Journal of
Gynecology and Women's Health
ISSN 2474-7602

Juniper Publishers
key to the Researchers

Mini Review
Volume 7 Issue 5 - December 2017
DOI: 10.19080/JGWH.2017.07.555721

J Gynecol Women's Health

Anogenital lichen Planus- Often Confused with Lichen Sclerosis

Sigrid Regauer*

Medical University Graz, Austria

Submission: November 11, 2016 ; **Published:** December 14, 2017

***Corresponding author:** Regauer S. Medical University Graz, Austria, Tel: +43 316 385 71728; Email: sigrid.regauer@medunigraz.at

Mini Review

Lichen planus is a systemic inflammatory mucocutaneous disease with a wide variety of clinical subtypes. The classic presentation of this papulo-squamous disease cornified skin is that of grouped, polygonal, flat-topped, violaceous papules and plaques with reticulated white lines, termed "Wickham's striae". Lichen planus in mucosal sites presents often as "erosive" disease with Wickham's striae surrounding the sharply outlined erythematous areas. Cutaneous lesions tend to be extremely pruritic, while lichen planus in mucosal sites is often painful. Rare manifestations include scarring alopecia, scarring nail dystrophies and bullous forms.

The anogenital region is commonly affected as well [1] with pruritic papules in the hair bearing cornified skin of mons pubis and labia minora, and the hairless cornified mucosa (also referred to as modified mucosa) of the interlabial sulci, labia minora and perineum, and with "erosive" mucosal lesions in the vestibular glycogenated mucosa. When larger areas are affected, confluent papules coalesce into plaques with an irregular and uneven surface, resembling a cobblestone pattern [2]. In contrast, lichen sclerosus is almost exclusively limited to the genital area, but never involves the vestibular mucosa or hair follicles. The classical clinical presentation is that of a solitary or several larger leukoplakic areas with smooth porcelain like surface [3].

Both diseases are characterized by an inflammatory infiltrate along the basement membrane, also referred to as lichenoid interface dermatitis. The inflammatory infiltrate is dominated by T-lymphocytes and in particular, activated CD4-positive T-lymphocytes. The cytokines secreted by activated CD4 T-lymphocytes along the basement membrane determine not only the activity and course of the disease, but alsothe histological features. Cytokines can be divided into type 1/pro-inflammatory (e.g. interleukin 1,2,18, INF, TNFα) and type 2/ anti-inflammatory (e.g. interleukin 4, 6, TNFβ, INFγ). While a pro-inflammatory cytokine pattern is typical for lesions of psoriasis, lichen sclerosus is characterized by an anti-inflammatory pattern.

In lichen planus, however, Type 1 and 2 CD4 T-lymphocytes with pro- and anti-inflammatory cytokine profile are simultaneously activated, and Therefore a simultaneous secretion of pro-inflammatory and anti-inflammatory cytokines occurs [4-6]; for review [4-6]. The balance of the secreted cytokines therefore determines both clinical and histological criteria of lichen planus. Typical for highly active lichen planus are lymphocyte infiltrates hugging and destroying the basement membrane, elongated pointed epithelial rete ridges with keratinocyte apoptoses, and circumscribed areas with accentuated so-called "wedge shaped" stratum granulosum (the histological correlate of Wickham striae). When CD4-T-lymphocytes with an anti-inflammatory profile become activated focal areas of fibrosis and basement membrane thickening will develop. Therefore it is not unusual to find various histological features in a biopsy of lichen planus, often with abrupt transitions from inflammation to basement membrane thickening or subepithelial sclerosis. It remains unclear to date, what triggers or stimulates the different T-lymphocytes subsets. Since lichen sclerosus shows a solely anti-inflammatory T-lymphocytic infiltration, the sclerosis is typically band-like and uniform. It is not unusual, however, that any basement membrane thickening or sub epithelial sclerosis in inflammatory dermatoses - no matter how focal is interpreted as lichen sclerosus.

Therefore, it is important to keep in mind, that the mere presence of sclerosis in a dermatosis (histologically as well as clinically) is no discriminating feature for lichen sclerosis. Careful search or serial sectioning will allow identification of (few) keratinocyte apoptoses and areas of accentuated stratum granulosum, even in advanced "burned out" cases of LP, but their demonstration needs attention to detail and screening at high magnification. As the histological microscopic features typically mirror the clinical appearance. An irregular, uneven, micro-papular or lichenified surface of the leukoplakic area in LP created by confluence of small papules and focal areas of sclerosis

is to be expected, while LS is characterized by smooth porcelain-like surfaces reflecting the uniform band-like sclerosis. Clinical evaluation of vulvar dermatoses and leukoplakic areas with a vulvoscope will assist in correct interpretation. In advanced cases of lichen planus with large areas of confluent papules with extensive lichenification, sclerosis with hyperkeratosis and hypergranulosis can be observed, although it is never as bland and uniform as that of lichen sclerosus.

Knowing the pathophysiology as well as the wide variation of clinical presentation of lichen planus, one might speculate that many published cases of cancers arising in lichen sclerosus actually represent advanced cases of unrecognized lichen planus. Along these lines many gynecologists/pathologists being unaware of the specific cytokine profile of lichen planus will interpret cases of lichen planus such as those presented in [7] either as lichen sclerosus or overlap/comorbid lichen sclerosus-lichen planus. Interdisciplinary evaluation of patients (dermatology, gynecology and specialized pathologists) will assist in correct identification of vulvar lichen planus and lichen sclerosus. Correct interpretation of the two dermatoses has therapeutic and clinical consequences.

