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Esophageal Lichen Planus as a Cause of Dysphagia: Literature Review and Clinical Observation

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Aim: to analyze the literature data, and to raise awareness of doctors of various specialties about the methods of diagnosis and treatment of esophageal lichen planus (ELP).
Key points. In a 67-year-old female patient with complaints of difficulty swallowing solid food and weight loss, esophagogastroduodenoscopy revealed subcompensated stenosis of the middle third of the esophagus and signs of fibrinous esophagitis. Based on the characteristics of the endoscopic picture and the detection of apoptotic Civatt bodies in esophageal biopsies, a diagnosis of ELP was established. Treatment with glucocorticosteroids led to relief of symptoms and positive endoscopic dynamics. ELP is rare and the least studied, data on this disease in the literature are presented mainly in the form of clinical observations and analysis of series of cases. Typical clinical manifestations include dysphagia and odynophagia. Despite the low prevalence, ELP can be associated with serious complications: stenosis and esophageal squamous cell carcinoma. Endoscopic examination reveals characteristic signs in the esophagus: swelling, thickening and increased vulnerability of the mucosa, often with fibrin, formation of membranes and strictures. The histological picture is represented by epithelial dyskeratosis with exfoliation, lichenoid lymphocytic infiltration. The most specific histological sign is the presence of apoptotic Civatte bodies. Recommendations for the treatment of ELP are limited to the results of a series of clinical observations and include the prescription of systemic corticosteroids. The issue of supportive therapy is the least studied.
Conclusion. Analysis of the literature data and the clinical case demonstrate that lichen planus of the esophagus is one of the rare causes of dysphagia. Characteristic endoscopic and histological signs are key for the diagnosis. The management of patients with esophageal lichen planus is insufficiently defined and today includes taking of glucocorticosteroids, endoscopic dilation of stricture and dynamic endoscopic observation, given the high risk of squamous cell carcinoma in this category of patients.
Keywords: dysphagia, lichen planus, stenotic lesion of the esophagus
Conflict of interest: the authors declare no conflict of interest.

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Красный плоский лишай пищевода как причина дисфагии: обзор литературы и клиническое наблюдение

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Цель: представить клиническое наблюдение и провести анализ данных литературы о методах диагностики и лечения красного плоского лишая (КПЛ) пищевода.
Основные положения. У пациентки 67 лет с жалобами на нарушение глотания твердой пищи и снижение массы тела при эзофагогастродуоденоскопии выявлен субкомпенсированный стеноз средней трети пищевода и признаки фибринозного эзофагита. На основании особенностей эндоскопической картины и обнаружения апоптозных телец Сиватта в биоптатах пищевода установлен диагноз «красный плоский лишай пищевода». Лечение глюкокортикостероидами привело к купированию симптомов и положительной эндоскопической динамике. Эзофагеальная локализация КПЛ является редкой и наименее изученной, данные об этом заболевании в литературе представлены в основном в виде клинических наблюдений и анализа се-

рий случаев. Типичные клинические проявления включают в себя дисфагию и одинофагию. КПЛ может быть связан с серьезными осложнениями — стенозом и плоскоклеточным раком пищевода. Эндоскопическое исследование позволяет выявить в пищеводе характерные признаки: отек, утолщение и повышенную ранимость слизистой оболочки, часто с налетом фибрина, на фоне которых формируются мембраны и стриктуры. Гистологическая картина представлена дискератозом эпителия с отслоением, лихеноидной лимфоцитарной инфильтрацией. Наиболее специфичный гистологический признак — наличие апоптотических телец Сиватта. Рекомендации по терапии КПЛ пищевода ограничены результатами серий клинических наблюдений и включают назначение системных кортикостероидов. Наименее изучена проблема поддерживающей терапии.
Заключение. Анализ данных литературы и приведенный клинический случай показывают, что красный плоский лишай пищевода является одной из редких причин дисфагии. Решающее значение для постановки диагноза имеют характерные эндоскопические и гистологические признаки. Тактика ведения пациентов с красным плоским лишаем пищевода недостаточно определена и на сегодня включает назначение глюкокортикостероидов, эндоскопическую дилатацию при наличии стриктуры и динамическое эндоскопическое наблюдение, учитывая высокий риск развития плоскоклеточного рака у данной категории больных.
Ключевые слова: дисфагия, красный плоский лишай, стенотическое поражение пищевода
Конфликт интересов: авторы заявляют об отсутствии конфликта интересов.

