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# Remission of refractory esophageal lichen planus induced by tofacitinib

## Remission eines refraktären Lichen planus im Ösophagus durch Tofacitinib

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### ABSTRACT

Esophageal involvement in lichen planus (ELP) is an often overlooked and underreported aspect of lichen planus (LP), a prevalent dermatological disorder with an autoimmune basis. Reproducible endoscopic findings such as mucosal denudation, tearing, and trachealization, along with histological features like band-like T-cell infiltration, mucosal detachment, epithelial cell apoptosis, and dys- or hyperkeratosis, have been documented in recent years. Clinical manifestations of ELP encompass a wide spectrum, ranging from asymptomatic courses to mild to severe dysphagia and, in extreme cases, upper gastrointestinal bleeding. Untreated, chronic ELP can lead to distal esophageal stenosis and may serve as a precursor to esophageal squamous cell carcinoma.

As of now, there exists no established therapy for ELP. Retinoids, which are standard in treating cutaneous LP, do not exhibit positive effects in ELP. While topical glucocorticosteroids often yield favorable responses in esophageal inflammation, some cases prove recalcitrant or refractory. In such instances, various immunosuppressive therapies have been attempted with variable success.

This report details a severe case of ELP that showed resistance to prednisolone, acitretin, alitretinoin, adalimumab, tacrolimus, hydroxychloroquine plus mycophenolate mofetil, and cyclophosphamide. The initiation of the JAK inhibitor tofacitinib induced an impressive clinical, endoscopic, and histological remission. This positive response to a JAK inhibitor is discussed in the context of our evolving understanding of the immune-mediated pathogenesis of this disease.

### ZUSAMMENFASSUNG

Eine Beteiligung des Ösophagus (ELP) ist eine selten diagnostizierte Manifestation eines Lichen planus, einer gut bekannten, wahrscheinlich autoimmun-vermittelten Hauterkrankung. In den letzten Jahren wurden Kriterien zur Diagnose vorgestellt: Bei der Endoskopie sieht man eine Schleimhautablösung, ein Einreißen der Schleimhaut, eine Trachealisierung ähnlich wie bei der eosinophilen Ösophagitis. Die Histologie zeigt eine bandförmige Infiltration mit T-Lymphozyten im Bereich der Basalmembran mit Übergreifen in das Epithelium und einer Ablösung der Schleimhaut, Apoptosen der Epithelzellen, und eine Hyperkeratose, auf deren Boden sich ein Plattenepithel-Karzinom entwickeln kann. Führendes klinisches Symptom ist eine Dysphagie. Symptomfreie Verläufe kommen vor, aber auch Fälle mit oberer gastrointestinaler

Blutung. Eine etablierte Therapie des ELP gibt es noch nicht. Meist spricht die Erkrankung auf topische Kortikosteroide an. In schweren Fällen wurden verschiedene immunsuppressive Therapien angewandt.

Wir beschreiben eine Patientin mit einem schweren ELP, die auf eine Therapie mit topischen oder systemischen Kortikosteroiden, Acitretin, Alitretinoin, Adalimumab, Tacrolimus, Hydroxychloroquin kombiniert mit Mycophenolatmofetil und Cylophosphamid nicht oder kaum ansprach. Nach Gabe des JAK-Inhibitors Tofacitinib kam es zu einer eindrucksvollen klinischen, endoskopischen und histologischen Remission. Wir diskutieren diesen positiven Effekt eines JAK-Inhibitors bei einem ELP in Zusammenhang mit neuen Erkenntnissen über die Immunpathogenese des Lichen planus.

## Introduction

Lichen planus (LP) is a common mucocutaneous disorder affecting 0.5 to 2.0 % of the general population with a notable female predominance [1, 2]. Its manifestations primarily involve the skin, oral, and genital mucosa, with additional occurrences in the nails, scalp, eyes, ears, urinary bladder, and nasal mucosa [3]. Esophageal lichen planus (ELP), while described in case reports and a few case series, has only recently been characterized by reproducible clinical, endoscopic, and histopathological features [4, 5, 6, 7, 8, 9, 10, 11]. Predominantly found in middle-aged women, ELP typically presents with dysphagia as the primary symptom [4, 5, 6, 7, 8, 9, 10, 11]. The spectrum of symptoms varies from asymptomatic cases to mild or severe dysphagia and, in rare instances, to upper gastrointestinal bleeding [10]. ELP should be considered in cases of esophageal bolus obstruction [12]. If left untreated, ELP can progress to squamous cell esophageal carcinoma [13, 14].

For LP, clinical guidelines recommend topical glucocorticosteroids, retinoids, or cyclosporin [15, 16]. In severe or unresponsive cases, a range of interventions has been attempted, including intralesional of systemic glucocorticosteroids, calcineurin inhibitors, sulphasalazine, azathioprine, hydroxychloroquine, methotrexate, mycophenolate mofetil, or TNF inhibitors.

