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STUDY CORRELATION BETWEEN SOME IMMUNE MARKER in PATIENTS with LICHEN PLANUS

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Abstract:
Lichen planus is an autoimmune disorder that primarily affects the body's skin and multiple mucous membranes. In multicellular species, protein 53 and syndecan-1protein play an significant role since they control the cell cycle and thus play a major role in tumor suppression and cancer prevention. The p53 gene was therefore identified as a "genome protector," referring to its role in preserving the integrity of genetic information by preventing gene mutations. The purpose of this analysis was to study the association between certain immune markers in lichen planus patients.
The aim of this study : Study correlation between some immune marker in patients with lichen planus
Methods 'Thirty formalin-fixed , paraffin-embedded tissue pieces, diagnosed as Oral Lichen planus(OLP), were included in the sample of this report.
Results: Evaluation of P53 Immunohistochemistry and Evaluation of syndecan-1protein Immunohistochemistry Conclusion: the marker p53 expression is high in the patient with Lichen Planus while the second studed marker syndecan-1 protein is less than marker p53 .

Keywords: inflammatory disease, immune marker, patients, lichen planus,p53.

INTRODUCTION

Lichen planus is an inflammatory condition that affects the skin and the body 's distinct mucous membranes in particular. The skin that is on the inside, Lichen planus is marked by purple-coloured superficial ulcers that also cause inflammation and irritation[1]. When the lichen planus emerges on the mucous membrane wall, The sores are white, with a stripe-like pattern, and can be uncomfortable, such as: the oral cavity and vagina. Lichen planus is not infectious because it is an autoimmune condition that, for no reason, stimulates the immune response against the body's own cells[2]. The element which triggers an attack on the body by the immune system is undisclosed. The primary explanation is that the immune system targets the skin and mucous membranes and occurs as a consequence of the immune reaction of the body and there is no specific trigger for this immune response to occur, although there may be certain causes that can raise the probability of its existence and induce lichen planus such as the genetic factorOf certain dyes and additives, as well as the use of influenza vaccines and radiation[3,4].This means researching the immune system 's responses and materials. In the experimental research, numerous immunohistochemistry techniques were developed and enhanced, In separate fields of virology, lichen histopathology is characterized by an abnormal expansion of the upper layers of the skin (epidermis) and the colloid bodies surrounding them with the degradation of the basal layer[5,6]. A lichenoid ring-like ring in the upper dermis comprises of the lymphocytes and histiocytes. P53 (also known as the protein suppressor in tumors 53), Is a human tumor suppressor gene encoded in humans with the TP53 gene for chromosome 17[7]. Protein 53 plays an important role in multicellular

organisms because it controls the progression of the cells and thereby plays an important role in tumor suppression and cancer prevention[8]. He thus identified the p53 gene as a "genome protector," Referring to its role in keeping genetic material intact through the avoidance of gene mutations. Lichen planus is an infectious condition that predominantly affects the body 's skin and separate mucous membranes[9]. It is not infectious when, for no reason, the immune system triggers against the body's own cells[10].The factor that causes immune system to attack the body is unknown. The use of influenza vaccines and exposure to certain dyes and chemicals may increase the likelihood of its occurrence[11- 32]. The histopathology of lichen is characterized by irregular enlargement of the upper layers of skin (epidermis) and colloid bodies around them with destruction of the basal layer (Epidermis). P53 (also known as tumor suppressor protein 53), is a human tumor suppressingor gene encoded by the TP53 gene on chromosome 17 in humans. It plays an important role in multicellular organisms because it regulates the cell cycle and plays a major role in suppressing tumors and preventing cancer[12,13].The aim of this study : Study correlation between some immune marker in patients with lichen planus .

MATERIAL AND METHODS

"The Sample of this study included thirty formalin-fixed, paraffin- embedded tissue blocks, which have been diagnosed as Oral Lichen planus(OLP), dated from (2016 till 2017) The study samples were obtained from the archives of the department of Oral and Maxillofacial Pathology/ College of Dentistry/ University of Baghdad.

Control groups:
Positive control:- Positive control was (accomplished or obtained with effort) based on the disease-fighter manufacturer's data sheet. Slides were developed from blocks of patients with tissue believed to contain the target (a germ the body is attempting to defend against).