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Lichen planus (LP) is a chronic inflammatory disease of the skin and mucous membranes, less commonly affecting nails and hair, the typical elements of which are lichenoid papules. The incidence of LP is 0.14–1.27 %, middle-aged and elderly women predominate [1].

The etiology and pathogenesis of LP remain poorly understood, currently the dominant idea is that LP is an autoimmune disease mediated by T lymphocytes. It has been suggested that in response to exogenous or endogenous stimuli, Langerhans cells are activated, followed by the presentation of antigen to $CD4^+$ T lymphocytes, which are sent to the lamina propria upon activation of adhesion molecules. The production of proinflammatory cytokines contributes to the stimulation of $CD8^+$ T lymphocytes and further damage to the epithelium [2]. Stress, taking various medications (NSAIDs, antihypertensive drugs, chemotherapeutic agents, hydroxychloroquine, sulfonylureas, tumor necrosis factor inhibitors) and genetic predisposition are considered as predisposing factors to LP [3, 4]. The infectious theory has been widely discussed as a pathogenetic model of LP, but no convincing evidence of this association including with viral hepatitis C has been obtained [5]. A number of studies have shown the combination of LP with other autoimmune diseases (autoimmune thyroiditis, celiac disease) and thymoma [6–9]. The association with primary biliary cholangitis has been demonstrated in isolated cases and is not currently generally accepted [10].

The variety of clinical forms of LP, in addition to skin, nails and hair, includes damage to the anogenital area and esophagus [11]. Esophageal localization of LP is rare and least studied, data on this disease in the literature are presented mainly in the form of clinical observations and analysis of case series. However, a number of authors believe that esophageal LP is not so much a rare disease as a poorly studied disease, the frequency of which in the population can reach 0.1 % [12]. LP can be associated with serious complications – stenosis and squamous cell carcinoma of the esophagus [4, 13].

The presented clinical observation aims to increase the awareness of different specialties doctors about the diagnosis and treatment of esophageal LP.

Clinical observation

A 67-year-old female patient consulted a gastroenterologist with complaints of difficulty swallowing solid food and weight loss. For the first time, dysphagia was noted three months before, when it became necessary to drink liquid after eating solid food. Taking proton pump inhibitors in standard doses had no effect on clinical manifestations: dysphagia persisted, and she lost 10 kg. During fluoroscopy of the esophagus and stomach, the contrast freely passed into the stomach, in the middle third of the esophagus, a narrowing of the esophagus over 4 cm was revealed, and an axial hiatal hernia was detected. She was sent for esophagogastroduodenoscopy (EGD), which confirmed subcompensated middle third esophagus stenosis and signs of fibrinous esophagitis (Fig. 1).

Histological examination of the initial biopsy showed the presence of young granulation tissue

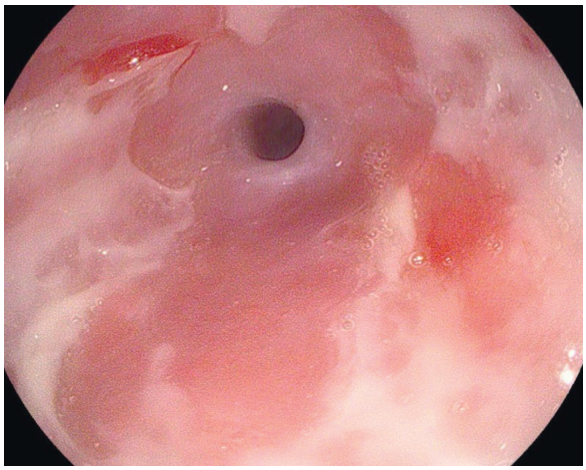

Figure 1. Subcompensated stenosis of the middle third of the esophagus against the background of fibrinous esophagitis

