

## Assessment of Serum Procalcitonin Level and Neutrophil /Lymphocyte Ratio in Patients with Lichen Planus

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### Abstract

**Background:** Lichen It is believed that dyslipidemia and diabetes mellitus have a tight relationship with the aetiology of papulosquamous chronic plaques, or lichen planus (LP). The goal of this study is to examine all aspects of LP patients' blood procalcitonin levels and neutrophil/lymphocyte ratios, including their epidemiology, pathophysiology, and clinical features. Conclusions: The neutrophil/lymphocyte ratio and serum procalcitonin levels show potential as diagnostic indicators for LP. Their strong associations with illness severity point to their possible use in measuring the severity and scope of this ailment.

Topics covered include lichen planus, serum procalcitonin, and neutrophil to lymphocyte ratio.

**Keywords:** Lichen Planus; Neutrophil /Lymphocyte Ratio; Serum Procalcitonin.

### 1.Introduction

Lichen planus is an autoimmune disorder that causes inflammation and wears down the skin, as well as the oral and vaginal mucosa, the scalp, and the nails. The six P's are used to characterise LP lesions (planar [flat-topped], purple, polygonal, pruritic, papules, plaques). When it first appears, it often affects the flexor surfaces of the legs, wrists, and forearms. Wickham striae, which are whitish and reticular, cover the lesions. It is possible to make a clinical diagnosis of classic LP [1].

Linear, annular, atrophic, hypertrophic, vesiculobullous, and erosive/ulcerative LP are some of the shapes that cutaneous LP may take [2].

The 116-amino acid protein known as procalcitonin (PCT) has a molecular weight of 13 kDa and is a precursor to calcitonin. Tripeptide calcitonin (with 32 amino acids), katalcain (with 21 amino acids), and an N-terminal fragment known as aminoprocaltinin (with 57 amino acids) are the three separate molecules that result from its cleavage in the neuroendocrine cells of the thyroid, lung, and pancreas [3].

No one knows for sure what PCT does physiologically. It implies that PCT is essential to the host response and the final result of sepsis, and that it mediates the inflammatory response in a way comparable to IL-6 and IL-8. It is plausible to assume that PCT may work similarly to TNF- $\alpha$ , with a positive impact when used in small amounts but a negative one when used in large numbers [4].

An indication of the body's overall inflammatory condition is the blood neutrophil-to-lymphocyte (N/L) ratio. Systemic

inflammation may be revealed using biochemical and hematologic indicators; however, these markers are often costly and usually fail to provide useful results [5].

The ratio of neutrophils to lymphocytes, or N/L, is a measure of systemic inflammation. When looking at systemic inflammation, the N/L ratio is a good indicator of many chronic diseases. An elevated inflammatory response is significantly associated with a more severe illness. By analysing the N/L ratio, we may learn about two distinct immunological mechanisms. Regarding neutrophils, which cause chronic inflammation, and lymphocytes, which show the regulatory route, in that order [6].

The goal of this review is to cover all the bases when it comes to what is currently known about the clinical features, pathophysiology, and epidemiology of LP and blood PCT levels and neutrophil/lymphocyte ratios.

### 2.Skin Condition

The skin, nails, hair, and mucous membranes are all impacted by lichen planus (LP), an inflammatory and immune-mediated chronic illness. In middle-aged adults, cutaneous LP (CLP) often manifests as tiny, itchy violaceous papules and affects the flexor surfaces of the limbs [7].

### 3.Lichen Planus Epidemiology

From 0.22% to 5% of the global population is thought to have LP. Neither standardised procedures nor established diagnostic criteria are present in the epidemiological research. The most prevalent subtype of OLP is asymptomatic and has a wide range of clinical presentations,

which contributes to the underdiagnosis of the condition [8]. 1.27% of the population in Sweden was affected, with 0.96% of males and 1.57% of women being affected after adjusting for age. In the UK, the prevalence of LP ranged from 0.032% to 0.037%. Men and women in their mid-thirties are the usual victims of LP. There seems to be no sexual preference, yet there have been reports of a little female preponderance (up to a ratio of 2:1). [9].

