, at which time she had a 5-year history of lichen planus and a history of proximal esophageal stenoses. Frequent dilations had been performed, and she had a history of Nissen fundoplication for reflux esophagitis.

Table 2. Summary of treatments for cutaneous and esophageal lichen planus in Case 2

	Treatment	
Year	Dilation no.	Other
1999	4	Prednisone (20–40 mg/day), four esophageal injections, omeprazole (20 mg)
2000	4	Prednisone (5 mg/day), tacrolimus ointment, two esophageal injections
2001	3	January–March: tacrolimus ointment, omeprazole (40 mg), prednisone (20 mg/day every other day), one esophageal injection

In December 1988, lichen planus of the vulvovaginal wall was diagnosed at our institution. The patient was examined by a dermatologist, who confirmed oral, vaginal, and probable esophageal lichen planus. She was given fluocinonide gel for the vulva. The mouth and vulva irritation improved, and the fluocinonide therapy was tapered. She returned in 1995 with oral erosive lichen planus. She was given adrenocorticotropic hormone injections and fluocinonide ointment with dexamethasone, which she was instructed to spit and swallow three times a day after meals and at bedtime. Her oral lesions improved after administration of adrenocorticotropic hormone. Zinc was added to her regimen after the serum zinc level was found to be low. In February 2000, oral tacrolimus was prescribed. During the 12-year span between 1988 and 2001, the rate of esophageal dilations was 0.5–1.5 dilations per year. A flare of esophageal lichen planus occurred in early 2001. Initially, the 10-mm endoscope could not go through her proximal stenosis. Three dilation sessions were performed over 2 months. At the time of the most recent dilation in April 2001, the esophagus was

injected with 7.0 mg of triamcinolone acetate; the esophageal lumen was dilated to 15 mm. At a 6-month follow up, the patient reported mouth pain. Her swallowing had improved.

DISCUSSION

Clinical and endoscopic findings

Lichen planus is an inflammatory papulosquamous disorder affecting less than 1% of the general population. In addition to glabrous skin involvement, painful oral and gingival lesions occur frequently. Involvement may take many forms: there may be a single localized lesion or multiple lesions with cutaneous, oral, and genital involvement. The classic skin lesion is a violaceous polygonal papule. Oral involvement includes atrophic lesions, erosions, and lace-like reticulated plaques. The differential diagnosis of oral lichen planus includes ulcerative stomatitis and immunobullous diseases, including pemphigus and pemphigoid. Oral lichen planus appears to be associated with an increased risk of oral malignancy.[3–7] Lichen planus is associated with liver disease (including hepatitis C[8] and cholestatic and autoimmune hepatitis), ulcerative colitis, diabetes, hypertension, and certain dental amalgam restorations. Numerous medications are implicated in lichen planus and lichenoid eruptions, including thiazide diuretics, non-steroidal anti-inflammatory drugs, and allopurinol.[3,4]

The precise cause of lichen planus is unknown. Pathophysiologically, it resembles graft-versus-host disease. Activated T-lymphocytes attack an antigen in the basal epithelium. Autoantibodies and humoral immune mechanism have not been implicated.[3–5,7]

The frequency and characteristics of esophageal involvement in patients with cutaneous lichen planus were studied by Dickens *et al*.[9] They evaluated symptoms and dermatologic and esophageal findings in a cohort of 19 patients with a diagnosis of lichen planus. Five patients had esophageal findings. Only one of these had dysphagia; this patient was the only patient with severe erosive esophagitis. The remaining four patients had only subtle esophageal papules.[9] The majority of these patients were asymptomatic. That study suggests that asymptomatic esophageal involvement is common but that symptomatic esophageal involvement occurs in less than 10% of patients with lichen planus. The prevalence of esophageal lichen planus was studied by Eisen,[10] who retrospectively studied 584 patients with documented oral lichen planus. In contrast with the high prevalence reported by Dickens, Eisen found only eight patients who complained of dysphagia and underwent endoscopy. Out of these patients, only four had endoscopic and histologic abnormalities consistent with esophageal lichen planus.