Lichen sclerosus is better controlled by topical cortisteroid treatment, remissions are achieved faster than in lichen plan us and last longer. Mucosal involvement of lichen planus leads to great morbidity with scarring stenosis of introitus vaginae and vagina [8]. Cancer risk and location of cancer also differs for lichen sclerosus and lichen planus. About % of invasive vulvar cancers are HPV-negative [9]. As lichen sclerosus does not involve mucosal sites, only patients with lichen planus will develop dermatosis-associated cancers in the vestibular mucosa [10]. Particularly easily missed are cancers arising between clitoris/anterior commissure and the external orifice of the urethra. Most HPV-negative cancers are detected in the invasive stage in patients with untreated dermatoses, as they arise through the rapidly progressing precursor differentiated VIN [11,12]. Publications on prevalence as well as the risk of cancer development in vulvar/anogenital lichen planus are not available. The constantly cited cancer risk of up to 6% for lichen sclerosus may not be accurate if one assumes that an unknown percentage of mis diagnosed cases of lichen planus are included. The cancer risk in lichen planus depends on site of manifestations. In extragenital cutaneous locations lesions of lichen planus have no increased cancer risk, but in oral and esophageal mucosa a risk between 0.5 and 5% has been described [13,14]. Lichen planus associated vulvar cancers tend to arise in the hairless modified mucosa and the vestibulum rather than the hair bearing vulvar skin [7]. Inflammation probably plays a role in development of these HPV-negative cancers as it was shown in one publication that cancers were observed only in patients with untreated dermatoses [15]. HPV-negative cancers are the more aggressive cancers when compared to HPV- induced vulvar cancers and patients with vulvar dermatoses need close follow-up and treatment for prevention and/or early recognition of malignant transformation.

References

1. Di Fede O, Belfiore P, Cabibi D, De Cantis S, Maresi E, et al. (2006) Un expectedly high frequency of genital involvement in women with clinical and histological features of oral lichen planus. Acta Derm Venereol 86(5): 433-438.
2. Pittelkow MR, Daoud MS (2008) Lichen planus, in Fitzpatrick's dermatology in general medicine. In: Wolff K (Ed.), McGraw-Hill Companies, USA, pp. 244-258.
3. Powell JJ, Wojnarowska F (1999) Lichen sclerosus. Lancet 353(9166): 1777-1783.
4. Yamamoto T, Nakane T, Osaki T (2000) The mechanism of mononuclear cell infiltration in oral lichen planus: the role of cytokines released from keratinocytes. J Clin Immunol 20(4): 294-305.
5. Khan A, Farah CS, Savage NW, Walsh LJ, Harbrow DJ, et al. (2003) Th1 cytokines in oral lichen planus. J Oral Pathol Med 32(2): 77-83.
6. Rhodus NL, Cheng B, Ondrey F (2007) Th1/Th2 cytokine ratio in tissue transudates from patients with oral lichen planus. Mediators Inflamm 2007: 19854.
7. Regauer S, Reich O, Eberz B (2014) Vulvar cancers in women with vulvar lichen planus: A clinic pathological study. J Am Acad Dermatol 71(4): 698-707.
8. Cooper SM, Wojnarowska F (2006) Influence of treatment of erosive lichen planus of the vulva on its prognosis. Arch Dermatol 142(3): 289-294.
9. De Sanjose S, Alemany L, Ordi J, Tous S, Alejo M, et al. (2013) Worldwide human papillomavirus genotype attribution in over 2000 cases of intraepithelial and invasive lesions of the vulva. Eur J Cancer 49(16): 3450-3461.
10. Chiu TL, Jones RW (2011) Multifocal multicentric squamous cell carcinomas arising in vulvovaginal lichen planus. J Low Genit Tract Dis 15(3): 246-247.
11. Derrick EK, Ridley CM, Kobza-Black A, McKee PH, Neill SM (2000) A clinical study of 23 cases of female anogenital carcinoma. Br J Dermatol 143(6): 1217-1223.
12. Crum C (2014) Tumours of the Vulva. Epithelial tumours. In: Kurman RJ (Ed.), World Health Organisation, Classification of Tumours of the Female Genital Tract. Lyon, pp. 232-236.
13. Gandolfo S, Richiardi L, Carrozzo M, Broccoletti R, Carbone M, et al. (2004) Risk of oral squamous cell carcinoma in 402 patients with oral lichen planus: a follow-up study in an Italian population. Oral Oncol 40(1): 77-83.
14. Chryssostalis A, Gaudric M, Terris B, Coriat R, Prat F, et al. (2008) Esophageal lichen planus: a series of eight cases including a patient with esophageal verrucous carcinoma. A case series. Endoscopy 40(9): 764-768.
15. Cooper SM, Madnani N, Margesson L (2015) Reduced Risk of Squamous Cell Carcinoma With Adequate Treatment of Vulvar Lichen Sclerosus. JAMA Dermatol 151(10): 1059-1060.

How to cite this article: Regauer S. Anogenital lichen Planus- Often Confused with Lichen Sclerosis. J Gynecol Women's Health 2017; 7(5): 555721. DOI: 10.19080/JGWH.2017.07.555721.

DOI: 10.19080/JGWH.2017.07.555721

How to cite this article: Regauer S. Anogenital lichen Planus- Often Confused with Lichen Sclerosis. J Gynecol Women's Health 2017; 7(4): 555721. DOI: 10.19080/JGWH.2017.07.555721.