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Original Article

C-reactive Protein and Erythrocyte Sedimentation Rate Levels in Chronic Low Back Pain Patients

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ABSTRACT

Introduction: Lumbar disc degenerative disease, intervertebral disc herniation, lumbar stenosis, and arthritis are usual causes of chronic nonspecific low back pain. Patients with acute lumbosciatic pain usually have elevated levels of inflammatory markers such as highly sensitive C-reactive protein. However, studies that analyze the levels of Erythrocyte sedimentation rate and C-reactive proteins in patients with chronic low back pain are insufficient. Hence, this study aimed to assess the value of these markers for the determination of inflammation in patients with chronic low back pain.

Method: A cross-sectional, hospital-based, observational study was conducted after receiving ethical approval from the Institutional Review Committee. The study started on Feb 20, 2023, and lasted till Aug 10, 2023. Statistical analysis of the level of inflammatory markers was done by evaluating their mean values.

Results: 120 patients who presented with nonspecific chronic low back pain were evaluated clinical-radiologically and sent for blood investigations. The mean erythrocyte sedimentation rate was 11.54 mm/h, and the mean C-reactive protein level was 5.90 mg/L. Blood sedimentation rate was found to have a significant positive correlation with increasing age and female sex.

Conclusion: Chronic nonspecific low back pain does not elicit a systemic inflammatory response, as evidenced by normal ESR and CRP levels.

Keywords: Blood Sedimentation; C-Reactive Protein; Low back pain

INTRODUCTION

Pain, rigidity, or tension occurring between the gluteal line and the 12th rib posteriorly and lasting for more than twelve weeks is chronic low back pain.[1,2] In more than 85% of patients, the precise etiology of low back pain can't be identified; it can be due to myofascial spasm or spinal stenosis or can originate in the intervertebral discs, facet joints, or sacroiliac joints.[2] In these conditions, inflammatory mediators such as interleukin 6 are assumed to cause local inflammation and play a major role in the propagation of pain.[3] These mediators in high concentration can cause a systemic inflammatory response, which can be identified by measuring the C reactive protein (CRP) level and the Erythrocyte sedimentation rate (ESR).[3]

METHODS

A cross-sectional, hospital-based, observational study was conducted from Feb 20, 2023, to Aug 10, 2023 in KIST Medical College. Before starting the study, ethical approval was taken from the Institutional Review Committee (Ref no 2079/80/85). Patients who were 18 years and older and of both sexes and suffering from nonspecific chronic low back pain were included in the study. Firstly, a proper history was taken to look for constitutional symptoms like fever, significant weight loss, anorexia, nocturnal pain, and night sweats. Then, a local examination was performed to look for raised temperature, palpable swellings, discharges, etc. After that, an X-ray of the lumbosacral spine - anteroposterior and lateral views was taken to look for any bony destructions, deformities, intervertebral space reductions, and paraspinal collections.

Non-consenting patients and patients with red flags like infection, malignancy, trauma, and neurologic compromise were excluded. The participants were first explained in detail about the study. They were then assured of confidentiality, and after that, their informed written consent was taken. The sampling method was probability sampling, where a coin toss was performed, and the patients with a 'head' result on the coin toss were selected.

According to a paper by Sharma et al. published in 2019, the prevalence of low back pain among all chronic pain

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conditions in Nepal is 13%.[4] Based on this finding, the following formula was used to calculate the sample size:
$N= z^2 \times p (1-p)/ e^2$ where
N = sample size
Z=1.96 at a 95% confidence interval
P= prevalence of low back pain among the Nepalese population (13%)
E= margin of error, 6%

N= 1.962 x 0.13 (1-0.13) / 0.062
i.e. N = 120

The sample size was calculated to be 120. Hence, we included 120 participants in the study.
After the 120 eligible participants signed the written consent, they were included in the study. After that, a blood investigation evaluating the levels of ESR and CRP was sent. After the reports were received, proforma was filled, and the mean ESR and CRP levels of the study population were calculated to detect if they were elevated or were in the normal range for these tests.
Data entry was done using Microsoft Excel 2010. Statistical analysis was done by using Statistical Package for Social Sciences (SPSS) version 16 for Windows. Statistical analysis of the levels of ESR and CRP was done, taking into account the mean values.

