

Study Correlation between Some Immune Markers in Patients with Lichen Planus: Review Article

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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-1 protein play a 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 markers 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-1 protein Immunohistochemistry

Conclusion: The marker p53 expression is high in the patient with Lichen Planus while the second studied markersyndecan-1 protein is less than marker p53.

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

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 factor of 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]. 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 suppressing or 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.

I. 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

II. 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 over expression. 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 over expression, 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 \pm 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].

III. 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).

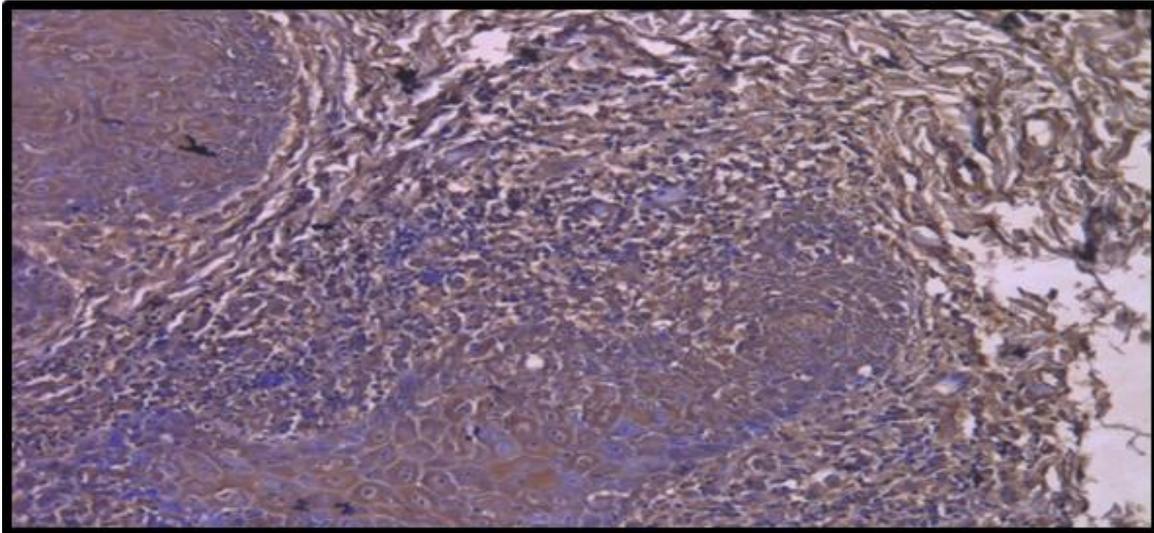


Figure 1: Positive immunofluorescence in moderately differentiated oral lichen (OLP) (10x).