Negative control:- This has been used to (show or demonstrate) the habit / desire of staining ways of doing stuff as a constructive control. One adverse control was used for each series of test runs.

Immunohistochemistry(IHC)
Immunohistochemical staining is used a great deal in the oral lichen planus OLP (identification of a disease or problem, or its cause) .

According to Dako 's detection device instructions, immunohistochemistry tests were treated and done . Slides were washed with a cleaning buffer, Peroxidase-Blocking Reagent was added and all slides were developed / grown for 10 minutes. A double-headed light microscope was used to grade slides by a scientist who is a disease specialist. In at least 10 percent of the cell centers (of cells or atoms), positive centers (of cells or atoms) staining p53 is cautiously considered / believed to be p53 overexpression. Those with less than 10% of a cell or atom 's positive cell center is considered / believed to be natural expression. According to the DAKO rules of conduct, the p53 values are cut off. At room temperature in the fume hood, the stained slides were left to dry for at least one hour and then gathered together by a DPX mounting medium. Immunohistochemistry tests were treated and performed according to the instructions of Dako's detection method. When washing the slides with a laundry shield, Peroxidase-Blocking Reagent was added and all slides were created / grown for 10 minutes. The edges around the samples were marked by liquid blocker pap ink in order to prevent the distribution of the materials from the samples during the IHC run. After (more than two, but not many) experiments, the best incubation time of 60 minutes was determined using various

incubation times (20, 30, 45, 60 and 90 minutes) for the collection of Ag samples, the washing buffer washed the samples, the reagent blocking (Ab diluent) was applied for 30 minutes to eliminate the excess position of other non-clearly stated / particular proteins and to minimize the growth. A qualified physician (a doctor who is a disease expert) used a double-headed light microscope to grade slides. Cut-off values were carried out with the help of a (doctor who is a disease expert) for all disease fighters used in the analysis [14,15]. In at least 10 percent of cell centers (of cells or atoms), positive centers (of cells or atoms) staining p53 were carefully considered / believed p53 overexpression, while those with less than 10 percent positive cell center (of a cell or atom) were considered / believed normal expression. The stained slides were left for at least one hour to dry in the fume hood at room temperature. [16,17].

Statistical Analyses

Test with Chi-square and mean ± S.D. They were included in clinicopathology trials. All the mathematical work was performed in SPSS version 13.0 (SPSS, Inc., Chicago, USA) and in Microsoft Excel[16].

RESULTS AND DISCUSSION

Evaluation of P53 Immunohistochemistry

Positive P53 Immuno staining was observed in all cases of oral lichen plate (OLP) as brown nucleic expression (1,2,3,4,5) cases of P53 immuno staining of oral lichen plate (OLP) were summarized in Table (1) showing that (2) cases (6,668%) had strong positive expression, (8) cases (26,672%) had strong positive expression, and (20) cases (66,66%) displayed moderate positive expression.

Table (1): P53 IHC expression in cases involving oral lichen planus (OLP)

P53 score*	No.	%
1	2	6.668%
2	8	26.672%
3	20	66.66%
Total	30	100%

*1(weak expression),2 (moderate expression), 3 (strong expression).

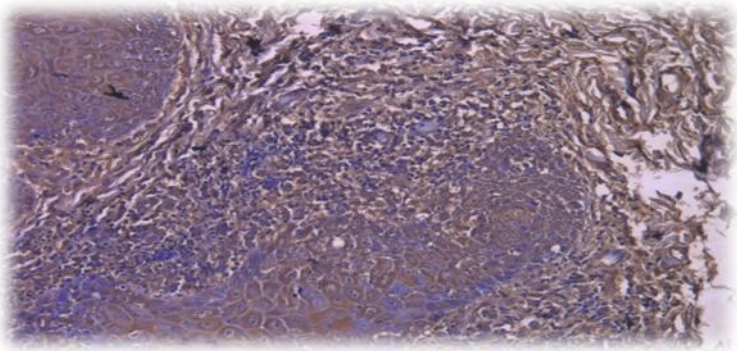


Figure (1) Positive immunofluorescence in moderately differentiated oral lichen (OLP) (10x)