Endoscopically, ELP exhibits mucosal denudation, tearing, and a ring-like appearance of the esophagus, referred to as “trachealization”, resembling the trachea. Prolonged courses may lead to hyperkeratosis and scarring stenosis. Histopathological features include lymphoid inflammatory infiltrates at the interphase between the lamina propria and epithelium associated with mucosal detachment, T-cell spillover into the epithelium, apoptotic squamous cells (Civatte bodies), epidermoid metaplasia, and hyperkeratotic lesions with low- or high-grade dysplasia as a risk factor for invasive squamous cell carcinoma. Despite numerous interventions, there is no established therapy for ELP [10]. Applying the same therapy as for “classical” LP may be successful in most cases. However, refractory courses present a challenge for dermatologists and gastroenterologists. Surprisingly, retinoids, which are part of the standard repertoire of therapy for cutaneous LP, have neither prevented the occurrence of ELP nor reduced its severity [8].

This case report details a patient with severe, progressive, and treatment-resistant ELP unresponsive to nine therapies: topical rather than systemic. Ultimately, tofacitinib, targeting JAK1 and JAK3, induces a remarkable clinical, endoscopic, and histologic remission.

## Case report

A 56-year-old female patient sought care at the gastroenterology and dermatology outpatient clinics, presenting with a constellation of complex symptoms. These included retrosternal pain, dysphagia, and a persistent cough, notably exacerbating by the ingestion of solid food. Additionally, she reported on severe itching of the scalp followed by scarring alopecia and painful burning of the oral and genital mucosa. She had a prior diagnosis of monoclonal gammopathy of undetermined significance and arterial hypertension.

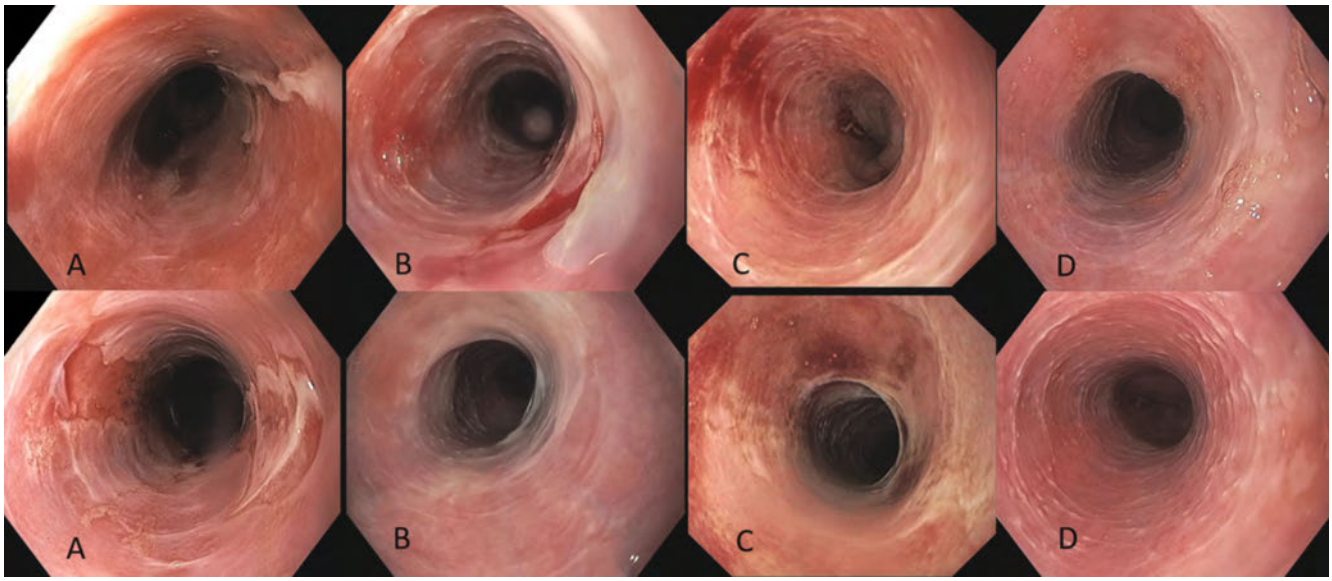
An initial investigation included an external esophagogastroduodenoscopy (EGD), which revealed “oesophagitis of unknown origin”. Subsequent histological examination of an esophageal mucosal biopsy initially prompted suspicion of mucous membrane pemphigoid as the probable diagnosis. Clinically, the patient exhibited erosive mucosal lesions in the oral and genital regions, along with erythematous papules on the scalp, indicative of lichen planopilaris.