Рисунок 1. Субкомпенсированный стеноз средней трети пищевода на фоне фибринозного эзофагита

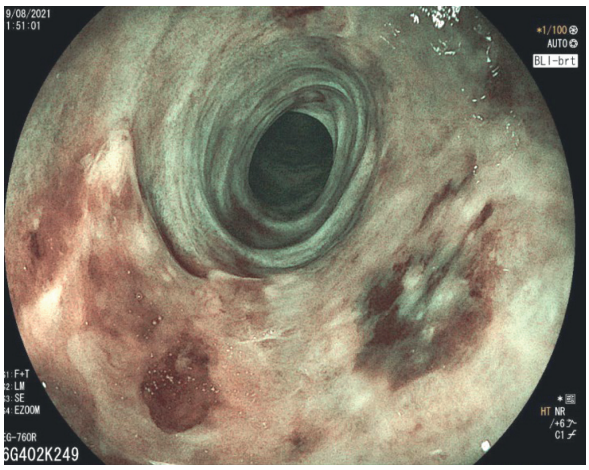

Figure 3. Trachealization of the lower third of the esophagus (inspection in the BLI-brt narrow spectrum mode)

Рисунок 3. Трахеализация нижней трети пищевода (осмотр в режиме узкого спектра BLI-brt)

with fibrinous-purulent exudate and intraepithelial neutrophilic infiltration of stratified squamous epithelium. In a hospital setting, the patient underwent six sessions of endoscopic bougienage using a Savary bougie guide with a diameter of 7 to 14 mm, with a positive effect. A main feature of the endoscopic picture was the localization of circular stenosis in the middle esophagus, as well as increased fragility of the surrounding mucous membrane, which, upon contact with the endoscope, ruptured and peeled off in large layers with bleeding from the underlying layers (Fig. 2).

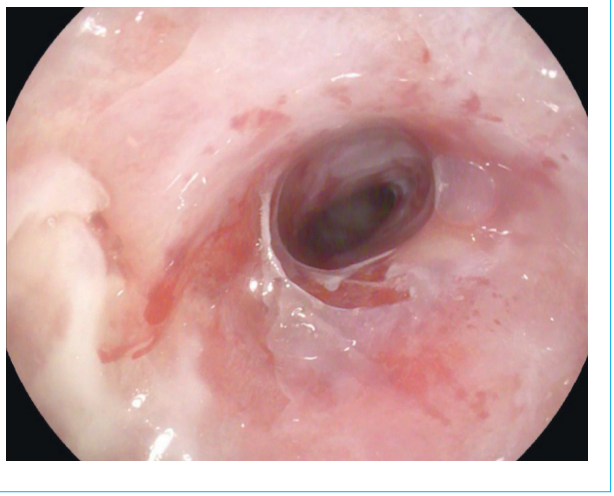

Figure 2. Matte, fragile mucous membrane of the esophagus with detachment of layers, bleeding of the underlying layers in lichen planus

Рисунок 2. Матовая хрупкая слизистая оболочка пищевода с отслоением пластов, кровоточивостью нижележащих слоев при красном плоском лишае

In the distal esophagus, trachealization-like multiple circular thin membranes were identified, slightly narrowing the lumen of the esophagus (Fig. 3).

From the medical history it was known that the patient did not smoke, denied alcohol abuse and the fact of a chemical burn of the esophagus. Previously worked as a salesperson. She lived in an industrial city. The parents had suffered from cardiovascular diseases and the relatives had had no malignant neoplasms of the gastrointestinal tract. She was being observed by a general practitioner for arterial hypertension. On an ongoing basis, she was taking perindopril 4 mg and atorvastatin 10 mg; denied taking other medications or dietary supplements. The patient did not note the presence of rashes on the skin and mucous membranes. No episodes of increased body temperature, lymphadenopathy or arthritis were detected.

