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Measurement of some immunological Indicators in patients with recurrent oral lichen planus and some viral infections

Arjwan Mohammed Shuker[1]*, Mahmood abd Aljabbar ALTobje[2]

[1]Department of Dental Basic Sciences, College of Dentistry, University of Mosul, Mosul, IRAQ.
[2]Department of Biology, College of Science, University of Mosul, Mosul, IRAQ.

*Corresponding Author: Arjwan Mohammed Shuker

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ABSTRACT: Oral lichen planus(OLP) usually appears in the form of skin lesions with multiple oral symptoms. Research indicates that it may be an autoimmune disease and is believed to be related to some viral infections. aims to determine the level of some immune indicators in patients with OLP and some types of viral infections associated with it. The research also aimed to determine the level of these indicators in patients who suffered from recurrence of the infection. Methods: ELISA technology was used to determine the level of immune indicators, Results the percentage of patients infected with OLP was 48.9%, while those infected with OLP+HCV, OLP+HBsV, and OLP+HSV-1 were 9.4%, 6.2%, and 5.2%, respectively. Some immune indicators are in higher concentrations in females than in males. While there was variation in the levels of some other measured indicators, as they appeared in higher concentrations in patients infected with OLP+HBsV than in other groups, followed by the group of patients infected with OLP+HCV. In the group of patients with OLP, the infection was recurrent at a rate of 93.6%, while the non-recurrence rate was 6.4%. The immune indicators S-IgA, IgG, and C3 each appeared in higher concentrations in non-recurring disease cases, while the rest of the immune indicators appeared in higher concentrations in recurrent cases, and they varied. The levels of these immune indicators in other disease groups under study among those who have recurrence than in the other group. Conclusion: Some immunological indicators are increase in OLP patients and also, in HBsV infections and the disease may be recurrent in same family.

Keywords: Oral lichen palus OLP, Hepatitis C, B, Herpes Simplex-1 virus, complement C3

1. INTRODUCTION

Oral lichen planus (OLP) is a chronic inflammatory condition of the oral mucosa, and is one of the most prevalent oral abnormalities seen in clinical settings. Although multifactorial in origin, classical OLP is not strictly an autoimmune disease because it is an immunologically mediated condition. Lesions that are white, reticular, erosive, or ulcerative may develop. Recent research suggests that an abnormal immune response to self-antigens is a crucial pathogenic element for OLP, even though the precise reason is yet unknown. Its pathophysiology may involve hereditary factors, medications, dental fillings, stress, immunity, and hypersensitivity reactions [1]. When viewed clinically, the lesions typically manifest as many bilateral lesions spread across the oral cavity. Clinical diagnosis can be given for the following six forms of OLP: reticular, papular, plaque-like, atrophic/erosive, ulcerative, and bullous. [2]. According to an epidemiological study, the prevalence of OLP is estimated to be between 0.1% and 4% of the general population. The age group most affected by OLP is middle-aged and older, with 50 and 59 years of age. OLP affects women twice as often as men, while some have discovered a ratio of 3:1 or 4:1. [3]. In other study, suggest that autoimmune illnesses lead to LP. Certain theories suggest that changes to the outer layer of keratinocytes cause the substantial inflammatory infiltration observed in OLP. Cell lines derived from LP lesions have revealed T-cells cytotoxic against keratinocytes [4].

Several reports indicate the possibility of a link between OLP and viral infections. Numerous subtypes of the human herpesvirus family, such as Epstein-Barr, Cytomeg-alovirus, Herpesvirus, and Herpes simplex, have been examined and researched. Wether these compounds are linked to OLP or if infection aggravates pre-existing lesions is

