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Evaluation Of HscrpIn Schizophrenic Patients

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Abstract
Background*:*
Increased Levels Of Inflammatory Markers Have Been Reported In Schizophrenia, But Few Studies Have Examined Levels Of High Sensitivity C-Reactive Protein (Crp), A Non-Specific Inflammatory Marker. The Aim Of This Study Was To Assess The Level Of Inflammatory Markers High Sensitive C-Reactive Protein (Hscrp) In Patients With Schizophrenia.
Methods*:*
Levels Of High Sensitivity Crp Were Measured In Individuals With Schizophrenia, And Non-Psychiatric Controls.
Results*:*
The Sample Consisted Of 100 Individuals: 50 With Schizophrenia, 50 Normal Patients As Control. The Levels Of Crp In The Schizophrenia Group, Were Significantly Increased Compared To Controls.
Conclusions:
Individuals With Schizophrenia May Be At Risk For The Adverse Health Consequences Associated With Elevated Crp In The Overall Population. Trials Of Interventions Directed At Lowering The Level Of Crp And Other Inflammatory Markers Are Indicated.
Key Words-*Schizophrenia, Hscrp.*

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I. INTRODUCTION

Schizophrenia is a psychiatric illness characterized by a variety of psychotic symptom, including delusions and hallucinations, altered emotional reactivity and disorganized behaviour.

Over the years,Schizophrenia is speculated to be associated with immune or inflammatory reactions mediated by cytokines. It is proposed that chronic inflammation might damage the micro-vascular system of brain and hamper cerebral blood flow. Schizophrenia is one of the most severe and debilitating of the psychiatric disorders. The disorder comprises delusions, hallucinations disorganized speech, bizarre behaviour, and negative symptoms. Symptoms typically emerge in late adolescence or early adulthood. Schizophrenia affects about 1% of the general population. Schizophrenia is considered a lifelong condition with no known cure. Scientific evidence suggests that an increase of stress hormone like norepinephrinemay activate the inflammatory arm of the immune system and trigger the expression of genes that cause chronic, low-grade inflammation. The complex interactions between altered immune signalling and the brain were postulated in the development of schizophrenia. This allegation came from the striking findings of elaborative studies that showed increased pro-inflammatory cytokine and decreased anti-inflammatory cytokine plasma levels, up-regulated inflammatory gene expression, evidence of inflammation in certain cortical areas in post-mortem brains, a higher prevalence of autoimmune disorder comorbidity, and symptomatologic improvement with anti-inflammatory drugs, in schizophrenia. CRP is a well-documented circulating marker for systemic inflammation, and albumin is a negative acute-phase protein, serum levels of which are down-regulated in response to an inflammatory state. Unlike proinflammatory cytokines, both CRP and albumin have longer half-lives and are easier to measure in a blood screening. Increased plasma CRP, and decreased serum albumin levels have been shown in patients with schizophrenia in comparison with healthy controls.Importantly, many studies including our findings revealed the alteration of the immune system in schizophrenia (Boyajyan et al., 2008, Radulescu,

2009, Monji et al., 2009, Bilbo and Schwarz, 2009, Singh et al., 2009, Singh et al., 2011). This has stimulated researchers to study the role of a well known inflammatory marker, C-reactive protein (CRP) in etiopathology of schizophrenia and its potentiality as inflammatory marker for schizophrenia. Therefore, the aim of this article is to evaluate the values of hsCRP in schizophrenia patients and establish a relationship , if any.