Physical examination: the patient had a hypersthenic physique and overweight (body mass index — 25.8 kg/m^2). Thyroid gland — grade 0 according to WHO classification. The respiratory rate — 16 per minute. Breathing was vesicular, carried out in all parts of the chest, there were no adverse respiratory sounds. Pulse — 62 per minute. Systolic blood pressure — 130 mmHg, diastolic blood pressure — 60 mmHg. Heart sounds were rhythmic and clear. The mucous membrane of

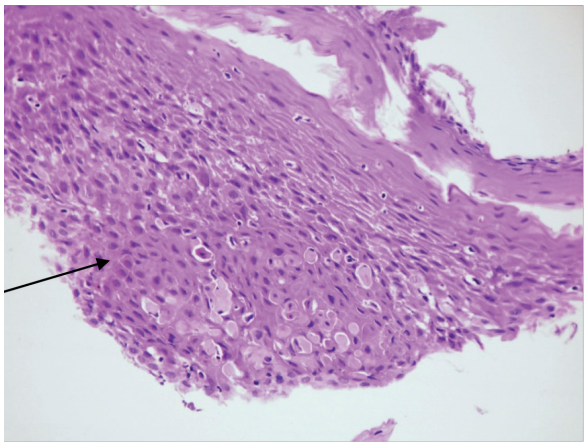

Figure 4. Matte, fragile mucous membrane of the esophagus with detachment of layers, bleeding of the underlying layers in lichen planus

Рисунок 4. Матовая хрупкая слизистая оболочка пищевода с отслоением пластов, кровоточивостью нижележащих слоев при красном плоском лишае

the oral cavity was clean, the tongue was moist and not coated. The abdomen was soft, painless on palpation. The edge of the liver did not protrude from under the edge of the costal arch, it was smooth and elastic. There was no swelling. Examination of the skin revealed no changes.

An additional examination was carried out: general clinical, biochemical laboratory tests and ultrasound examination of the abdominal organs were normal. Additionally, studies were carried out to exclude tuberculosis: chest X-ray and Diaskintest, which did not reveal any pathological changes. Serological markers of herpetic and cytomegalovirus infections were negative. Ileocolonoscopy did not demonstrate changes in the colon and ileum characteristic of inflammatory bowel diseases.

Repeat biopsy was performed in the upper, middle and lower esophagus after endoscopic bougienage, proton pump inhibitors and antacids therapy. Histological findings showed esophageal squamous non-keratinizing epithelium with a mature lining the surface in fragment of the mucosa. The angiomatosis, diffuse pronounced lymphocytic and neutrophil infiltration were noted in lamina propria. No band-like lymphocytic infiltration with degeneration of basal keratinocytes was detected. Layers of mature squamous non-keratinizing epithelium involving single oxyphilic apoptotic keratinocytes (Civatte bodies) were located separately (Fig. 4). Taking into account the specific endoscopic picture of the esophagus (mucosal fragility, mucosal denudation, fibrinous plaque, stenosis, trachealization) and the detection of apoptotic Civatte bodies, the diagnosis of esophageal lichen planus was made.

Taking into account the progressive clinical picture of esophageal stenosis, a decision was made on the need for further therapy with

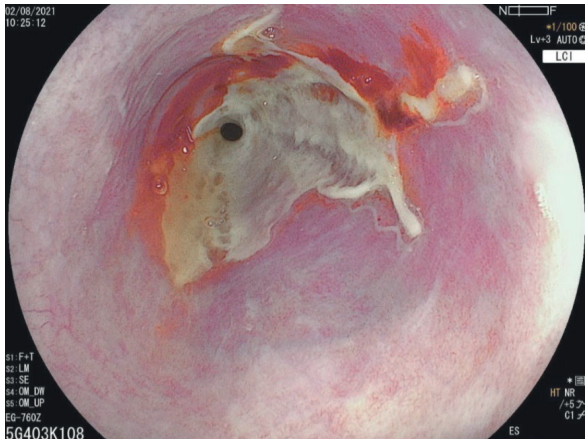

Figure 5. Endoscopic picture of lichen planus of the esophagus after a course of endoscopic bougienage against the background of antireflux therapy before the prescription of glucocorticosteroids (examination in the LCI color visualization mode)

