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Co-existence of lichen planus and nephrotic syndrome; a rare case report

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ABSTRACT

Glomerulopathies can occur primarily or as a secondary event to a systemic disease. Although many disorders can induce secondary glomerulopathy, the association between this disease and lichen planus is unclear. In this paper, we present a 20-year-old man, who was a known case of lichen planus, presented with nephrotic syndrome as a feature of glomerulopathy.

Keywords: Lichen planus, Nephrotic syndrome, Glomerulopathy

Case Report

Implication for health policy/practice/research/medical education:
All physicians involving the care of lichen planus patients must be aware of the probability of co-existence of lichen planus with glomerulopathies and especially the nephrotic syndrome, so prescribing a urinalysis in all lichen planus patients seems to be necessary.

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Introduction

Lichen planus is a disease of unknown etiology which affects the skin, but the mucous membranes, hair follicles, and the nails may also be involved (1). On the other hand, nephrotic syndrome is defined as proteinuria that is more than 3.5 grams in a 24 hour-period for 1.73 m^2 of body surface area, hypoalbuminemia, edema, dyslipidemia, and lipiduria. This syndrome is a feature of glomerulopathy, which can be primary (idiopathic), or secondary to other disorders or even secondary to drugs (2). The kidney biopsy is the gold standard method for determining the etiology of glomerulopathies (3).

Although several disorders are clearly defined as the causes of secondary glomerulopathy (2,4), the association between lichen planus with glomerulopathies has not been proved, and there are only very few case reports about the co-existence of these two entities in the literature. Herein, we report a 20-year-old man with eruptive form of lichen planus with oral involvement, who presented with the nephrotic syndrome.

Case Presentation

A 20-year-old man was referred to nephrology clinic from the department of dermatology of our hospital to evaluate his new onset proteinuria. He was an immigrant from Afghanistan who worked as a shoemaker. He was a known case of eruptive lichen planus with oral involvement for 5 months, which was confirmed by skin biopsy (Figure 1).

Other than lichen planus, he did not have any diseases in his past medical history. He did not smoke, consume alcohol, or use illicit drugs. In his physical examination, vital signs were in their normal ranges, there was a 2 plus pitting edema in his lower limbs, and healing skin hyperpigmentations of his previous skin lesions were visible on his trunk and upper and lower extremities. The head and neck examination and the auscultation of his cardiac and lung sounds seemed to be normal. In his lab data, there was a 2 plus proteinuria, hematuria, and lipiduria in his urinalysis, and dyslipidemia in his blood tests (Table 1).

His kidneys' sizes and shapes were in their normal

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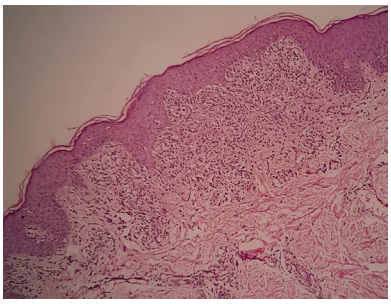

Figure 1. Orthokeratotic epidermis with saw tooth acanthosis, lichenoid basal damage and band-like lymphocytic infiltrate in superficial dermis, which suggests lichen planus (H&E stain ×100).

ranges in ultrasonography, but his left kidney was in the pelvic area. For determining the amount of proteinuria, we requested a 24-hour urine collection that later revealed a nephrotic- range proteinuria (Table 2).

For evaluating the secondary etiologies of glomerulopathies, his blood viral and serologic markers were evaluated, as seen in Table 2. Then, a percutaneous kidney biopsy was scheduled for him. An expert renal pathologist studied the renal samples with the light and

Table 1. The patient's primary lab data, taken at the first month of his presentation, when the diagnosis of eruptive lichen planus was made for him

Parameter	Result
WBC (#/ mm^3)	11300
Hb (g/dL)	15.8
Hct (%)	44.9
Plt (#/ mm^3)	205000
ESR (mm/h)	5
FBS (mg/dL)	94
BUN (mg/dL)	25
Cr (mg/dL)	0.7
Total cholesterol (mg/dL)	221
LDL cholesterol (mg/dL)	104
HDL cholesterol (mg/dL)	56
TG (mg/dL)	349
AST (IU/L)	21
ALT (IU/L)	19
ALP (IU/L)	156
HBs Ag (ELISA)	Negative
Anti-Hbc Ab (ELISA)	Negative
Anti-HCV Ab	Negative

WBC: White blood cell; Hb: Hemoglobin; Hct: Hematocrit; Plt: Platelet; ESR: Erythrocyte sedimentation rate; FBS: Fasting blood glucose; BUN: Blood urea nitrogen; Cr: Creatinine; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; TG: Triglyceride; AST: Aspartate aminotransferase; ALT: Alanine transaminase; ALP: Alkaline phosphatase; HBs Ag: Hepatitis B surface antigen; Anti-Hbc Ab: Hepatitis B core antibodies; Anti-HCV Ab: Anti-hepatitis C virus Ab.

