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RESEARCH ARTICLE

LICHENOID CONUNDRUM: A CLINICOPATHOLOGICAL STUDY.

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Abstract

Aim: To establish a correlation between the clinical and histopathological features in the diagnosis of Oral Lichen Planus (OLP) and Oral Lichenoid Lesions (OLL).

Objective: To compare the clinicopathological features in the diagnosis of OLP and OLL based on modified WHO diagnostic criteria proposed by Van der Meij and Van der Waal in 2003 and the criteria proposed by Cheng at al (2016) for OLP.

Materials and Methods: Thirty patients with a clinical provisional diagnosis of oral lichen planus, referred for incisional biopsy were clinically and histologically evaluated using haematoxylin and eosin stained sections based on the modified WHO criteria. This was compared with Cheng's proposed criteria for OLP.

Results: The number of patients diagnosed with Oral Lichenoid lesions was higher using the criteria proposed by Cheng et al.

Introduction:-

Lichen Planus is a T-cell mediated disease with various triggering and contributing factors affecting the skin and mucous membrane.[12] It is a chronic inflammatory disease frequently involving the oral cavity. The prevalence in the Indian population is 2.6% as compared to the Japanese population(0.5%) and the Swedish population(1.9%).[24] Various non-idiopathic oral mucosal lesions simulate oral lichen planus clinically as well as histopathologically which are known as oral lichenoid lesions. These lesions are attributed to type IV hypersensitivity reactions and may be seen in association with tobacco, amalgam, cinnamon and various categories of drugs. [2,30,31]

Of specific importance is the occurrence of oral epithelial dysplasia with lichenoid features and the potential for malignant transformation. The overall malignant transformation rate of OLP is estimated to be 1–2% [23] while that in OLL was estimated to be higher. These lichenoid lesions differ in their etiology, treatment modalities and prognosis, making the distinction between oral lichen planus (OLP) and oral lichenoid lesions (OLL) mandatory.
Hence, this study aimed to establish a correlation between the clinical and histopathological features in the diagnosis of OLP and OLL based on the modified WHO diagnostic criteria proposed by Van der Meij and Van der Waal in 2003 and the criteria proposed by Cheng at al (2016) for OLP.

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Materials And Methods:-

The study included 30 patients who attended the Goa Dental College and Hospital from the year 2017-2018 who were referred for an incisional biopsy with a clinical provisional diagnosis of Oral Lichen Planus. The patients were clinically re-evaluated in the Department of Oral and Maxillofacial Pathology, Goa Dental College and Hospital and the histopathological features of the haematoxylin and eosin stained sections were assessed, based on the modified WHO diagnostic criteria proposed by Van der Meij and Van der Waal in 2003 (Table 1).[4]