Endoscopic findings in esophageal lichen planus include elevated lacy, white papules, esophageal webs, pseudomembranes, desquamation, and superficial pinpoint erosions with and without stenosis.[9–16] Poor distensibility of the esophagus also has been described.[16] Simultaneous oral and mucous membrane involvement is common.[9–11] *Candida* esophagitis complicating lichen planus (as in our Case 1) has been described previously.[17]

Lichen planus-associated esophageal stenosis should be suspected when the following are present: (1) occurrence in a middle-aged woman; (2) other erosive mucosal lesions; (3) location of stenosis in the upper third of the esophagus; (4) histologic changes consistent with lichen planus; (5) flare-up of buccal lesions after esophageal dilation; and (6) dramatic improvement with systemic steroids.[11] All of our patients had oral and mucous membrane disease, however, their oral lesions did not flare after esophageal dilation. In contrast with oral lichen planus, no known progression of esophageal lichen planus to esophageal malignancy has been described.[12] One author suggested that surveillance of the esophagus for development of squamous cell carcinoma may be indicated.[13]

Treatment

Many treatments have been successful for esophageal lichen planus. Dilation is commonly used despite concerns about worsening esophageal stenosis or oral lesions due to Koebner phenomenon. Other regimens described include adrenocorticotropic hormone injections, oral corticosteroids, etretinate, and topical corticosteroids.[1,11–23] Topical tacrolimus, a potent inactivator of T-lymphocytes used initially in patients with transplants, was successful for oral lichen planus in six patients.[24–26] Table 3 summarizes data for 24 patients with esophageal lichen planus (including our three patients).

Intralesional corticosteroid injections have been used to treat refractory esophageal strictures in adults and children.[2,27–36] Peptic strictures, postoperative condition, post-radiation condition, and lye ingestion were the most common indications. The dose used ranged from 3 mg to 80 mg of triamcinolone acetate per injection session. One series was a controlled trial comparing corticosteroid injection plus dilation with dilation alone in patients with peptic strictures; that report, available in only abstract form, suggested that injection plus dilation was superior to dilation alone.[29] One report[33] recommended the use of an endoscopic ultrasound probe to direct injections more accurately. No patients with lichen planus-associated esophageal stenosis are described in these reports. Table 4 summarizes the available information (including from this report) regarding corticosteroid injection for esophageal stenoses.

Table 3. Summary of reported data for patients with esophageal lichen planus

Reference	Clinical presentation	Treatment	Result
Sheehan-Dare *et al.*[13]	Dysphagia	Prednisone 20 mg/day	Improved
Al-Shihabi and Jackson[18]	Dysphagia, sore mouth, genital lesions	Dilation, ACTH	Improved
Lefer[16]	Dysphagia, sore mouth	Not reported	Not reported
Jobard-Drobacheff *et al.*[11]	Two patients, dysphagia	One, corticosteroid; one, etretinate, dilations, oral, topical corticosteroids	Improved
Van Maercke *et al.*[19]	Dysphagia, oral lesions	Dilation × 6, oral etretinate, acid suppression	Dysphagia improved
Torrelo *et al.*[20]	Dysphagia, use of cyanamide	Oral prednisone	Improved
Yoon and Sullivan[25]	Heartburn, odynophagia	Home dilation, acid suppression	Remained active
Coelho-Borges *et al.*[14]	Dysphagia, odynophagia, oral lesions	Oral prednisone	Improved
Holder *et al.*[12]	Dysphagia, stenosis	Balloon dilation	Improved
Celinski *et al.*[26]	Xerostomia, dysphagia, white plaque	Not reported	Not reported
Kirsch[15]	Dysphagia, cervical esophageal stricture	Dilation	Improved
Souto *et al.*[23]	Dysphagia, oral and genital lesions	Oral corticosteroids	Disease present, symptoms better
Evans *et al.*[21]	Dysphagia, vulvar and cutaneous involvement	Dilations every 2 months and oral pulse methylprednisolone at high doses (500–1000 mg)	Somewhat improved (still needs dilations)
Bobadilla *et al.*[22]	Dysphagia, odynophagia, genital involvement	Cyclosporin 2 mg/kg per day	Clinical remission and improvement of endoscopy findings for 1 year, taper of cyclosporin planned
Harewood *et al.*[1]	Six patients, dysphagia, oral lesions (5/6), genital lesions (3/6)	Oral prednisone (4/6 patients) 40–60 mg/day	Improved; one patient required maintenance prednisone 10 mg/day
This study	Case 1: dysphagia, oral, cutaneous, and genital lesions	Case 1: etretinate, ACTH, oral corticosteroids, esophageal corticosteroid injection	Case 1: Improved symptoms and esophageal stenosis; less frequent dilation
	Case 2: dysphagia, oral, genital, and cutaneous lesions	Case 2: prednisone, griseofulvin, oral tacrolimus, esophageal corticosteroid injection	Case 2: improved symptoms, less frequent dilation
	Case 3: dysphagia	Case 3: oral prednisone, oral tacrolimus, corticosteroid injection	Case 3: 6-month follow-up only; probably improved