Surprisingly, the female-to-male ratio in the three biggest case series of childhood LP is 2:1 in a Canadian research, 1:1.5 in an Indian cohort, and 1:2 in a US sample. One possible explanation for this variation is because various research used different criteria for inclusion and exclusion [10].

Because autoimmune diseases typically affect females, the fact that boys are more likely to develop juvenile LP raises the possibility that other, as-yet-undiscovered pathways contribute to the disease's development. African Americans have a higher prevalence of childhood LP. [11].

#### 4.The Origins of Clear Skin

In the beginning, T-cells mostly settle in the dermal-epidermal junction and deeper layers of the epidermis. Along with an increase in the transport of mononuclear cells to the interface area, LP lesions show a considerable overexpression of chemokines associated to CCR5 and CXCR3. It seems that T-cell migration inside LP lesions is influenced by both self-recruiting mechanisms and processes that stimulate keratinocytes. On top of that, chemokines associated with CCR6 trigger Langerhans cell recruitment. Immune cells eliminate keratinocytes when activated T-cells trigger the Th1 response [8].

Lowered T-cell apoptosis and enhanced keratinocyte apoptosis are two major pathogenetic mechanisms of LP. By attaching to Fas on keratinocytes, activated cytotoxic T-cells may increase Fas ligand expression and trigger keratinocyte death in the suprabasal cell layer. The granzyme B/perforin pathway is another potential mechanism by which cytotoxic T-cells and NK cells promote cell death. More cases of this immunologic mechanism that causes autodestructive damage occur in OLP than in CLP. Apoptosis of basilar keratinocytes caused by the altered immunological response causes the basal layer to liquefy. Perhaps the disease's activity is reflected in these apoptotic alterations as well [12].

#### 5.Procalcitonin

The production of the biomarker procalcitonin (PCT) in response to bacterial infections allows for the discrimination of infectious process origin. It has a role in antibiotic stewardship since it may be used to advise optimal antibiotic treatment. The PCT has emerged as a novel and exciting biomarker for the early diagnosis of systemic bacterial infections in modern clinical practise [13].

#### 6.How Procalcitonin Works in the Body

Thyroid C cells initiate the production of pre-procalcitonin during normal homeostasis. Some time later, endopeptidases cleave a signal sequence of 25 amino acids off this peptide, converting it into PCT. After being converted by the enzyme prohormone convertase, the 32-amino acid hormone calcitonin is produced. It is responsible for regulating blood calcium levels. Serum PCT values below 0.05 ng/mL are typical under healthy circumstances. On the other hand, circulating endotoxins and cytokines including IL-6, TNF-alpha, and IL-1b may operate on different organs to boost PCT production by a ratio of 100 to 1000. [14].

Notably, PCT is not only synthesised in the thyroid gland but also in the liver, pancreas, kidneys, lungs, intestines, and leukocytes; in fact, when bacterial infection is not present, PCT production is reduced in these tissues. Another benefit of PCT assays is that they reveal which cytokines, including interferon (INF)-gamma, are secreted after a viral infection and which cause PCT to be down-regulated [15].

#### 7.The Importance of Procalcitonin in Clinical Practice

The sensitivity and efficiency required to treat patients quickly are unfortunately lacking in most first-line tests for identifying infection, including C-reactive protein and blood cultures. In order to help doctors pinpoint the cause of a systemic inflammatory response early, PCT serum tests were developed (e.g., bacterial versus non-bacterial). The development of antibiotic resistance and the overuse of antibiotics by patients when they are no longer necessary are both mitigated by early identification [16].

There is a favourable link between illness severity and higher PCT serum levels, which has proven clinical importance for PCT's predictive usefulness, particularly in septic patients. The question of whether PCT assays are cost-effective remains open, despite the fact that they have shown significant potential. The lack of sufficient criteria for when these tests

should and should not be done has led to their veruse, according to recent study [17].

### 8.The ratio of neutrophils to lymphocytes

One biomarker that combines the two sides of the immune system—the innate immune response, which is primarily caused by neutrophils, and the adaptive immune response, which is supported by lymphocytes—is the neutrophil-to-lymphocyte ratio (NLR), which is simply determined as the ratio of the neutrophil and lymphocyte counts in peripheral blood [18].