*Corresponding author: arjwanarjwan@uomosul.edu.iq

unclear [5]. Research on the genesis of OLP has focused mostly on three viruses: the hepatitis C virus (HCV),An analysis found that, in comparison to the general population, those infected with the hepatitis C virus (HCV) had a twice higher risk of developing OLP. However, it appears that these outcomes are related to particular regions [6],[7]. The hepatitis C virus (HCV) is a hepatotropic non-cytopathic virus that effectively eludes the immune system survive in infected hosts. The host's immune system mounts an innate as well as an adaptive defense against a viral infection [8]. Numerous studies have connected OLP infection to hepatitis B and C to compare OLP patients with healthy controls to ascertain the prevalence of hepatitis C virus (HCV) antibodies and hepatitis B virus (HBsV) antigen [7]. Likewise, reported that 10% of the persons with lichen planus had hepatitis surface antigen, 15% had hepatitis B antibodies (vaccinated), and 9% had hepatitis C antibodies [9]. The possibility of recurrence of the disease is something that naturally concerns the patient to a great extent. This is especially true when the disease is of serious prognostic importance or may cause the patient significant discomfort while walking. Lichen planus, due to its recurrent chronic course. OLP is a chronic disease, are decreasing the frequency of relapses is the most important requirement for patients and clinicians. However, to date, no treatment for OLP is completely curative, and most available treatment modalities have focused on eliminating the signs and symptoms of the disease [10].

2. MATERIALS AND METHODS

2.1 Selection of group

This study was conducted on 82 individuals who were receiving care at Nineveh City hospitals.. (“Oral medicine and dermatology consultants”). between November 11, 2023, and February 2, 2023. Fifteen healthy adults of both sexes, ranging in age from 30 to 69, made up the control group. The study group included 67 people, aged between 15-75 years old, (32) males and (35) females attended to Oral Medicine Unit in College of Dentistry/Mosul University and Dermatologist units in Nineveh hospitals. The patients' medical examinations based on the clinical criteria and histopathological features for OLP issued by the World Health Organisation (WHO) and depending on the specialist physician[11]:

. Histopathologic criteria

- Band-like or patchy, predominately lymphocytic infiltrate in the lamina propria confined to the epithelium–lamina propria interface
- Basal cell liquefactive (hydropic) degeneration
- Lymphocytic exocytosis
- Absence of epithelial dysplasia **[11].**

My research is An observational study design known as a case-control study compares two groups: cases, or people who have a specific disease or result, and controls, or people without the disease or outcome.

2.2 Collection of Samples

Serum and saliva were collected at the same visit from each patient. Each patient provided a 5 ml blood sample, which was collected in a sterile plastic tube. Whole unstimulated saliva samples were collected at least two hours after any food intake [12].

2.3. Immunological Assays

1- Human secretory immunoglobulin A (S-IgA and IgG) ELISA.

ELISA Assays were used in this study to detect S-IgA and IgG in the Silva of the study and control groups (SunLong Co., China).

2- Human Interleukin 17(IL-17), C3), TNF-α, HSV1, HCV-IgG and HBsV-IgG kit.

“ELISA” assays were also used to detect them (ELISA kit uses Sandwich-ELISA as the method- SunLong Co.).

Data were statistically analyzed using "SPSS" (Ver. 25). When a P-value was ≤ 0.05, differences between observations were considered significant. & the highly significant at P-value ≤ 0.01. The following statistical methods were used for the analysis of data. And according to immunological indicators the data was parametric and non-parametric.

1. Standard statistical methods were used to determine the number, percentage, the mean, standard deviation (SD).

2. Paired student t-test was used among the factors for comparisons.
3. Pearson correlation coefficient was used to find the relationship among the measured variables.

3. RESULTS

The samples in this study consisted of (82) subjects, the study Group was (67) patients with oral lichen planus (OLP), the study group consisted of four groups and their percentage rates and numbers, as the first group OLP appeared by 48.9% (47) while the second group of patients with OLP appeared with viral hepatitis C 9.4% (9), as the third group appeared For people with OLP with viral hepatitis B by 6.2%(6), as well as the fourth group of people with OLP with herpes simplex type 1 by 5.2%(5). And 5.7% (15) was control negative as the healthy human, (Figure -1).