Рисунок 5. Эндоскопическая картина красного плоского лишая пищевода после курса эндоскопического бужирования на фоне антирефлюксной терапии до назначения глюкокортикостероидов (осмотр в режиме улучшения цветовой визуализации LCI)

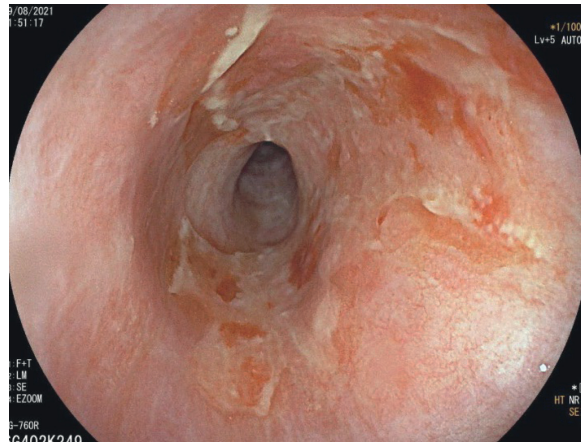

Figure 6. Endoscopic picture of lichen planus of the esophagus after the administration of glucocorticosteroids (prednisolone 40 mg) on day 14: expansion of the lumen of the esophagus, decrease in the thickness of fibrinous plaque

Рисунок 6. Эндоскопическая картина плоского лишая пищевода после назначения глюкокортикостероидов (преднизолон 40 мг) на 14-е сутки: расширение просвета пищевода, уменьшение толщины фибринозного налета

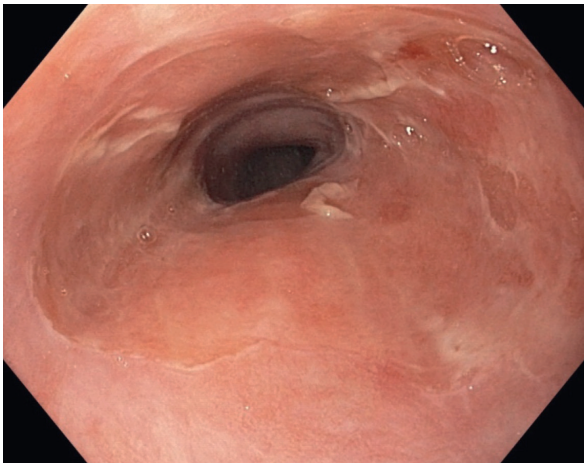

Figure 7. Endoscopic picture of lichen planus of the esophagus after a month of treatment with glucocorticosteroids (prednisolone 40 mg), positive dynamics

Рисунок 7. Эндоскопическая картина красного плоского лишая пищевода спустя месяц лечения глюкокортикостероидами (преднизолон 40 мг), положительная динамика

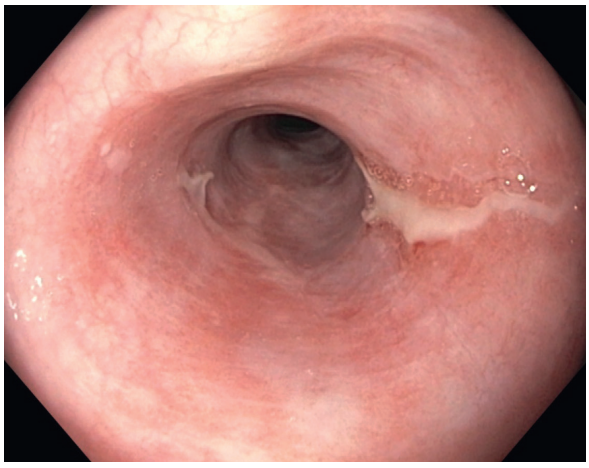

Figure 8. Endoscopic picture of lichen planus of the esophagus after reducing the dose of glucocorticosteroids (prednisolone 10 mg) after 3 months

Рисунок 8. Эндоскопическая картина плоского лишая пищевода после снижения дозы глюкокортикостероидов (преднизолон 10 мг) спустя 3 месяца

glucocorticosteroids. While taking 40 mg prednisolone during the first month, the symptoms of dysphagia were relieved and positive endoscopic image were noted (Figs. 5–7).