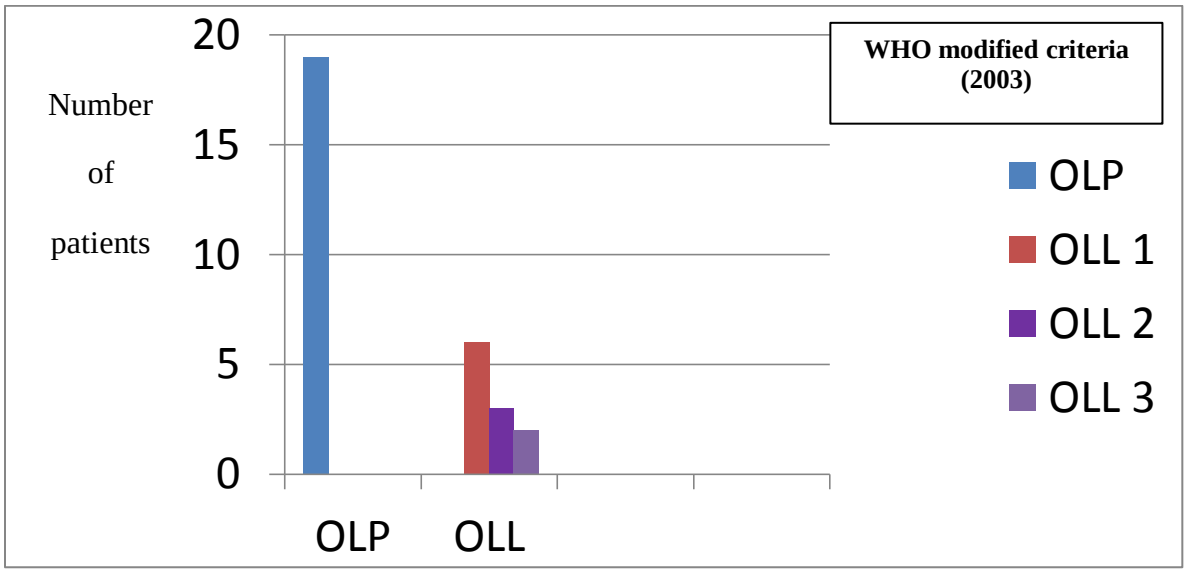
Modified who criteria
Clinical criteria
Presence of bilateral, more or less symmetrical lesions
Presence of a lace-like network of slightly raised gray-white lines (reticular pattern)
Erosive, atrophic, bullous, and plaque-type lesions are accepted only as a subtype in the presence of reticular lesions elsewhere in the oral mucosa
In all other lesions that resemble OLP but do not complete the aforementioned criteria, the term "clinically compatible with" should be used
Histopathologic criteria
Presence of a well-defined band-like zone of cellular infiltration that is confined to the superficial part of the connective tissue, consisting mainly of lymphocytes
Signs of liquefaction degeneration in the basal cell layer
Absence of epithelial dysplasia
When the histopathologic features are less obvious, the term "histopathologically compatible with" should be used
Final diagnosis OLP or OLL
To achieve a final diagnosis, clinical as well as histopathologic criteria should be included
OLP: A diagnosis of OLP requires fulfilment of both clinical and histopathologic criteria
OLL: The term OLL will be used under the following conditions:
Type 1: Clinically typical of OLP but histopathologically only compatible with OLP
Type 2: Histopathologically typical of OLP but clinically only compatible with OLP
Type 3: Clinically compatible with OLP and histopathologically compatible with OLP

Table 1:-Modified WHO diagnostic criteria proposed by Van der Meij and Van der Waal in 2003

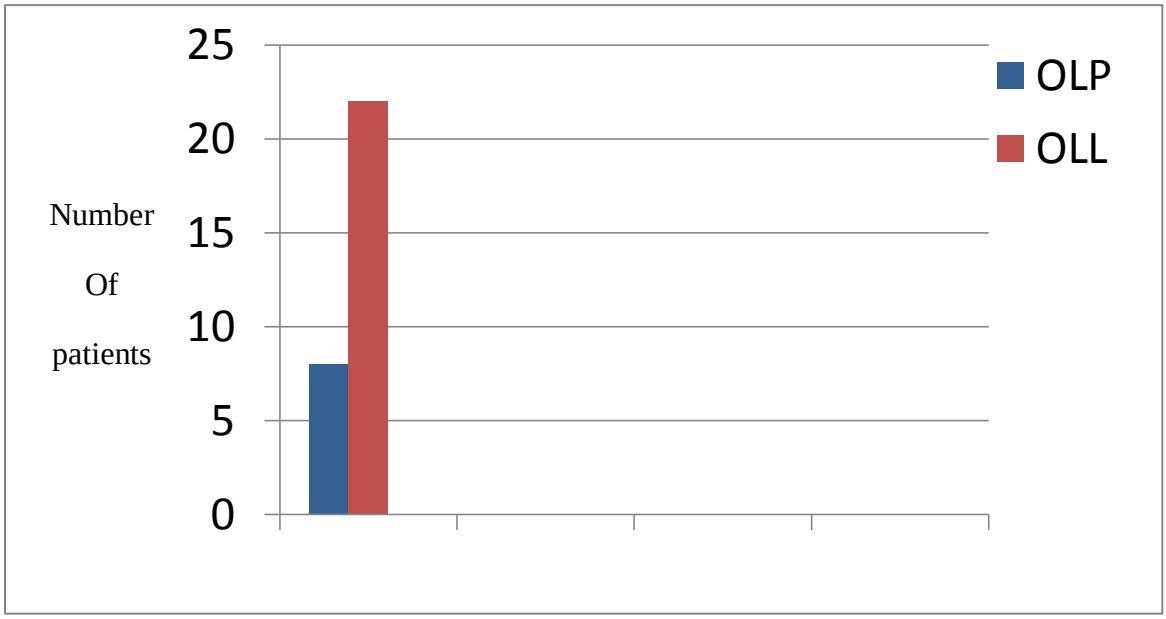
The results obtained were recorded then compared with the results obtained by using Cheng's proposed criteria for OLP (Table 2).[6]