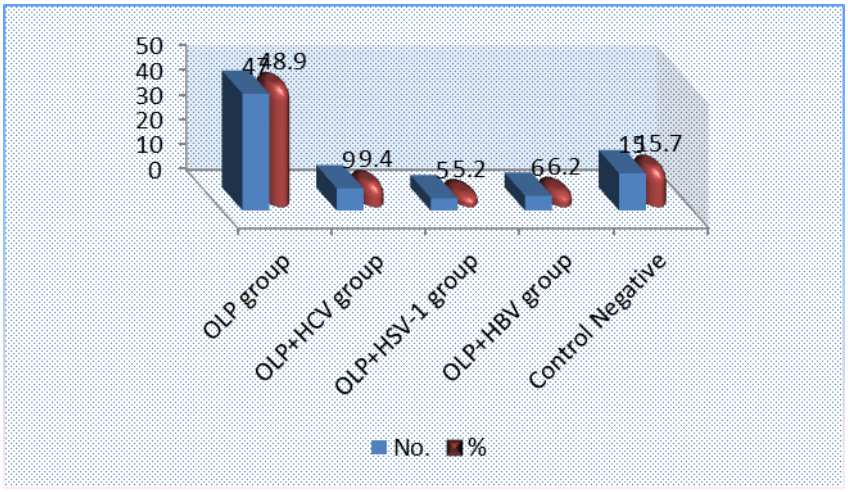

FIGURE 1. - The numbers of patients and the percentage of the four groups of the disease with control negative

In the present study, (Table-1) showed that males were (32) (47.8%), while females were (35) (52.0%) in the four study groups .Also the Immune indicators showed variation in their concentrations, as S-IgA was at a higher rate in males than in females in the OLP group, while it appeared at a higher rate in females than in males in the OLP + HCV as well as OLP + HBsV groups, and we did not obtain samples from females. There were only males infected with OLP+HSV-1, compared to the control negative group, where the S-IgA rate was higher in males than in females. On the other hand, IgG appeared in the OLP and OLP + HBsV groups at a higher rate in females than in males in addition to the control group, while the rate was close between males and females in the OLP + HCV group. The rate of IL-17 concentration was higher in males than in females in patients infected with OLP and OLP+HBsV, while its rate in patients infected with OLP+HCV was higher in females than in males compared with the control negative group. The concentration of complement C3 was higher in females than in males in the OLP and OLP + HCV groups, while it was higher in males than in females in the OLP+HBsV compared to the control group, as the concentration in males was higher. It was found that the rate of TNF-α concentration was higher in males than in females in the OLP group, while the rate was close in the OLP + HCV group, and it was higher in females than in males in the OLP+HBsV group compared to the control group, as the concentration in males was higher(Table-1).

Table 1. - Means of immune indicators for groups of patients by sex

Cases	Sex		S-IgA pg/ml	IgG ng/ml	IL-17 pg/ml	C3 ng/ml	T.N.F pg/ml
OLP Group	female	No. %			**27 (57.4%)**		
		Mean	1062.34	6.000	78.741	8.411	18.602
	male	No. %			**20 (42.6%)**		
		Mean	1338.88	5.580	92.748	7.234	19.560
OLP+HCV	female	No. %			**5 (55.6%)**		
		Mean	1554.09	6.109	101.725	7.780	21.956

	male	No. %			**4 (44.4%)**		
		Mean	1224.918	6.352	71.916	5.016	21.689
OLP+HSV-1	female	No. %			**0 (0.0%)**		
		Mean	0	0	0	0	0
	male	No. %			**5 (100%)**		
		Mean	1338.34	5.302	62.636	8.147	17.963
OLP+HBsV	female	No. %			**3 (50.0%)**		
		Mean	1264.19	7.917	75.02	10.29	22.471
	male	No. %			**3 (50.0%)**		
		Mean	1069.5	5.885	93.36	11.81	19.42
Control Negative	female	No. %			**7 (46.7%)**		
		Mean	601.041	6.931	57.132	16.085	11.789
	male	No. %			**8 (53.3%)**		
		Mean	804.994	5.436	39.58	17.393	12.613