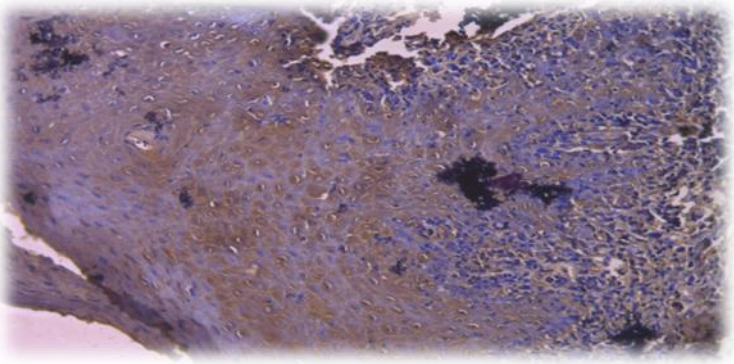


Figure (2) Immunohistochemistry in (OLP) (10x).

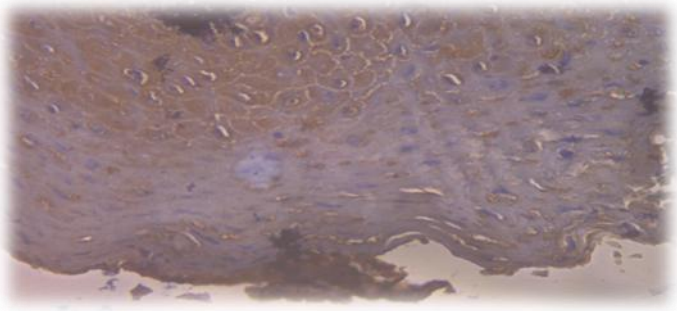


Figure (3) P53 positive immunofluorescence (OLP) (40X)

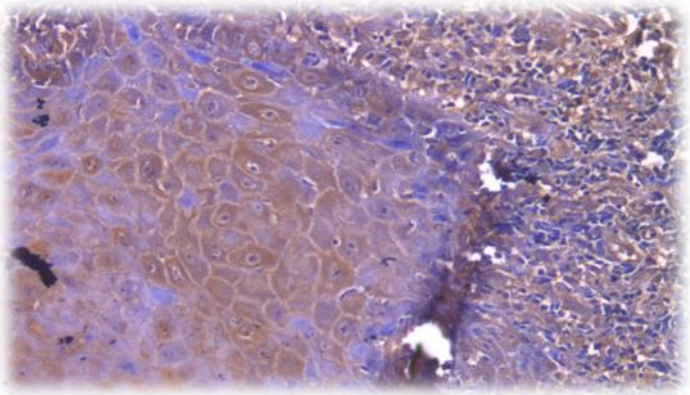


Figure (4) Positive Immunohistochemistry for the Brown Protein Nucleus (40X)

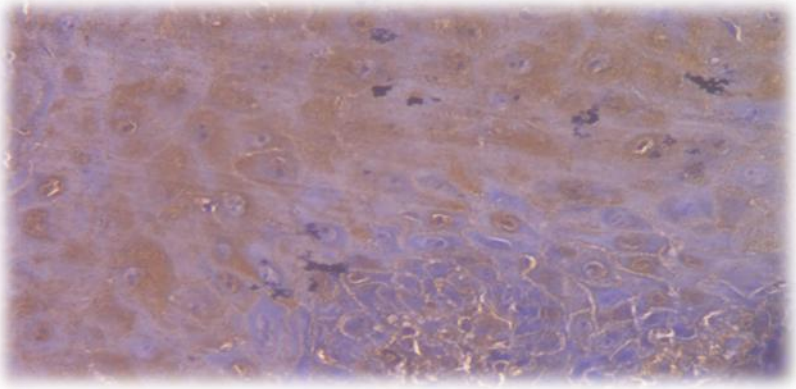


Figure (5) Positive immunohistochemistry inoculation of the brown protein p53 in differentiated poor oral lichen planus (OLP). (40X)

17 cases (56.7 percent) were male and 13 cases (43.3 percent) were female and about 1.3:1 were male / female[18,19,20]. Over the past half century, the difference in the male : female ratio has become less profound and this trend has been due to a growth in women's

consumption of smoking and alcohol. The most predominant location was the lips, which is in line with the results of previous research. 17 cases (56.7 percent) were male and 13 cases (43.3 percent) were female and about 1.3:1 were male / female[21]. Over the past half century, the difference in the male : female ratio has become less profound and this trend has been due to a growth in women's consumption of smoking and alcohol.The most predominant location was the lips, which is in line with the outcomes of prior research. The lower lip is the most common site and a common site for the development of oral lichen plauns is the lower lip. Prolonged exposure to environmental carcinogenesis might clarify the correlation between the growth of oral Lichen Plauns (OLP) and aging[22] [31].