CHENG'S PROPOSED CRITERIA FOR OLP
Clinical criteria
Multifocal symmetric distribution White and red lesions exhibiting one or more of the following forms: - Reticular/papular - Atrophic (erythematous) - Erosive (ulcerative) - Plaque - Bullous
Lesions are not localized exclusively to the sites of smokeless tobacco placement
Lesions are not localized exclusively adjacent to and in contact with dental restorations
Lesion onset does not correlate with the start of a medication
Lesion onset does not correlate with the use of cinnamon-containing products
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
Absence of verrucous epithelial architectural change

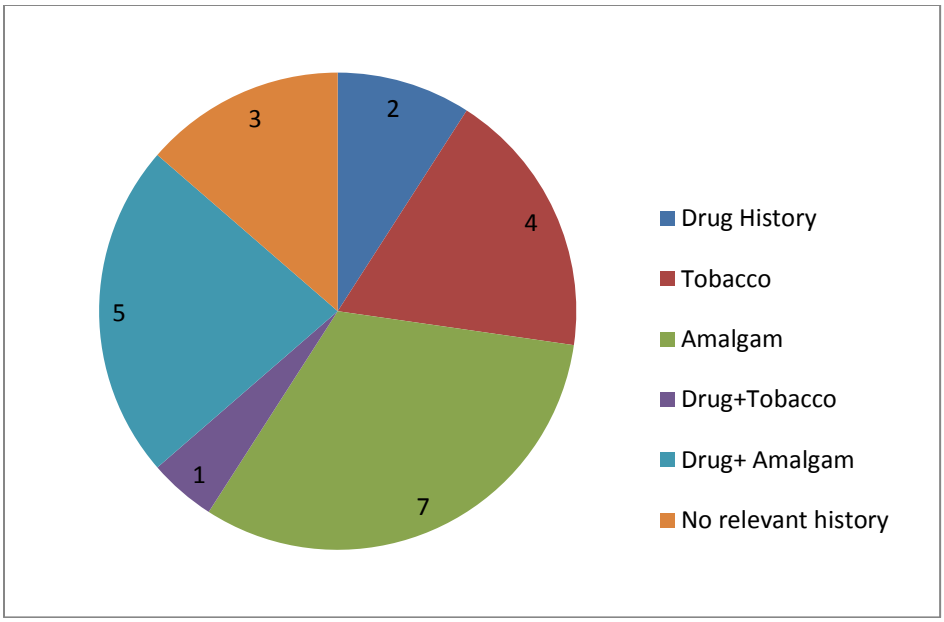
Results:-



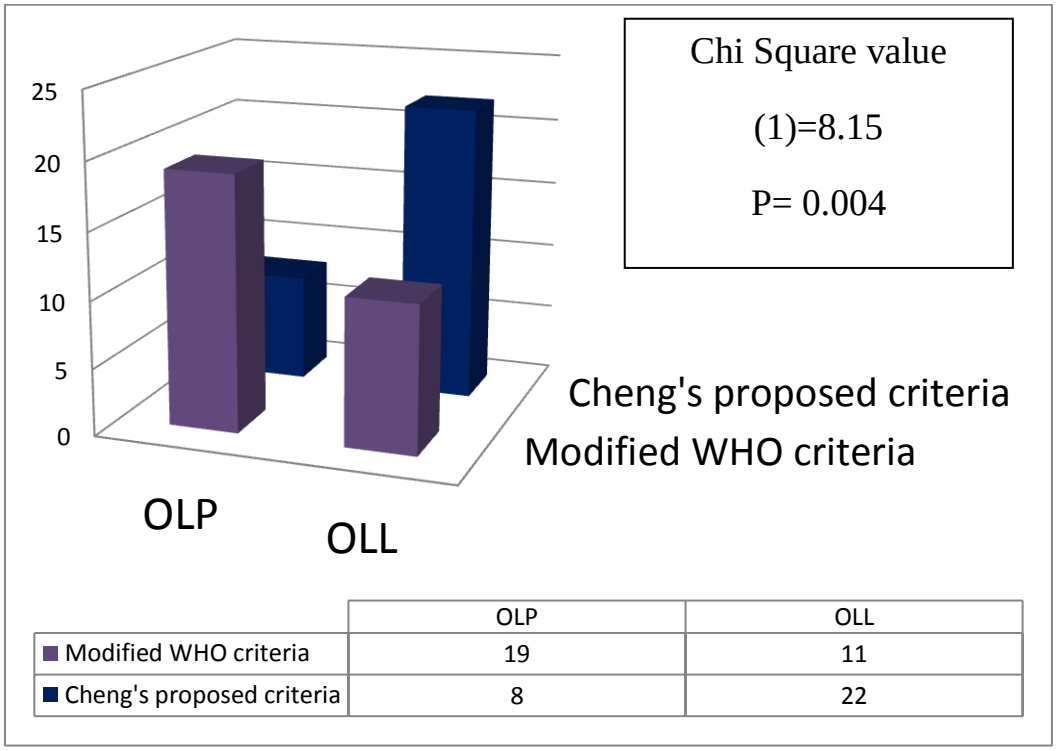
Graph 1:-Results obtained by using the WHO modified criteria (2003)