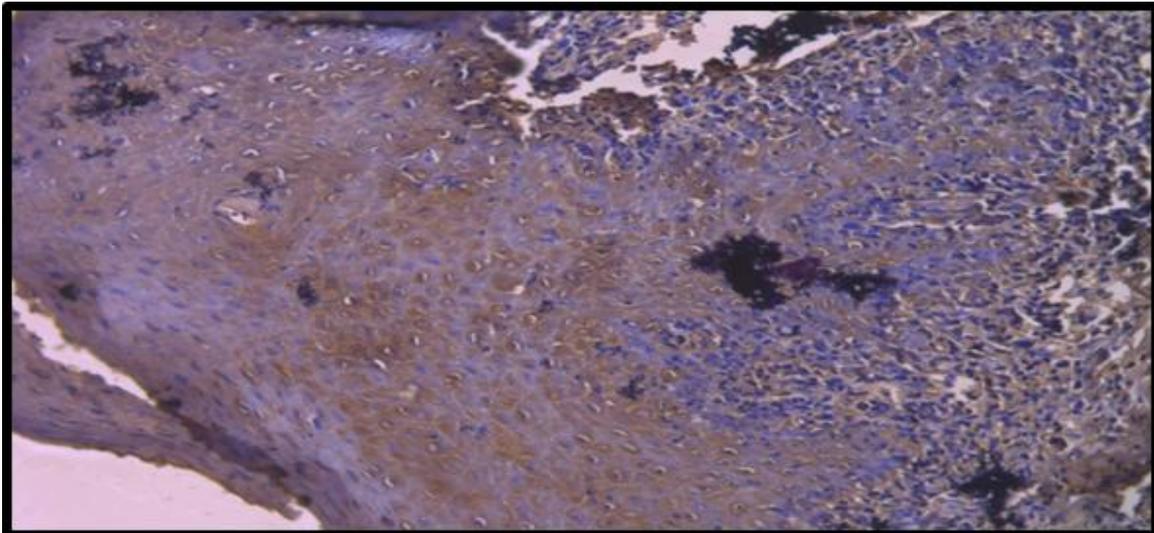


Figure 2: Immunohistochemistry in (OLP) (10x).

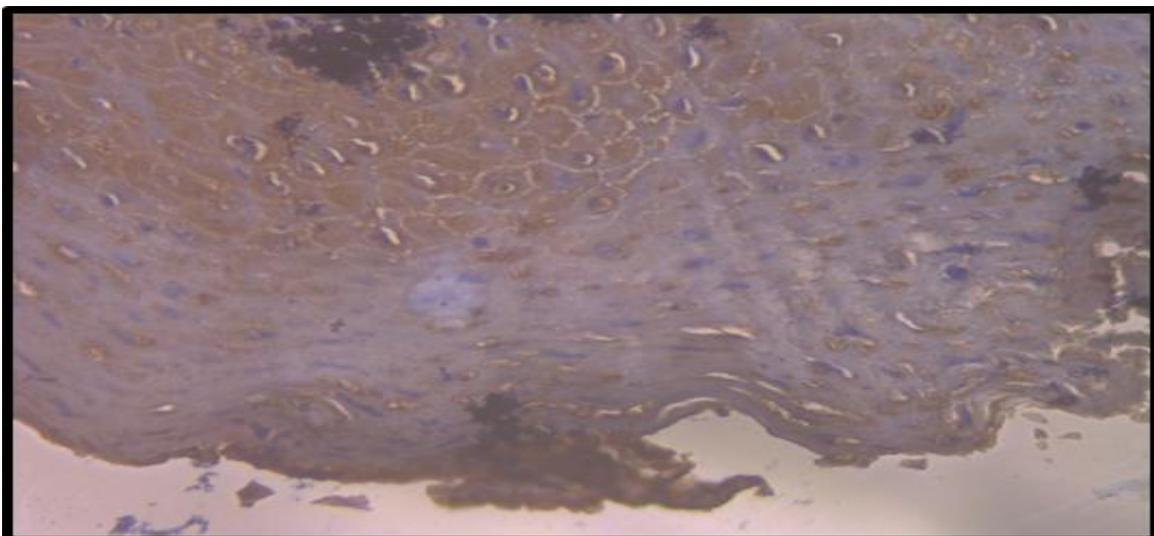


Figure (3) P53 positive immunofluorescence (OLP) (40X).