Evaluation of syndecan-1protein Immunohistochemistry
As a dark cytoplasmic staining of cells, positive syndecan-1protein immunostaining has been observed. Illustrations (6). Positive IHC expression was reported in all cases of oral lichen planus (OLP) as shown in Table (2), which indicates that (6) cases (20.004%) showed poor positive expression, (7) cases (23.338%) showed strong positive expression, and (17) cases (65.678%) showed high positive expression.

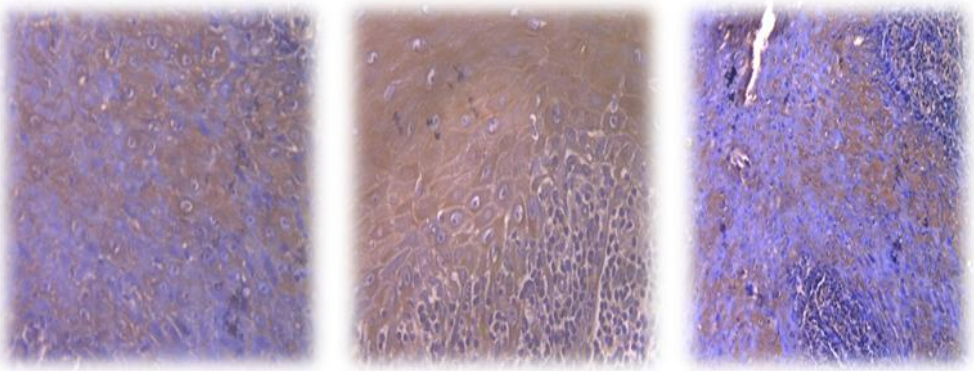
Table (2): In oral lichen planus (OLP) cases, syndecan-1protein IHC expression

syndecan-1protein score*	No.	%
1	6	20.004%
2	7	23.338%
3	17	56.678%
Total	30	100%

*1 (weak expression), 2(moderate expression), 3(strong expression).

The results of this study demonstrated a statistically non-significant relationship with the expression of syndecan-1 protein in relation to age (p-value=0.181) and gender (p-value=0.276) according to the chi-square test, and demonstrated a statistically significant correlation with respect to age (p-value=0.181) and gender (p-value=0.276, (p-value=0.276, (p-value=0.181)

Significant correlation of expression of syndecan-1protein with expression of the cell site (p-value=0.015) and clinical expression (p-value=0.003)



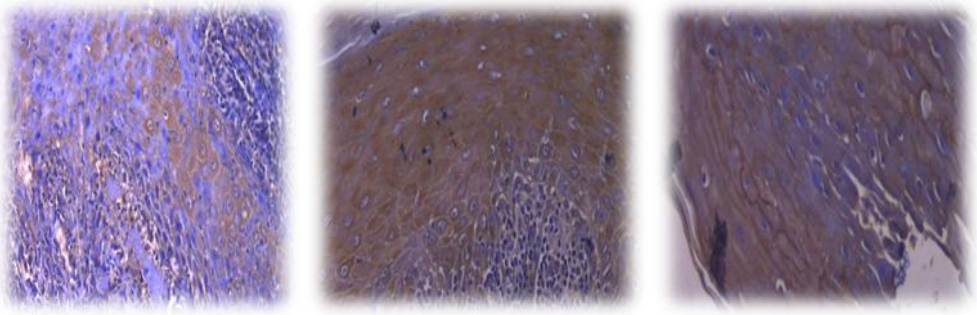


Figure (6) immunostaining of syndecan-1protein in (OLP)

Endophytic(ulcer) clinical function (63.3 percent) 19 cases followed by exophytic mass, which (36.7 percent) 11 cases, was the most prevalent clinical presentation reported in this report.