Graph 2:-Results obtained by using Cheng's proposed criteria



Graph 3:-Results obtained by using Cheng's proposed criteria



	OLP	OLL
Modified WHO criteria	19	11
Cheng's proposed criteria	8	22

Graph 4:-Comparing the results obtained by using the modified WHO criteria (2003) and Cheng's proposed criteria for OLP

Discussion:-

Lichen Planus was first described by the Austrian dermatologist Ferdinand von Hebra who termed the disease "lichen ruber". However, in the year 1869 an English surgeon and dermatologist Erasmus Wilson gave the first clinical description of Lichen Planus.[7] It most commonly affects the oral mucosa (Oral lichen planus), but can rarely involve other sites such as skin, genital mucosa, scalp and nails. 50% patients with cutaneous lesions have oral involvement whereas only 25% of patients with oral lesions have skin involvement. [9]

Pinkus published the first microscopic description of lichenoid reactions in 1973.[19] The term oral lichenoid lesion (OLL) was proposed by Finne *et al* in 1982.[20] OLLs are lesions clinically similar to OLP lesions and are described as lesions that may be related to dental restorations or cinnamon products or systemic medication associated with graft-versus-host disease or systemic illness.

A lesion clinically similar to OLP lesions but lacking the characteristic crisscrossing pattern of striae commonly noted in OLP, occurring among users of betel quid (BQ) was formerly called "oral lichen planus like-lesion". It was first described in 1980 by Daftary et al.[25] This lesion was said to have arisen from contact with BQ and is an oral lichenoid contact lesion type.[27] In 1996, this lesion was then termed as betel-quid lichenoid lesion (BQLL) in a workshop held in Kuala Lumpur, Malaysia.[28] Clinically, there is the presence of fine, white, wavy, and parallel lines that do not overlap or crisscross, are non-elevated, and in some instances radiate from a central erythematous area.[25] This lesion may regress with decrease in frequency, duration, or change in site of placement of the quid. There may be complete regression when the quid habit is given up. [26]

In 1910, Hallopeau reported a case of carcinoma arising in OLP, hence highlighting the potentially malignant nature of oral lichen planus (OLP). However, the overall malignant transformation rate of OLP is estimated to be 1–2%[23] while that in OLL was estimated to be higher.

Krutchkoff and Eisenberg (1985) introduced the term 'Lichenoid Dysplasia (LD)' to describe a distinct histopathologic entity defined as a lesion that histopathologically revealed characteristics of OLP with the additional presence of dysplastic features within the overlying epithelium.[21]

The word lichenoid is a histologic descriptive term encompassing any two or more of the following four criteria[21]:

1. hyperkeratosis or parakeratosis
2. superficial bandlike infiltrate in lamina propria composed chiefly of lymphocytes
3. intimate intermingling of cells (chiefly lymphocytes) with surface epithelium,
4. frequently with an indistinct or liquefactive basal cell region jagged or saw-toothed" rete pegs.
5. The latter are frequently indistinct.