RESULTS

One hundred twenty patients with complaints of nonspecific chronic low back pain presenting in the orthopedics outpatient clinic of Kist Medical College were first examined clinical-radiologically to rule out red flag signs like infection, malignancy, trauma, and neurologic compromise, and then their blood samples were sent to find out the values of ESR and CRP.
The age of the participants in our study ranged from 20 years to 65 years, with a mean age of 44 years. There were 50 male patients and 70 female patients. The ESR level among all participants ranged from a minimum of 3 mm/h to a maximum of 40 mm/h with a mean value of 11.54 mm/h. Similarly, CRP values ranged from 5 mg/L to 15.90 mg/L, with an average of 5.90 mg/L. The sex-wise distribution of these inflammatory markers is depicted in Table 1.

Table 1: Sex-wise distribution of inflammatory markers

	Number	Minimum	Maximum	Mean	Standard Deviation
ESR in male	50	3 mm/h	14 mm/h	7.20 mm/h	3.12
ESR in female	70	3 mm/h	40 mm/h	14.64 mm/h	11.43
CRP in male	50	5 mg/L	15.90 mg/L	6.19 mg/L	3.28
CRP in female	70	5 mg/L	11.50 mg/L	5.84 mg/L	1.80

ESR was found to have a significant positive correlation with age (p-value: 0.001), as shown in Table 2, while CRP had no such strong correlation (p-value: 0.063) (Table 3).

Table 2: Correlation between Age and ESR levels

		Age	ESR
Age	Pearson Correlation	1	0.30
	Significance (2-tailed)		0.001
	Number	120	120
ESR	Pearson Correlation	0.30	1
	Significance (2-tailed)	0.001	
	Number	120	120

Table 3: Correlation between Age and CRP levels

		Age	ESR
Age	Pearson Correlation	1	0.17
	Significance (2-tailed)		0.063
	Number	120	120
ESR	Pearson Correlation	0.17	1
	Significance (2-tailed)	0.063	
	Number	120	120

DISCUSSION

The mean ESR (11.54 mm/h) and mean CRP (5.9 mg/L) values in our study were well within the normal range for these inflammatory markers.[5,6] This finding indicates that chronic low back pain elicits very little inflammatory response. ESR was seen to have a significant positive correlation with increasing age, a finding consistent with a paper by Brigden.[7] This might be because of higher disease prevalence in the elderly, which increases the fibrinogen levels.
Similar to our study, Park and Lee evaluated 273 patients with nonspecific chronic low back pain and calculated the mean ESR to be 18.8 mm/h and mean CRP to be 1.1 mg/L. Both values were within the normal range; hence, they concluded that no significant inflammatory process occurs in a patient with nonspecific chronic low back pain, and if there is elevation of these markers, they recommended the clinicians look meticulously for other conditions that cause systemic inflammation.[3]
In contrast to our study, a systemic review by Berg et al. and studies by Le Gars et al., Sugimori et al., and Ackerman and Zhang reported higher CRP and hs-CRP levels in patients with nonspecific low back pain.[8–11] Berg et al. analyzed ten studies and concluded that CRP levels were elevated in those patients who had increased severity of back pain.[8] Le Gars et al. found the ultrasensitive CRP levels to be significantly elevated in 35 patients with sciatica as compared to normal controls.[9] Similarly, Sugimori et al. found a statistically significant increase in hs-CRP levels in 48 patients with lumbar disc herniation as compared to 53 controls and suggested that there is a systemic inflammatory response in these patients with nerve root impingement.[10] Ackerman and Zhang also evaluated 60 male patients and found out that the number of patients with elevated hs-CRP levels increased with the increased degree of prolapse.[11]

All these studies support the fact that inflammatory markers like CRP and hs-CRP levels are increased in patients with more severe pain, a finding reiterated by the study of Sturmer et al.[12]. They observed a strong association between pain severity and hs-CRP levels in patients with acute sciatic pain but not in those with chronic low back pain, hence concluded that pain severity in patients with acute sciatic pain may be more closely linked to inflammatory changes than in patients with chronic low back pain in whom pain severity may be due to weight and psychological issues.[12]

The basic theme of our study was to look for inflammatory responses in chronic nonspecific low back pain patients, but there were certain limitations to our study. Our study was conducted in a single center, and we could not evaluate other inflammatory markers like interleukin 6 (IL-6) and tumor necrosis factor-alpha (TNF-α). We just ruled out red flag conditions based on clinical acumen and X-rays and did not look for the exact diagnosis. We also did not consider the severity of pain in these patients.

CONCLUSION

Chronic, nonspecific low back pain does not elicit a systemic inflammatory response, as evidenced by normal ESR and CRP levels in our study. If these markers are raised in any patient with chronic low back pain, after ruling out red flag conditions, the clinicians should focus on looking for other diseases that cause systemic inflammatory reactions.

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