Endophytic(ulcer) clinical function (63.3 percent) 19 cases followed by exophytic mass, which (36.7 percent) 11 cases, was the most prevalent clinical presentation reported in this report. The fact that the present research and some of the others are not an epidemiological style of analysis may be due to these discrepancies, because the small number of cases prevent conclusive clinical results,These findings are consistent with previous studies[23].

Over the last half century it has been less profound and this trend is due to the rise in women's smoking and alcohol use. However, depression and the increase in the number of female factory employees may be contributing causes, the tongue is the most prevalent site, and this is consistent with previous findings and varies from other research which have shown that the most prevalent location is the buccal mucosa, and indeed those which have shown that the lower lip is the most prevalent site[24].

In both oral lichen planus (OLP) cases, the findings of this analysis showed good syndecan-1 protein expression. The protein released by cancer cells is required to activate the cancer cells themselves and/or the broblasts for cancer invasion and development[25].

The findings of this analysis revealed positive syndecan-1 protein expression in all cases of oral lichen planus with a high positive score (56.678 percent) of cases[26]. The present finding was consistent with previous reports[27]Immunohistochemical review of syndecan-1 protein expression revealed that anti-syntecan neutralization therapy,1 protein pooled around oral lichen .[28,29]The study concluded that higher protein expression was demonstrated by cancer cells with greater invasion potential. The protein released by cancer cells is required to activate the cancer cells themselves and/or the broblasts for cancer invasion and development[30].

CONCLUSION

We concluded from the study that the marker p53 expression is high in the patient with Lichen Planus while the second studed marker syndecan-1 protein is stronger than marker p53.

REFERENCES

[1] Ghaderi R, Makhmalbaf Z. The relationship between lichen planus and hepatitis C in Birjand, Iran. Shiraz E-med J. 2007;8:72–9.
[2] Beaird LM, Kahloon N, Franco J, et al. Incidence of hepatitis c in lichen planus. J Am Acad Dermatol. 2001;44:311–1.

[3] Tovaru S, Tovaru M, Costache M, et al. Medicina si patologie Orala volumul I Q Med Publishing 2008
[4] Nagao Y, Myoken Y, Katayama K, et al. Epidemiological survey of oral lichen planus among HCV infected inhabitantsin a town in Hiroshima Prefecturrein Japan from 2000 to 2003. Oncol Rep. 2007;18:1177–81.
[5] Kirtak N, Inaloz HS, Ozgoztasi O, et al. The prevalence of hepatitis Cvirus infectionin patients with lichen planus in Gazinateq region of Turkey. Eur J Epidemiol. 2000;16:1159–61.
[6] Karaverioglu D, Koytak ES, Bazkaya H, et al. Lichen planus and HCV infection in Turkish patients. Turk J Gastroenterol. 2004;15:133–6.
[7] Harman M, Akdeniz S, Dursun M, et al. Lichen planus and hepatitis C virus infection: an epidemiologic study. Int J clin Pract. 2004;58:1118–9.
[8] Denli YG, Durdu M, Karakas M. Diabetes and hepatitis frequency in 140 lichen planus cases in Cukurova region. J Dermatol. 2004;31:293–8.
[9] Gimenez-Garcia R, Perez-Castrillon JL. Lichen planus and hepatitis C virus infection. J Eur Acad Dermatol Venerol. 2003;17:291–5.
[10] Giuliani M, Lajolo C, Miani CM, et al. Hepatitis C virus chronic infection and oral lichen planus: an italian case control study. Eur. J Gastroenterol Hepatol. 2007;19:647–52.
[11] Lodi G, Giuliani M, Majorana A, et al. Lichen planusand hepatitis C virus: a multicentre study of patients with oral lesions and a systematic review. Br. J Dermatol. 2004;151:1172–81.
[12] Mouelhi L, Debbeche R, Sfar I, et al. Auto-immune serological disorders in chronic viral C hepatitis: prevalence and clinical significance. Tunis Med. 2008;86:777–81.
[13] Ko HM1, Hernandez-Prera JC, Zhu H, et al. Morphologic Features of Extrahepatic Manifestations of Hepatitis C Virus Infection Clinical and Developmental Immunology. 2012;2012:740138–740138.
[14] Ibrahim HA, Baddour MM, Morsi MG, et al. Should we routinely check for hepatitis B and C in patients with lichen planus or cutaneous vasculitis? East Mediterr Health J. 1999;5:71–8.
[15] Gheorghe L, Csiki IE, Iacob S, et al. The prevalence and risk factors of hepatitis C virus infection in adult population in Romania: a nationwide survey 2006-2008. J Gastrointestin Liver Dis. 2010;19:373–9.
[16] Molnar GB, Cocean S, Jebeleanu L, et al. Evaluarea epidemiologica a infectiei anamnestice cu virus hepatitic C la populatia din 11 judete ale Romaniei. Rom J Infect Dis. 2005;8:3–7.
[17] Mohd Hanafiah K, Groeger J, Flaxman AD. Global epidemiology of hepatitis C virus infection: New estimates of age-specific antibody to HCV seroprevalence. Hepatology. 2013;57:1333–42.
[18] Sugerman PB, Savage NW, Walsh LJ, et al. The pathogenesis of oral lichen planus. Crit Rev Oral Biol Med. 2002;13:350–65.
[19] Tovaru S. Incidence of oral lesions in a selected hospital populations Poster 11 th Biennal Congress of EAOM Athens 2012
[20] Scully C, Carozzo M. Oral mucosa disease-lichen planus. Br J Oral Maxilofac Surg. 2008;46:15–21.
[21] Roopashree MR, Gondhalekar RV, Shashikanth MC, George J, Thippeswamy SH, Shukla A. Pathogenesis of oral lichen planus--a review. J Oral Pathol Med 2010;39:729-34.
[22] Sagari S, Sanadhya S, Doddamani M, Rajput R. Molecu-lar markers in oral lichen planus: A systematic review. J Oral Maxillofac Pathol 2016;20:115-21.