In a study conducted by Fitzpatrick SG et al in 2014, among 7,806 patients with OLP, 85 developed SCC and among 125 patients with OLL, 4 developed SCC. The rate of malignant transformation was 1.09 percent for OLP and 3.2 percent in OLL.[15] The presence or absence of additional risk factors (specifically, tobacco or alcohol use) was reported in 79 cases: of these, 57 percent (n = 45) reported no history of tobacco or alcohol use, 22 percent (n = 17) reported history of or current tobacco and alcohol use, 14 percent (n = 11) reported history of or current tobacco use only, 5 percent (n = 4) reported history of or current alcohol use only and 3 percent (n = 2) specified no history of or current tobacco use but did not mention alcohol consumption.[15]

Giuliani M et al in 2018, reported that 92 of 6,559 patients developed oral squamous cell carcinoma, with an overall transformation rate of 1.40% (1.37% for OLP and 2.43% for OLL).[14] Considering the classic risk factors of OSCC (smoking and alcohol), many studies on OLP have reported the habit of smoking or alcohol use in the population studied, but few studies have taken these risk factors into account when calculating the transformation rate.[14]

However, there is no grading system proposed for cases of oral lichenoid dysplasia. In 2017, Susan Müller stated that grading of oral epithelial dysplasia should be based on the degree of dysplasia (mild, moderate, and severe) rather than using the term lichenoid dysplasia.[29] Hence, this term as a diagnosis should be discouraged, as it may create confusion leading to suboptimal treatment.[29]

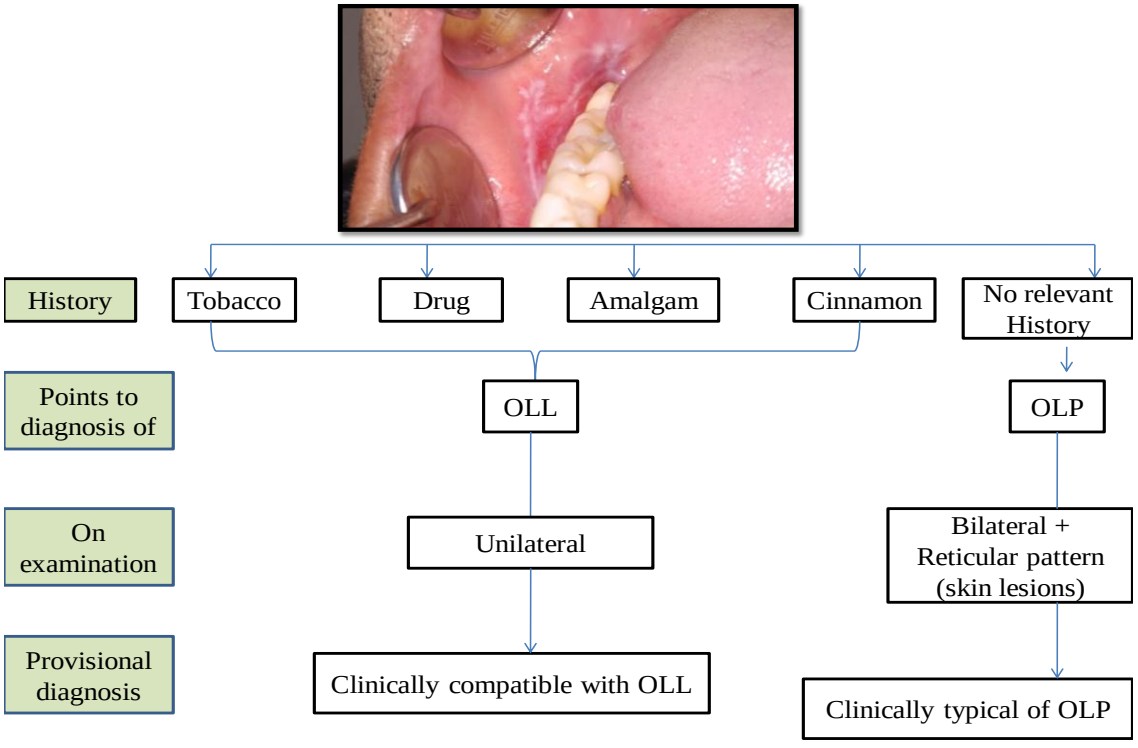
In 2003, Van der Meij and Van der Waal proposed a set of modified WHO diagnostic criteria in the diagnosis of OLP and OLL.[4] In 2016, Cheng at al proposed a set of criteria for the diagnosis of OLP.[6]

In our study, after evaluating the cases using the modified WHO criteria, 19/30 (63.3%) cases were OLP and 11/30 (36.6%) cases were OLL. While after evaluating the cases using the Cheng's proposed criteria, only 8/30 (26.6%) cases were OLP and 22/30 (73.3%) cases were OLL amongst which 2/22 cases were associated with drugs, 4/22 cases were associated with tobacco, 7/22 cases were associated with amalgam, 1/22 cases was associated with drugs and tobacco, 5/22 cases were associated with drugs and amalgam and 3/22 cases had no relevant history.