Immunological diagnostics, encompassing direct and indirect immunofluorescence, and serological testing for bullous pemphigoid antibodies via ELISA, as well as immunoblotting, yielded negative or no evidence of autoimmune blistering diseases. A repeated EGD and histopathological evaluation now supported the diagnosis of lichen planus affecting multiple sides and the esophagus.

The treatment approach began with a variety of intensive topical therapies. These included application of mometasonfuorate solution to the scalp and tacrolimus 0.1 % ointment for the genital mucosa. Encouragingly, these localized interventions showed positive responses. Oral budesonide gel 0.5 mg BID was tried for treatment of the esophagus [8].

Nevertheless, esophageal symptoms such as dysphagia, retrosternal burning, and tussive irritation showed no improvement. Over the course of almost nine years, alongside topical budesonide, we pursued various systemic interventions. These included prednisolone, acitretin, alitretinoin, adalimumab, tacrolimus, hydroxychloroquine combined with mycophenolate mofetil, and cyclophosphamide (administered in 10 cycles of 750 mg each).

Unfortunately, the majority of these treatments exhibited limited success. Although there were intermittent improvements in oral and genital mucosal lesions and scalp conditions, EGDs consistently revealed persistently active severe ELP**,** as depicted in ▶ **Fig. 1A**, marked by extensive spontaneous mucosal sloughing and tearing. Histological examinations consistently displayed the full-blown characteristics of severe ELP, as illustrated in ▶ **Fig. 2A**.

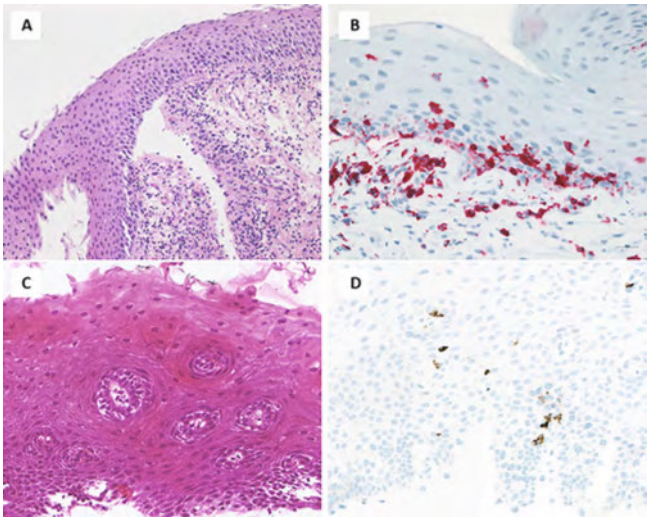



▶ **Fig. 1** Endoscopic aspect of the esophagus. **A** Before start of tofacitinib. The typical aspect of severe ELP is seen: mucosal sloughing, tearing, and trachealization. **B** Six months after start of tofacitinib: mucosal sloughing is still visible. A distal stenosis has developed. **C** Twelve months after start of tofacitinib: marked reduction in mucosal fragility. **D** Fifteen months after start of tofacitinib: no mucosal denudation. No spontaneous tearing. The trachealization is still present. The distal stenosis has abated.

Seven dilations to 12 mm were required to address the esophageal stenosis that gradually developed in the distal third of the esophagus. Eight years after first diagnosis of ELP a critical incident of food impaction occurred, necessitating an emergency procedure for bolus removal. Notably, only the cyclophosphamide treatment cycles provided temporary relief of dysphagia symptoms, with improved swallowing for approximately six months. Unfortunately, we were unable to continue cyclophosphamide therapy due to cumulative toxic hematologic effects and the lack of effect on skin.

After having stopped cyclophosphamide we initiated tofacitinib therapy. It was only when the full dose of 11 mg daily was reached that the esophageal symptoms began to improve. Despite the macroscopic findings six months after initiating tofacitinib (▶ **Fig. 1B**) not yet displaying clear changes compared to findings before (▶ **Fig. 1A**), a subsequent endoscopy twelve months on tofacitinib (▶ **Fig. 1C**) revealed significant improvement. The spontaneous mucosal denudation had notably decreased, although the distal stenosis remained visible, aligning with the patient's symptoms.

The latest endoscopic aspect after fifteen months of treatment is depicted in ▶ **Fig. 1D**: there was no longer spontaneous mucosal sloughing, indicating tight adherence of the mucosa to the submucosa. Only minimal tearing could be seen as an artificial lesion by the endoscope. Notably, the distal stenosis has completely abated. The only remaining characteristic was the marked trachealization of the esophageal wall. Histopathological examination confirmed the macroscopic changes: no mucosal detachment was evident, and the intra- and subepithelial lymphocyte count had normalized. Hyperkeratotic lesions were absent. ▶ **Fig. 2** allows a comparison between the characteristic histologic feature of the esophageal mucosa in biopsies obtained before the