ACTH, adrenocorticotropic hormone.

Table 4. Treatment of esophageal stenoses with intralesional corticosteroids

		Triamcinolone therapy		
Reference	No. of patients, cause	Dose, mg	Sessions	Results
Holder *et al.*[27]	10 children: 3 anastomotic 7 caustic	40	Up to 15 injections and dilations each	50% improved, 1 perforation with abscess
Mendelsohn and Maloney[31]	8 adults and children: 2 anastomotic 1 nasogastric tube 5 caustic ingestion	Up to 80	Variable	5/8 better
Gandhi *et al.*[34]	12 infants and children:			
	6 lye ingestion	4	2–13 injections	6/6 improved, 1 contained perforation
	5 postop	4	1–6 injections	6/6 improved
	1 unknown	4	1–6 injections	6/6 improved
Kirsch *et al.*[35]	2 peptic	20	1 injection and dilation; 1 dilation each before injection	Improved
Berenson *et al.*[28]	1 lye stricture	3–10	8 dilations and injections	Improved
Burdick *et al.*[2]	7 patients: 4 anastomotic, 3 peptic	125	15–21 dilations, crossover trial, 3 injections in 6 months	Steroid better than saline injection
Rupp *et al.*[29]	14 peptic (14 control)	80	1 session	Injection was superior to no injection
Bhutani *et al.*[33]	3 patients: 1 post xrt, chemo 1 peptic 1 anastomotic	80	1 injection with EUS miniprobe, 1–3 dilations	Improved
Lee *et al.*[32]	29 patients: 12 peptic 8 anastomotic 1 pill-induced 1 sclerotherapy 1 lye ingestion 6 radiation	Mean, 28		Improved
Zein *et al.*[30]	7 infants: 3 peptic 1 radiation 1 lye ingestion 1 anastomotic 1 congenital malformation	8	2–7	5 improved (transient fungal esophagitis developed in 2)
Kochhar *et al.*[36]	17 patients (13–52 years): 13 acid 4 alkali	20–30	1–3 injections	Improved symptoms and decreased dilations per year
This study	1 lichen planus	2–5	4 injections/5 years	Improved symptoms (3) and endoscopic findings (1 of 2 examined), less frequent dilations (3/3)
	1 lichen planus	2–26	11 injections/3 years	
	1 lichen planus	7	Injection in May 2001	

SUMMARY

We report our experience with three patients with lichen planus-associated esophageal stenosis. Unique aspects of the treatment of these patients are the use of intralesional injections of triamcinolone acetate into the esophagus and the use of tacrolimus ointment orally in two patients. All patients improved, requiring fewer dilation sessions with therapy. The dramatic improvement in Case 1 is the most striking. The patient noted a dramatic reduction in the frequency of dilation and improvement in dysphagia and esophagitis after therapy with low-dose intralesional triamcinolone acetate. In Case 2, the patient experienced a symptomatic response to steroid injections and dilation. This symptomatic improvement was enhanced by the addition of oral tacrolimus and by increasing the dosage of injected triamcinolone. In Case 3, the patient has been treated in this manner for only a few months. Intraesophageal corticosteroid injection and oral tacrolimus should be considered in patients with symptomatic

lichen planus-associated esophagitis. With this treatment in combination with esophageal dilation, patients and treating physicians can expect an improvement in symptoms and a decrease in dilation sessions.

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