The (Table-2), also in the present study shows the abnormal percentages of immune indicators for patients in the OLP group, as there is a percentage of 38.2% for S-IgA, 12.7% for IgG, 44.6% for IL-17, and 46.8% for TNF-α levels were abnormally high while complement C3 levels were abnormally low at 55.3% . While the immune indicators in patients of the OLP + HCV group appeared with abnormally high rates in S-IgA, IgG, IL-17, and TNF-α, by 55.5%, 11.1%, 11.1%, 22.2%, 55.5%. 77.7%, respectively, but the percentage of complement C3 was as low as 77.7%. As for the OLP + HSV-1 group, the percentages of the patients' immune indicators were high in S-IgA, IgG, IL-17and TNF-α, by 60.0%, 20.0%, 40.0%, 20.0%, and 40.0. %, 60.0% respectively but the percentage of complement C3 was abnormal as low as 60.0%. The abnormal rates of immune indicators for patients in the OLP + HBsV group were 33.3% for S-IgA, 33.3% for IgG, 83.3% for IL-17, and 66.6% for TNF-α. The percentages are abnormally high, while the percentage of complement C3 is abnormally low at 16.6%.

Table 2. - Rates of abnormal immune indicators for patients in the four groups and their percentages

Test		N.	Normal Range	Abnormal No.	Abnormal %	Mean	Std. Deviation
OLP: Oral Lichen Planus Group	S-IgA pg/ml	47	234-1170	18	38.2	1195.02	586.51
	IgG ng/ml	47	3.9-7.0	6	12.7	7.98	1.33
	IL-17 pg/ml	47	9.3 -73.5	21	44.6	84.70	98.48
	C3 ng/ml	47	8.2- 32.7	26	55.3	4.91	2.40
	T.N.F pg/ml	47	4.6-18.8	22	46.8	19.01	5.34
OLP+HCV	S-IgA pg/ml	9	234-1170	5	55.5	1407.79	741.15
	IgG ng/ml	9	3.9-7.9	1	11.1	9.10	--
	IL-17 pg/ml	9	9.3 -73.5	1	11.1	87.69	--
	C3 ng/ml	9	8.2- 32.7	7	77.7	4.83	2.78
	T.N.F pg/ml	9	4.6-18.8	7	77.7	21.78	4.79
OLP+HSV-1	S-IgA pg/ml	5	234-1170	3	60.0	1338.34	942.38
	IgG ng/ml	5	3.9-7.9	1	20.0	8.24	--
	IL-17 pg/ml	5	9.3 -73.5	2	40.0	74.03	32.97
	C3 ng/ml	5	8.2- 32.7	3	60.0	4.14	1.37
	T.N.F pg/ml	5	4.6-18.8	3	60.0	29.96	2.55
OLP+HBsV	S-IgA pg/ml	6	234-117	2	33.3	1458.35	347.53
	IgG ng/ml	6	3.9-7.0	2	33.3	8.34	0.51
	IL-17 pg/ml	6	9.3 -73.5	5	83.3	119.65	118.58

C3 ng/ml	6	8.2- 32.7	1	16.6	5.31	--
T.N.F pg/ml	6	4.6-18.8	4	66.6	24.0300	3.035

We have shown in Table (3) the cases of recurrence of OLP disease at a rate of 6.4%, and non-recurrence of OLP at a rate of 93.6% as the concentration rates of immune indicators varied between the two cases, as the rate of concentration of S-IgA in patients without recurrence of the disease was higher (1361.1 pg/ ml), while its concentration in recurrence is (1167.6pg/ml), as well as the average concentration of IgG and C3, where their concentration is higher in non-recurrence disease by 6.2ng/ml and 12.1ng/ml, respectively, while their concentration in recurrence is 5.7ng/ml. and 7.6ng/ml, respectively. While the following immune indicators, IL-17and TNF-α, their concentrations in disease recurrence were higher, as they were 85.9pg.ml, and 19.1pg/ml, respectively, and without statistically significant differences for the immune indicators. For patients when comparing recurrent groups with non-recurrent groups under study.