Subsequently, the dose of prednisolone was reduced to 10 mg, dysphagia resumed and esophageal changes recurred within several weeks when attempt was made to to reduce or completely cancel it (Fig. 8). Accordingly, a prolong prednisolone therapy with endoscopic management and control of side effects were recommended.

Discussion

The first description of esophageal lichen planus was made simultaneously by B.M. Al-Shihabi and L.G. Lefer in 1982 [14]. Since that time, the prevalence of esophageal lichen planus remain unspecified. A retrospective analysis of medical records of 584 patients with oral LP shown endoscopic signs of esophagitis in 4 patients only, endoscopy was performed in patients with dysphagia [15]. Subsequently, some authors demonstrated a significantly higher incidence of concomitant esophagitis in oral and cutaneous LP. R. Quispel et al. found esophageal changes in 50 % of patients with skin and oral rashes (12 out of 24 patients), later J.S. Kern et al. shown similar findings [16, 17]. Extraesophageal manifestations of cutaneous LP are not obligate, that cause of difficulty of assessing the true prevalence of esophageal lichen planus. L.P. Fox et al. investigated esophageal involvement as the soul or first manifestation of LP developed in 14 of 72 patients [18]. Oral LP was present in 89 % of patients, genital LP — in 42 %, cutaneous — in 38 %. F. Schauer et al. presented data on 52 patients with previously diagnosed cutaneous and mucosal LP, when 34 (65.4 %) patients had esophageal lichen planus. Findings shown no correlation between the frequency of involvement of the esophagus and the oral cavity, however, the authors noted combined severe esophagitis and genital involvement are associated with LP among patients [12]. The absence of cutaneous manifestations of lichen planus with esophageal changes in the presented clinical case demonstrates the difficulty of establishing the diagnosis, it required targeted biopsy. The possibility of asymptomatic esophageal LP also makes the diagnosis difficult. Typical clinical manifestations include dysphagia and odynophagia. Heartburn and regurgitation are not common, so many authors suggest previous unsuccessful therapy with proton pump inhibitors as an additional sign. Dysphagia and ineffective antireflux treatment can indicate to esophageal lichen planus in our clinical case. In general, the prevalence of symptomatic types varies from 33 to 70–81 % by various authors, but some researchers state a higher frequency [16–18]. Thus, according to the Mayo Clinic, all patients (25 people) had complaints [4], as well as per A. Rauschecker et al. (15 patients) [19]. One

recent study included 34 patients with esophageal lichen planus and 18 — without esophageal involvement of comparison group showed the importance of dysphagia as leading clinical symptom that correlates with the severity of esophageal lesion [12].

The endoscopic picture represented by specific mucosal changes and esophageal stenosis localized in the proximal esophagus, it is possible to differentiate lichen planus and reflux esophagitis. Endoscopy allows to identify characteristic signs in the esophagus, represented by edema, thickening, increased mucosal fragility with fibrinous surface, formed membranes and strictures. The findings are localized in the proximal esophagus in most cases (89 %) [18]. Altered esophageal mucosa is sensitive to any mechanical impact (passing an endoscope, biopsy forceps), may rupture and exfoliate with whitish plaques, severe bleeding and erosion of the underlying layer. The nonspecificity of endoscopic picture is another reason to make correct diagnosis difficult. ELP can be mistaken for various esophageal diseases: erosive reflux esophagitis, eosinophilic esophagitis, "slouthing" (exfoliative) esophagitis, epidermolysis bullosa, esophageal pemphigus, viral esophagitis [20, 21]. Concomitant *Candida* colonization of affected esophageal areas is possible.

The data on X-ray for esophageal lichen planus are extremely limited. Single studies demonstrate the presence of proximal strictures and diffuse narrowing of esophageal lumen as radiological signs of the disease, highlighting the difficulty to differ eosinophilic esophagitis [19].