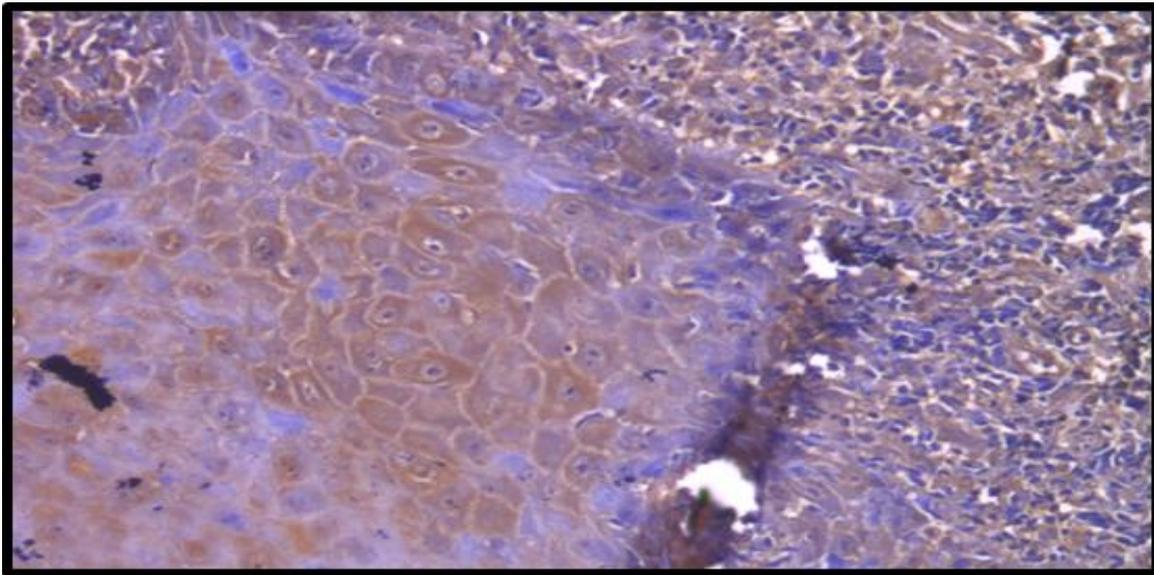


Figure 4: Positive Immunohistochemistry for the Brown Protein Nucleus (40X).

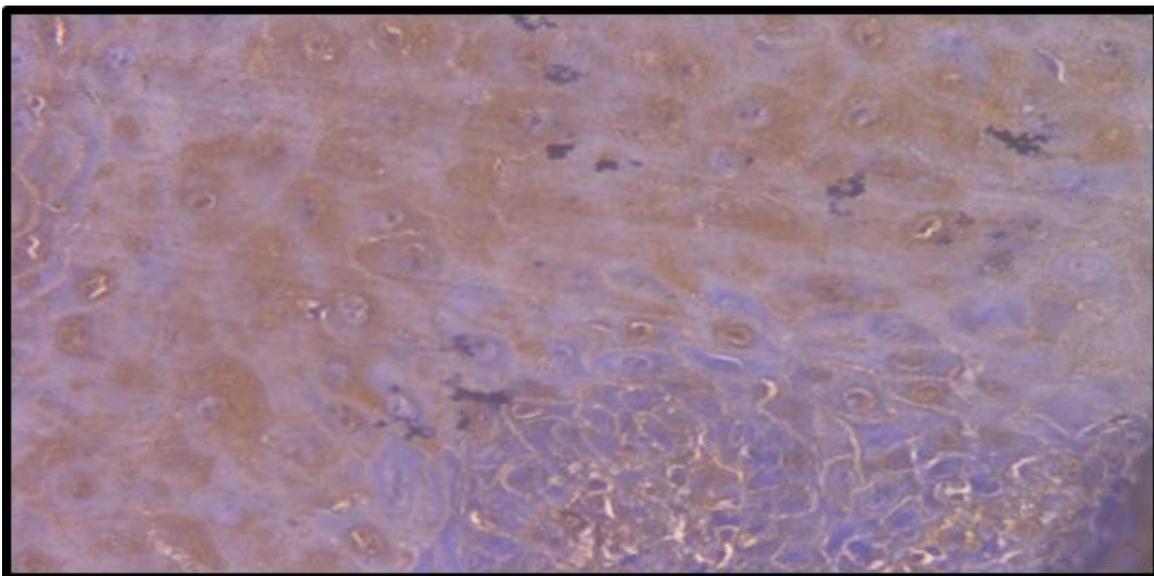


Figure 5: Positive immunohistochemistry inoculation of the brown protein p53 in differentiated poor oral lichen planus (OLP) (40X).

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].