Table 3. - Comparison between recurrence and non-recurrence in patients with OLP with the average concentration of immune indicators

FACTORS	Recurrence of OLP				t-value	P -value
	Recurrence No.3(6.4%).		Non recurrence No.44(93.6%).			
	Mean	S.D.	Mean	S.D.		
S-IgA pg/ml	1167.66	586.73	1361.2	676.53	0.54	0.58
IgG ng/ml	5.79	1.365	6.28	0.83	0.61	0.54
IL-17 pg/ml	85.93	101.48	66.65	34.01	0.32	0.74
C3 ng/ml	7.62	5.33	12.10	5.57	1.04	0.16
T.N.F pg/ml	19.17	5.42	16.60	3.67	0.80	0.42

S.D.=Standard Deviation

In this study, we have shown also cases of recurrence of OLP + HCV disease in patients from one family at a rate of 11.2%, and the non-recurrence of OLP + HCV disease at a rate of 88.8% as the rates of concentrations of immune indicators varied between the two cases, as the rates of concentrations of S-IgA, IL-17, , and C3 were 1416.1pg/ml, 77.6 ng/ml, and 6.6ng/ml, respectively, were higher in recurrence of the disease, while the concentrations in non-recurring disease were 1340.9pg/ml and 44.9pg. /ml, and 6.0ng/ml, respectively, while the concentrations of IgG and TNF-α were 7.0ng/ml and 26.1pg/ml, higher in disease recurrence patients than in non-recurrence patients. There were no statistically significant differences in the immune indicators. For patients when comparing recurrent groups with non-recurrent groups under study(Table-4).

Table 4. - Comparison between recurrence and non-recurrence in patients infected with OLP+HCV with the average concentration of immune indicators

FACTORS	Recurrence of OLP+HCV				t-value	P - value
	Recurrence No.1(11.2%).		Non recurrence No.8(88.8%).			
	Mean	S.D.	Mean	S.D.		
S-IgA pg/ml	1416.15	-	1340.9	791.87	0.09	0.93
IgG ng/ml	6.11	-	7.04	0.73	1.20	0.26

IL-17 pg/ml	77.66	-	44.97	150.46	0.39	0.70
C3 ng/ml	6.61	-	6.05	5.19	0.10	0.92
T.N.F pg/ml	21.29	-	26.15	4.83	0.94	0.37

While recurrence cases for OLP + HSV-1 patients appeared in (Table-5) at a rate of 20%, and the non-recurrence of the disease OLP + HCV at a rate of 80% as the rates of concentrations of immune indicators varied between the two cases, as the rates of concentrations of S-IgA, IgG, IL-17, C3, and TNF-α were 1500.2 pg/ml, 5.6ng/ml, 49.6pg/ml, 8.9ng/ml, and 18.3pg/ml, respectively, higher in recurrence of the disease in one family, while their concentrations were higher in non-recurrence cases. disease recurrence levels were 690.7 pg/ml, 3.7 ng/ml, 34.5 pg/ml, 4.7 ng/ml, and 16.3 pg/ml, respectively, It is higher than in recurrence, and without statistically significant differences in the immune indicators of patients when comparing the recurrent groups with the non-recurrent groups under study.

Table 5. - Comparison between recurrence and non-recurrence in patients infected with OLP+HSV-1 with the average concentration of immune indicators

FACTORS	Recurrence of OLP+HSV-1				t-value	P -value
	Recurrence No.1(20%).		Non recurrence No.4(80%).			
	Mean	S.D.	Mean	S.D.		
S-IgA pg/ml	1500.25	-	690.72	1004.66	0.72	0.52
IgG ng/ml	5.68	-	3.76	1.194	1.44	0.24
IL-17 pg/ml	49.66	-	34.53	33.47	0.93	0.41
C3 ng/ml	8.98	-	4.78	7.03	0.53	0.63
T.N.F pg/ml	18.35		16.39	2.76	0.63	0.57

As well as recurrence cases for OLP+HBsV patients, they appear in (Table-6) at a rate of 33.4%. and non-recurrence of OLP+HBsV at a rate of 66.6%, The rates of concentrations of immune indicators varied between the two cases, as the rates of concentrations of S-IgA, IgG, ANA, and TNF-α were 1418.3 pg. /ml, 6.6 ng/ml, and 26.2ng/ml, respectively, higher in non-recurrence of the disease in one family, while the concentrations in recurrent disease were 1041.1pg/ml, 6.2 ng/ml, and 29 3 pg/ml and 11.8 ng/ml, respectively, while the concentrations of IL-17and C3 were 182.1 pg/mland 11.8 ng/ml, higher in recurrence patients than in non-recurrence patients. And without statistically significant differences in the immune indicators of patients when comparing the repeated groups with the non-repeated groups under study.