### 9.The neutrophil lymphocytotic ratio (N/L) and its clinical features

Neutrophils play an important role in the host immune response via chemotaxis, phagocytosis, ROS release, granular protein creation and release, and cytokine production and release, among other processes. In addition to being the primary effector cells in the systemic inflammatory response, neutrophils have a crucial regulatory function in adaptive immunity (SIRS). Neutrophils play a role in innate immunity regulation by attracting, activating, and programming other immune cells. They do this by secreting a variety of cytokines and chemokines that can enhance the recruitment and effector functions of other immune cells, including DCs, B cells, NK cells, CD4, CD8,  $\gamma\delta$  T cells, and mesenchymal stem cells[19].Instances where there is tissue damage that triggers SIRS can lead to an increased neutrophil count and, by extension, an elevated NLR. These include bacterial or fungal infections, acute strokes, myocardial infarctions, atherosclerosis, severe traumas, cancer, post-operative complications, and severe trauma [18]. This is due to the fact that neutrophils and other inflammatory cells promote a proinflammatory state that characterises the early hyperdynamic phase of infection. Suppression of neutrophil apoptosis, a process that enhances the innate response via neutrophil-mediated death, is linked to SIRS. An increase in neutrophils and a decrease in lymphocytes are hence common characteristics of NLR [20].

Also, NLR has the potential to foretell death rates in the population at large. Cardiovascular illness (HR 1.17, 95 percent CI 1.06-1.29, per quartile of NLR), chronic lower respiratory diseases (1.24, 1.04-1.47), influenza/pneumonia (1.26, 1.03-1.54), renal disorders (1.62, 1.21-2.17), and overall mortality were all substantially higher in NLR. In contrast, we did not find any statistically significant correlations between NLR and death from cancer, CVD, accidents, or diabetes at all. Further, the Rotterdam trial demonstrated that

NLR levels were linked to a higher risk of all-cause death (HR 1.64; 95 percent CI 1.44-1.86) on their own [21].

Compared to other laboratory indicators such as white blood cell count, bacteremia, C-reactive protein, and CRP, NLR may serve as an early indicator of acute stress due to its rapid rise (<6 h) after acute physiological stress [22].

On the other hand, adaptive immunity relies heavily on lymphocytes of various types, including B cells, T cells that are CD4-positive, CD4/CD8-negative, or CD8-positive, and natural killer T cells. These cells provide an antigen-specific response that is controlled by the major histocompatibility complex (MHC) class I. In addition to SIRS, lymphocyte activity is implicated in the host's response to tumour cells, viruses, and atopy. As a reflection of a healthy immune system, a lower NLR is often linked to better prognostic indicators across all fields [23].

The delicate balance between these two parts of the immune response is susceptible to disturbance from a wide variety of physiological and pathological sources. Endogenous cortisol and catecholamines, for instance, might have a significant role in NLR. The numbers of neutrophils and lymphocytes are known to rise and fall, respectively, in response to elevated cortisol levels. There is a possibility of leukocytosis and lymphopenia due to endogenous catecholamines such as epinephrine. Involvement of cytokines and other hormones is also probable. Therefore, in order to give NLR change the right clinical significance, confounders need to be considered [20].

Ideas for the future and suggestions for improvement:

Future plans and recommendations for bettering the treatment of psoriatic pruritus have great potential. To validate these findings, more study is required with larger samples and cooperation across several centres. Furthermore, studies that track LP patients over time are important for tracking changes in neutrophil/lymphocyte ratios and serum PCT levels. as time progresses, more information about the disease's dynamics and the markers' stability will become apparent. Future research may evaluate the relationship between these serum indicators and the effect of LP on patients' psychosocial or quality of life status, as well as the underlying processes that link increased serum PCT and changed neutrophil/lymphocyte ratios to the pathophysiology of LP.

## 10. Conclusions

The use of serum PCT levels and neutrophil/lymphocyte ratios as indicators of LP shows potential. Their strong associations with illness severity point to their possible use in measuring the severity and scope of this ailment.

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