[23] Hadzi-Mihailovic M, Cakic S, Jankovic S, Raybaud H, Nedeljkovic N, Jankovic L. Ki-67 expression in oral li¬chen planus. J BUON 2012;17:132-7.
[24] Irani S, Esfahani AM, Ghorbani A. Dysplastic change rate in cases of oral lichen planus: A retrospective study of 112 cases in an Iranian population. J Oral Maxillofac Pathol 2016;20:395-99.
[25] Oliveira Alves M, Balducci I, Rodarte Carvalho Y, Cabral L, Nunes F, Almeida J. Evaluation of the expression of p53, MDM2, and SUMO-1 in oral lichen planus. Oral Dis 2013;19:775-80.
[26] Vousden KH. p53:death star. Cell 2000;103:691-4.
[27] de Oliveira MG, Ramalho LM, Gaião L, Pozza DH, de Mello RA. Retinoblastoma and p53 protein expression in pre-malignant oral lesions and oral squamous cell carcinoma. Mol Med Rep 2012;6:163-6.
[28] Sankari SL, Babu NA, Rajesh E, Kasthuri M. Apoptosis in immune-mediated diseases. J Pharm Bioallied Sci 2015;7(Suppl 1):S200-2.
[29] Dheeb B. I., (2015). Antifungal Activity of Alkaloids and Phenols Compounds extracted from black pepper Piper nigrum against some pathogenic fungi Jornal of Biotechnology Research Center (2015) 9 , 46-54.h.
[30] Lin T, Lin Y. p53 switches off pluripotency on differen-tiation. Stem Cell Res Ther 2017;28:8.
[31] Khaleel, Z. I., Mohammed, Z. H., & AL-Samarraie, M. Q. (2019). Histological Effect of the Alcoholic Extract of Nerium Oleander in the Heart and Brain in Mice and its Effect on the Lymphocytes (In Vitro). Indian Journal of Public Health Research & Development, 10(8), 2362-2366.
[32] Abdulazeez, M., Hussein, A. A., Hamdi, A. Q., & Mustafa, M. A. (2020). Estimate the Complications That Resulting from Delayed Management of Dental Trauma in Tikrit City. Journal of Cardiovascular Disease Research, 11(2), 80-82.