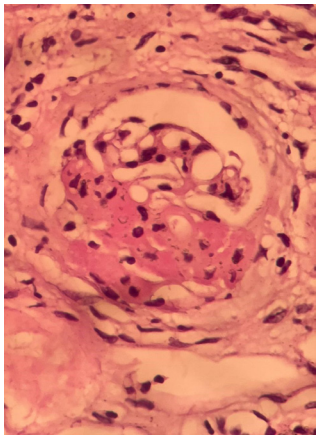

Figure 2. Cross-section of the needle kidney biopsy, shows perihilar variant of FSGS in our patient. Hyaline deposition and sclerosis are seen at the vascular pole of the glomerulus (H&E stain ×400).

immunofluorescence microscopy. As seen in Figure 2, the kidney biopsy pathology result was hyaline deposition and sclerosis seen at the vascular pole of the glomerulus by light microscopy. In immunofluorescence microscopic examination, there were not any deposits in IgA and IgG staining, with trace depositions in C3, across with 1$^+$ in IgM. These findings suggested the diagnosis of perihilar variant of focal and segmental glomerulosclerosis (FSGS). Then, proper treatment for his FSGS was commenced.

Discussion

Glomerulopathies present themselves with hematuria, hypertension, proteinuria, or renal failure. Nephrotic-range proteinuria, with or without the other features of the

Table 2. The patient's complementary lab data, taken about 5 months after the confirmation of his lichen planus, for evaluating the degree of his proteinuria and ruling out the secondary etiologies of glomerulopathy

Parameter	Result
24-hour urine volume (ml)	2400
24-hour creatinine amount (mg)	1000
24-hour protein amount (mg)	5935
HIV 1&2 Ab	Negative
c-ANCA* (U/mL)	0.6
p-ANCA* (U/mL)	1.0
Anti-HCV Ab.	Negative
ANA** (U/mL)	0.34
Albumin (g/dL)	2.0

HIV: Human immunodeficiency virus; c-ANCA: cytoplasmic Anti-neutrophil cytoplasmic antibody; p-ANCA: perinuclear Anti-neutrophil cytoplasmic antibodies; HCV Ab: Hepatitis C virus antibody; ANA: Anti-nuclear antibody.
* Normal range < 12.
** Normal Range <0.9.

nephrotic syndrome, is a hallmark of Glomerulopathies (4). This disease is divided into two categories; primary, which has no distinct disease as its etiology, and secondary, which occurs due to some autoimmune diseases, malignant neoplasms, infectious causes, or consuming drugs. For instance, membranous nephropathy can occur secondary to colon, breast, and lung cancer, rheumatologic disorders, like systemic lupus erythematosus, or drugs such as penicillamine, and FSGS can be a consequence of viral infections such as human immune deficiency virus infection, or conditions such as reflux nephropathy (2,4).

Although the relationship between many disorders with glomerulopathies has been clarified, the association of lichen planus with kidney diseases, especially glomerulopathies, has not been evaluated thoroughly to date.

Deng et al evaluated the prevalence of chronic kidney disease and its risk factors in patients suffering from oral lichen planus in China. They found a greater frequency of decreased glomerular filtration rate and albuminuria in their patients (5). In a cross-sectional study conducted by Lopez-Jornet et al, they found no association between autoimmune diseases and lichen planus (6).

Some investigators suggested that hepatitis C virus (HCV) infection could be associated with lichen planus (7). This association is controversial, and an etiological relationship is uncertain. Some studies found a statistically meaningful association between these two entities (8-10); since a prevalence of up to 62 percent has been reported (10). On the other hand, HCV infection is a proven etiologic factor for some kinds of glomerulopathies (11). It can theoretically suggest the potential relation of glomerulopathies and lichen planus. However, in our case, HCV Ab tests were negative, as seen in Table 1.

In 2017, Sargin and Gurer reported a 57-year-old man with coexistence of FSGS, late onset systemic lupus erythematosus, and lichen planus (12). There are some case reports of simultaneous occurrence of some variants of lichen planus and systemic lupus erythematosus (13,14). We cannot now rule out the possibility that our patient was an antinuclear antibody (ANA) -negative systemic lupus erythematosus whose ANA might become positive in the future, but repeated ANA tests for him have been negative until now.

Krishnamurthy et al reported the simultaneous occurrence of lichen planus and the nephrotic syndrome in an 8-year-old boy whose kidney biopsy revealed the diagnosis of minimal change disease (15), while Mancuso et al reported this association in a 30-year-old Italian woman (16). The appearance of both nephrotic syndrome and cutaneous manifestations of lichen planus in Mancuso's patient was simultaneous, while in our patient, skin manifestations of lichen planus preceded nephrotic syndrome a few months. To the best of our knowledge, our patient is the first adult patient with lichen planus who developed FSGS not related to systemic lupus erythematosus, and also it seems that this case is among the first lichen planus patients who developed nephrotic syndrome. Overall, we concluded that lichen planus might have an etiological role in nephrotic syndrome development, although enough data is lacking, and further evaluation is warranted.

Conclusion

This case shows the importance of noticing to urinalysis in patients suffering from lichen planus. In this case report, an FSGS is detected by performing renal bipsy in our 20-year-old lichen planus patient who had proteinuria in his urinalysis. The early detection of proteinuria in lichen planus patients and prompt referring to nephrologist is the cornerstone of management of a GP in these patients. The causal relationship between lichen planus and the nephrotic syndrome remains to be elucidated.

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Authors' contribution

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Writing–review & editing: Zeinab Bahrami Arz Aghdas.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

This case report was conducted in accordance with the World Medical Association Declaration of Helsinki. Patient has given us a written informed consent for publication as a case report. Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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