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

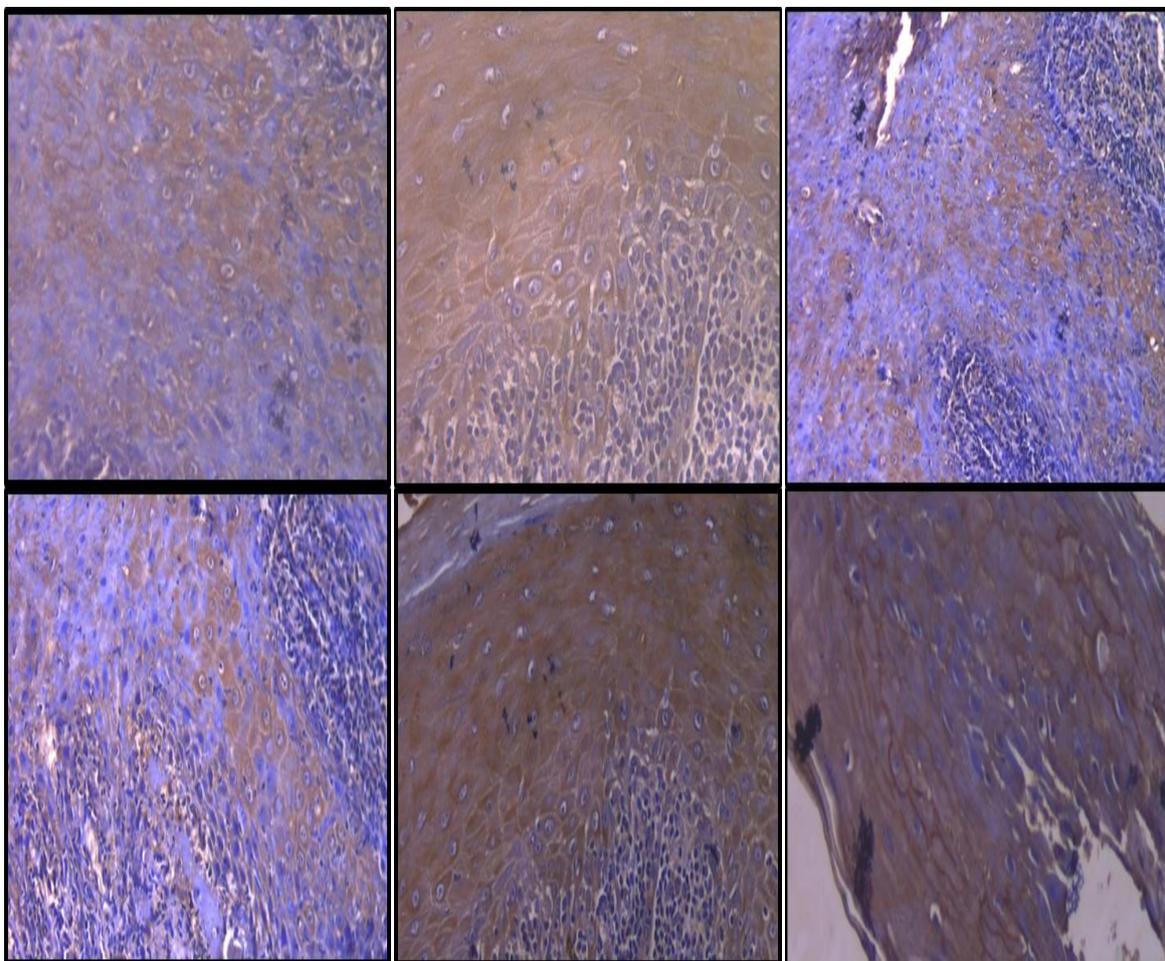


Figure 6: Immunostaining of syndecan-1protein in (OLP).

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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 fibroblasts 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-syndecan 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 fibroblasts for cancer invasion and development[30].

IV. CONCLUSION

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

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