Table 3:-Specific histopathological features associated with types of OLL[18,27]

Lichenoid reactions induced by drugs	Extensive degeneration in the lower prickle cell layer promoting spongiotic vesicle formation. Perivascular cuffing of inflammatory cells.
Lichenoid reactions induced by amalgam	Normal stratification. Basal cell liquefaction may be absent. Lymphoid follicle formation.
Lichenoid reactions induced by cinnamon	Hyperorthokeratosis/parakeratosis. Munro-type neutrophilic abscess in superficial epithelium. Perivascular cuffing by plasmacytes.
Graft versus host disease	Epithelial maturation disturbances and basal squamatization. Perivascular cuffing of lymphocytes.
Betel-quid lichenoid lesions	Atrophic epithelium with hyperkeratosis. Liquefaction degeneration of the basal cell layer. Band like inflammatory cell infiltrate with lymphocytes and plasma cells in the juxta-epithelial region.

After comparing the results obtained from both the studies, the patients diagnosed with Oral Lichenoid lesions was higher using the criteria proposed by Cheng et al. OLL having a higher potential for malignant transformation, implies that the diagnosis of OLP and OLL should not be based exclusively on the clinical and histopathological features. A detailed diagnostic workup (Fig. 1) can draw a clear line of demarcation hence establishing optimal treatment protocol. Treatment of OLP comprises mainly the utilization of topical corticosteroids whereas OLL is treated by removal of the causal factor and a long term follow up is necessary, especially among tobacco habitués to monitor progression into dysplasia and/or malignancy.



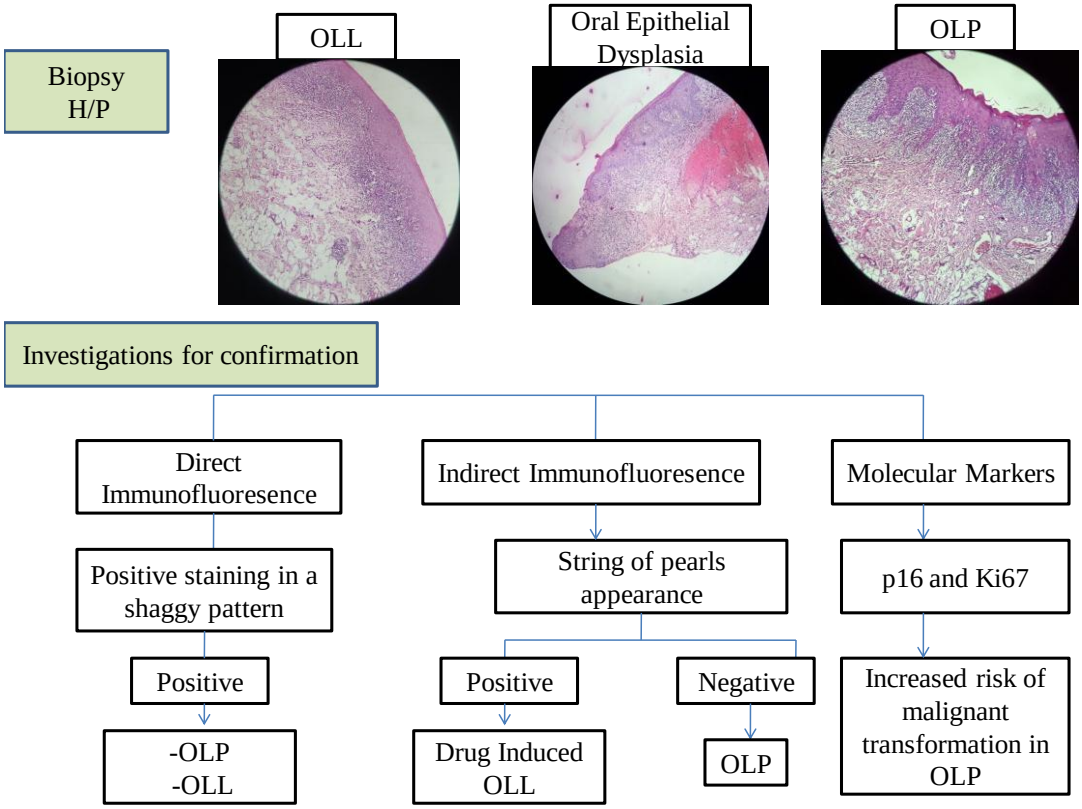


Figure 1:-Diagnostic Workup

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