▶ **Fig. 2** Histologic features of esophageal biopsies before initiating (**A, B**) and after 15 months of (**C, D**) tofacitinib treatment. **A** Dense band-like inflammatory infiltrate of the tunica propria spilling over to the squamous epithelium associated with an extended suprabasal epithelial detachment. **B** CD3+ T-cells (red) are the predominant cellular component of the sub- and intraepithelial inflammatory reaction. **C, D** Achievement of clinical improvement is highlighted by marked reduction in inflammation with residual intraepithelial T-cells (brown). Here, the apparent epithelial detachment represents an artefact due to specimen processing. **A, C** hematoxylin and eosin stain; **B, D** immunolabeling using a monoclonal anti-CD3 antibody and a red (**B**) or brown substrate (**D**). Original magnification: A x 10, B x 63, C, D x 40.

start of tofacitinib (A,B) and after 15 months of tofacitinib treatment (C,D), highlighting the excellent therapeutic response.

At the patient's most recent assessments 15 and 22 months under tofacitinib, she reported no more restrictions on food intake. This therapeutic approach has led to a notable enhancement in the patient's overall well-being, marked by weight gain and recovery of her appetite.

## Discussion

Esophageal involvement in LP is an underdiagnosed and underreported manifestation of this common manifold skin disorder. At present, the prevalence of ELP is still unknown. Preliminary data suggest that ELP might be more prevalent than another emerging inflammatory esophageal disease, e. g., eosinophilic esophagitis. Therefore, further research is necessary to determine the true prevalence of ELP. Given the different therapeutic responses of cutaneous and esophageal LP to standard therapy, this suggests possible differences in their underlying pathogenesis.

The immune response in potential immune-mediated skin diseases may be classified into six response patterns [17]. Studies on the composition of the cellular components of the lichenoid infiltrates revealed type-1 lymphocytes (Tc1-cells, Th1 cells, ILC1, NKT, and NK) as the predominant population with reaction to exogenous or self-altered antigens presented by APCs, DCs, or keratinocytes. The Th1-inflammatory response with secretion of proinflammatory cytokines, such as IL-23, IL-17, and IFN-γ, and secretion of the cytotoxic molecules perforin, granulysin, and granzym B seem to promote the main pathogenetic events. After binding to its receptor, IFN-γ activates JAK1 and JAK2 and activates STAT [18] in keratinocytes and infiltrating lymphocytes, mainly STAT-1 [19, 20]. Other authors rather demonstrated JAK2 activation in lymphocytes and STAT-1 activation in keratinocytes in active LP lesions [21, 22]. These data form a rational basis for the use of JAK inhibitors in LP (and in other skin diseases with similar pathogenesis such as psoriasis) [23]. Consequently, several studies confirmed that JAK inhibitors may be a pathogenesis-based rational approach to control LP, particularly in recalcitrant cases, when classical therapy fails [24, 25]. A recent review summarized the outcomes of patients with LP treated with a JAK inhibitor, encompassing the literature up to October 2022 [26]. In the tofacitinib group (JAK1/3), 21 out of 30 patients experienced either complete or partial resolution of the lesions. For baricitinib (JAK1/2), the responding figure was 9 out of 16, for ruxolitinib (JAK1/2) 12 out of 12, and for upatacitinib (JAK1) 2 out of 2. Meanwhile, several JAK inhibitors gained FDA approval for dermatological indications [27]. The current data, comprising case reports and small case series of patients with LP, lacks clear guidance on the preferred choice among available JAK inhibitors. However, targeting JAK1 may be a crucial factor for a successful outcome.

After several futile attempts to control the disease in this patient, we chose to apply the JAK1/3 inhibitor tofacitinib. Additionally, in the present cooperation between dermatology and gastroenterology, we favored this JAK inhibitor in analogy to the therapy of another gastroenterological disease, ulcerative colitis. Interestingly, the initial dose of 5 mg OD had only a very moderate effect. It was only the dose of 10 mg (or 11 mg) daily that showed efficacy, corresponding to published data applied in LP patients and in autoimmune bullous diseases [26, 28]. In ulcerative colitis, 10 mg BID is recommended for induction therapy, while the maintenance dose for this disease is 5 mg BID [29, 30, 31, 32].

The present case is interesting in several aspects: 1. it expands our knowledge on the treatment of ELP, an area where information is currently limited; 2. the present manuscript adds further data to the scant literature that demonstrates a positive response of a recalcitrant ELP to a JAK inhibitor [33, 34]; 3. the use of tofacitinib resulted in a robust macroscopic and histologic response. The esophageal stenosis nearly completely resolved, and clinical symptoms, including dysphagia and retrosternal discomfort, nearly disappeared. This underscores the potential efficacy of JAK inhibitors in managing challenging cases of ELP. Reports such as the present one aim to stimulate further investigations into this intersection of immunological diseases where dermatology and gastroenterology meet.

## Conflict of Interest

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