Table 6.- Comparison between recurrence and non-recurrence in patients infected with OLP+HBsV with the average concentration of immune indicators

FACTORS	Recurrence of OLP+HBsV				t-value	P -value
	Recurrence No.2(33.4%).		Non recurrence No.4(66.6%).			
	Mean	S.D.	Mean	S.D.		
S-IgA pg/ml	1041.14	116.54	1418.3	404.16	1.92	0.126
IgG ng/ml	6.26	0.78	6.6835	2.02	0.40	0.709
IL-17 pg/ml	182.16	152.55	128.67	70.80	0.45	0.675
C3 ng/ml	11.80	5.27	9.6385	1.00	0.54	0.616
T.N.F pg/ml	18.27	4.12	26.208	2.94	2.36	0.077

4. DISCUSSION

The work reported by [13]who indicated that roughly 0.5 - 4% of the general population has OLP. The condition occurs at all ages, peaking at the age of 40 It is more prevalent in women than in men, with a male to female ratio of 1:1.8 [14] showed an increased IgA and IgM mean serum levels were 2.49 g/L and 1.32 g/L in men and 1.98 g/L and 0.68 g/L in females, respectively, out of patients with reticular lichen planus. Thus, the results indicate that the mean serum IgA and IgM level were decreased in females compared to males in reticular OLP but the mean serum IgA level was slightly decreased and IgM was significantly increased in females compared to males in erosive OLP. In other study, showed that the mean serum level of IgA was decreased in females with comparison to males, [15]. IgA level in patients and the control group aged 20–40. This study concludes that IgA levels are significantly essential in people with lichen planus [16]. Also other research found that compared with the control group, OLP and patients had a significant increase in the average concentration of both stimulated and unstimulated salivary IgA [17].

Other study, indicated that the OLP patients had significantly higher levels of serum and salivary cytokines (tumor necrosis factor-alpha and interleukin-6), two key immunological factors implicated in systemic inflammation, than the controls. And, IgA and IgG levels in the saliva were elevated; only IgA levels showed a statistically significant change [18]. In other results reported that there was a significant difference between OLP patients and healthy controls in terms of salivary IgA and IgG levels, both of which were elevated [19]. According to Previous study, individuals with both non-erosive and erosive lichen planus had higher serum IL-17 levels than people in good health. Furthermore, erosive oral lichen planus patients had a significantly greater serum IL-17 level than non-erosive individuals [20]. Another study on interleukin 17 and transforming growth factor beta 1 in the hepatitis C patient group was carried out and The study found that the hepatitis C patient group had higher levels of IL17 than the control group [21]. In contrast, OLP patients had higher complement component C3 expression compared to healthy control subjects. Fibrinogen fragment D and complement component C3c were shown to be more expressed in the saliva of OLP patients, according to ELISA assays. The expression of salivary complement C3c varied statistically significantly between OLP patients and the healthy control group [22]. Through quantitative IgG antibody quantification using ELISA, the study had higher levels of IgG antibodies to HSV-1 than the control subjects [23]. Testing for HSV-1 and HSV-2 IgG antibodies in serum revealed that all tested OLP patients had an increase in HSV-1 IgG titer, while seven (41.2%) OLP participants had an increase in HSV-2 IgG. Nonetheless, IgG antibodies against HSV-1 and HSV-2 were also produced by normal, healthy people [24]. Elevations in CRP, IL-1α, IL-6, and TNF-α may be connected to oral carcinogenesis and OLP [25]. discovered links between liver cirrhosis, hepatitis B, and hepatitis C and LP. Patients with liver disease (LP) had a greater incidence of liver cirrhosis than the control group. In clinical practice, liver cirrhosis new onset may be significantly predicted by OLP [26].