II. METHODS

This cross-sectional retrospective study consecutively included data of patients who were admitted to the outpatient or the indoor patient in J.L.N Medical College, Ajmer for Psychiatry, Neurology, and Neurosurgery within a six-month period between March 2022 and March 2023, and who were diagnosed as having schizophrenia based on the International Classification of Diseases-10 (ICD-10). Inclusion criteria for patients were as follows: 1) age 18–65 years; 2) being admitted or hospitalized with a primary diagnosis of schizophrenia (ICD-10 codes between F20.0–F20.9). Exclusion criteria for schizophrenia patients were as follows: presence of a comorbid psychiatric disorder, presence of a systemic disease that may influence inflammatory status such as diabetes mellitus, hepatic or renal failure, hypertension, acute infection, acute or chronic immuno-inflammatory disease or pregnancy, obesity or being underweight (body-mass index >29.9 kg/m^2 or <18.5 kg/m^2, respectively), heavy smoking (20>cigarettes per day), being under an anti-inflammatory or immunosuppressive medication or psychotropic medication other than antipsychotics, documented laboratory findings of liver or renal pathology, nutritional deficiency of vitamin B12 or folate and iron-deficiency anemia, and not having a laboratory screening at the admission. A comparison group of healthy controls consisted of 50 individuals who visited our outpatient unit for purposes of pre-employment health check-ups or employee medical examinations, coded with ICD-10 Z00.00 (encounter for general adult medical examination without abnormal findings), aged 18–65 years, and without any previous psychiatric or medical diagnosis coded and any current medication.After the eligibility evaluation of the data for inclusion, the patients' hard-copy files, which were provided with patients' hospital registration number, were reviewed. Preliminarily, data of 50 patients who attended the outpatient unit or were admitted to the inpatient clinic within the designated time frame were screened. Remission was determined by clinical examination by a senior psychiatrist during outpatient follow-up or at predischarge evaluation, and systematic operational remission criteria based on the specific items of. Age, sex, and laboratory results of all individuals and clinical variables including illness duration, number of hospital stays, and total duration of hospital stays of patients were recorded anonymously. Because the data of the individuals were retrieved anonymously and file review was made retrospectively by the researchers, patient informed consent is not applicable. All procedures performed in this retrospective file review study involving human participants were in accordance with the ethical standards.hsCRP measurements were performed using Chemiluminescence immunoassay methods withMaglumi 800.All statistical analyses were performed using the SPSS. A chi-square test and an independent sample t-test were used for comparisons of categorical and parametric variables between the patient and the control groups. For the comparison of inflammatory markers amongst the schizophreniaand control group, one-way analysis of variance (ANOVA) was used. A p-value of less than 0.05 was considered statistically significant.

III. RESULTS

Fifty-eight percent of patients with schizophrenia had elevated hsCRP. The mean value of hsCRP in patients with schizophrenia and controls was 2.87 ± 5.6 and 0.67 ± 0.6 mg/L respectively. hsCRP level was statistically significantly associated ($p<0.05$) with having a diagnosis of schizophrenia. There were no associations between hsCRP and age of onset, duration of current episode, and smoking status.

Sl. No	Patients with schizophrenia	Controls
Mean value of hsCRP(in mg/L)	2.87 ± 5.6	0.67 ± 0.6

IV. DISCUSSION

The main finding of the study is that significantly higher levels of both hsCRP were observed in patients with schizophrenia compared with the control group. There is evidence of increase in the incidence and prevalence of some inflammatory diseases in the developing world, which may increase the significance of inflammation in neuropsychiatric syndromes.The result of the present study concurs with studies from Western countries that consistently indicate that patients with schizophrenia have high serum levels of hsCRP. Elevated inflammatory markers in patients with schizophrenia have been reported in case control studies and treatment studies. This is also found in people with both acute chronic and treatment-resistant illnesses. Because of the consistency of this finding, neuroinflammation has been linked with the causation of schizophrenia and other mental disorders. However, such studies are rarein low- and middle-income countries where the majority of the population of the world lives. We believe that this study contributes to this particular knowledge gap and the broader issue of lack of such studies even in the general population. hsCRP appears to be an important

inflammatory marker in this particular setting although additional confirmatory studies would be needed. The pathophysiology of schizophrenia has been linked with chronic inflammation, which stimulate inflammatory markers like hsCRP. hsCRP has important roles in the inflammatory processes and hsCRPhas been widely considered as a state marker along with other cytokines like TNF alpha. CRP is an acute phase protein and produced by hepatocytes when stimulated by inflammatory markers including IL-6. Under normal conditions, CRP does not cross the blood-brain barrier. Increasing serum level of CRP may increase the permeability of blood-brain barrier by affecting the function of tight junction which facilities the entry of pro-inflammatory cytokines and CRP itself into the central nervous system. This would support the potential role of CRP in the pathophysiology of schizophrenia,

This study compares the inflammatory markers of patients with schizophrenia with a control group. The result suggests that there is a higher level of hsCRPin patients with schizophrenia compared to their control groups..

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