Histopathology is a main method that verifies the diagnosis, which allow to reveal epithelial dyskeratosis with detachment, lichenoid lymphocytic infiltration and the most specific sign of intraepithelial apoptosis — Civatte bodies look like homogeneous eosinophilic globules in basal epithelium and superficial lamina propria [12]. Civatte bodies are specific feature of esophageal LP, but incidence is low — about 40 % of cases [22]. Our clinical observation demonstrates informativeness of histology increase with biopsy in all parts of the esophagus, while taking biopsy samples of fibrinous plaques should be observed. Lymphocytic mucosal infiltration in the esophagus is not pathognomonic of ELP and immunohistochemical studies to establish T cell association (CD3, CD4 and CD8) do not help differentiate esophageal LP from other diseases including drug-induced and infectious esophagitis [2]. M. Pittman et al. reported esophageal lymphocytic infiltration occurs both in Crohn's disease and in other autoimmune disorders [23].

Recommendations for the treatment of esophageal LP are limited by results of clinical case series. As a rule, most of the presented cases of esophageal LP were accompanied by dysphagia and stricture required endoscopic bougienage or balloon dilatation. However, endoscopic treatment have unstable effect and high rate of restenosis remains as with many other systemic esophageal disorders, thus it requires the search of specific therapy [24].

Most authors consider systemic or topical corticosteroids as the first line of therapy in symptomatic patients. Induction dose of prednisolone 40–60 mg (or methylprednisolone 32 mg) for 4–6 weeks leads to positive clinical and endoscopic dynamics in most patients [18, 25]. The effectiveness of topical corticosteroids was studied in retrospective review of patients cards treated from 1995 to 2016 at the Mayo Clinic: 32 patients were prescribed 3 mg budesonide dissolved in 10 mL sirupus 2 times or 0.3 mg/mL budesonide-rincinol gel and 8 patients were prescribed 880 mcg fluticasone 2 times a day. Evaluation of the response to therapy performed after an average of 3.2 months: the majority of patients noted reduction of dysphagia and improvement of endoscopic picture; 18 % patients required to switch to another therapy. However, even with an effective induction regimen in patients with esophageal LP, when GCS is discontinued, the relapse rate reaches 84 % [26].

Our clinical observation shows the need for futher research to allow regulating maintenance treatment. Currently, some authors recommend systemic corticosteroids in lower doses, switching to topical corticosteroids or combination corticosteroids with azatiprine 2 mg/kg body weight as maintenance therapy [17, 27]. It should be noted that data on the effectiveness of therapy required to clarife in future investigation. Management for patients who do not respond to corticosteroids are even less certain. Successful using of triamcinolone injections in the esophageal strictures have been reported in nine cases [28]. Typically, the retinoic acid analogue acitretin or various immunosuppressants (methotrexate, cyclosporine, rituximab, tacrolimus, thalidomide and mycophenolate mofetil) are prescribed in most cases of failure of the first-line therapy [29]. There is evidence of effective use of the Janus kinase inhibitors tofacitinib and upadacitinib in esophageal lichen planus [30]. A case of covered metal stent installation into esophageal lumen with relief of clinical symptoms and previous unsuccessful corticosteroids therapy was also described [24].

Although data on the natural history of esophageal LP are limited, there is increasing evidence that lichen planus may be associated with esophageal squamous cell carcinoma. K. Ravi et al. showed developed carcinoma during endoscopic observation that in 8 out of 132 patients (6.1 %). The analysis revealed no additional factors associated with esophageal cancer including age, prevalence of lichen planus and previous therapy. Given that cancer developed within the first year in half of the patients, repeat endoscopy after 6–12 months can be appropriate [31].

Conclusion

Esophageal lichen planus is one of rare causes of dysphagia. Additional criteria are female gender, older age, cutaneous and mucosal lichen planus allow including esophageal lichen planus in the differential diagnosis of dysphagia. Endoscopy and histology are crucial for the diagnosis.

Management of patients with esophageal lichen planus include glucocorticosteroids, endoscopic dilation and endoscopy surveillance, as there is high risk of developing squamous cell carcinoma.

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