In previous study showed that the chronic inflammatory disease known as oral lichen planus (OLP) affects epithelia with squamous differentiation. Our objective was to characterize the recurrence patterns of 'patients oral cavity' squamous cell carcinoma that developed in the context of oral lipoprotein poisoning [27]. Another study showed that 46 patients (52.9%) in the OLP group experienced one or more recurrences. The expected mean number of recurrences per patient after 10 years was 1.93 [28]. Another study found a number of genes interact with one another directly or indirectly to produce the complicated physiopathology of OLP. Genes encoding cytokines have polymorphisms that are essential for controlling the immune response. These variations result in different functional scenarios, which ultimately impact the development and course of the disease [29].

Some Studies showed OLP patients, had significantly higher mean salivary IgA and IgG levels than the normal group (P-value < 0.05) [30]. Also, previous study found In comparison to healthy controls, patients with OLP had more levels of both IgA and IgG in their saliva [31]. This is consistent with findings from earlier research that showed elevated serum IgA and IgG levels in OLP patients [32]. Compared to reticular OLP, erosive OLP has more strong IL-17 overexpression, which may indicate a positive relationship between IL-17 levels and disease severity [33]. According to) found, that Reticulated and erosive oral lichen planus may be pathophysiologically associated with Th17, and Th2 cells, respectively [34]. showed that Patients with OLP increased production of cytokines, such as TNF-α (tumor necrosis factor-alpha), IFN-γ (interferon-gamma, a pro-inflammatory signaling molecule), and IL-17 (interleukin-17, a cytokine implicated in immune response modulation). Interleukin 17 (IL17), which is released during inflammation in a variety of infectious disorders, has also been connected to this function [35]. Even though both conditions are inflammatory and eventually progress to chronic stages of carcinoma, further research is needed to understand the pathophysiology of specific biomarkers. Thus, the purpose of this work is to evaluate biomarkers related to the pathophysiology of OLP and HCV [36]. Numerous cytokines, including IFN-γ, TNF-α, IL-4, and IL-10, have been identified to have gene polymorphisms that may contribute to an individual's susceptibility to OLP [37]. Previous study discovered that interleukin-17 (IL-17), a proinflammatory cytokine, has a role in the inflammation associated with numerous autoimmune disorders. We measured the amounts of IL-17 in the serum and tissues of patients infected with the chronic hepatitis B virus (HBV), and we specifically assessed the function of IL-17 in the development and course of liver fibrosis [38]. The inflammatory response of the hepatic tissues in CHB, a significant amount of TGF-β can be produced by activated interstitial cells. In order for IL-17 to differentiate, TGF-β is essential. And Found That degree of liver fibrosis was observed to correlate with an increase in IL-17 expression. This implies that IL-17 may promote chronicity and the advancement of the disease in addition to inducing inflammation [39]. On the other side, Patients with OLP had lower serum and salivary levels of total C3 and C4 than did healthy controls. Consequently, they might be able to distinguish between OLP and healthy [40]. Park and Eung, (2000) showed the average HSV-1 and HSV-2 IgG titers were, across seventeen patients for both viruses. In comparison to the normal range, these were 10.5 and 3.5 times higher [41].

On the other side, in the patients with oral lichen planus, serum levels of TNF-α are significantly elevated. The inflammatory process during the course of the disease is explained by the elevation of TNF-α serum levels in OLP patients [42]. The TNF-α level was higher in OLP patients than in normal people, suggesting that measuring TNF-α levels could help determine prognosis and therapy efficacy.

5. Conclusion

The concentrations of IgA and IL-17 both increase significantly and the concentration of C3 also decreases significantly when infected with oral lichen planus, and the condition is more severe with viral infections. And, The recurrence of lichen infection appears at a reasonable rate, indicating that a hereditary or genetic factor played a role in the recurrence of the infection.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest

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