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Original Article

Clinical Severity of Guillain-Barre Syndrome in Relation to Inflammatory Markers and Nerve-Conduction Studies

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ABSTRACT

Introduction: Guillain-Barre Syndrome (GBS)is a rare but acute demyelinating polyneuropathy and constitute a neurologic emergency. It is autoimmune in origin causing significant morbidity and 2-12% mortality globally. Incidence of GBS is 1.1-1.8 cases in 100,000 per year. Approximately 10-15% of patients require assistance with long-term residual disability. The aim of this study is to correlate the severity of GBS with inflammatory markers like ESR, CRP level and findings of nerve conduction studies.

Methods: It was a hospital based cross-sectional study conducted in the department of neurology in collaboration with dept. of physiology in VIMSAR, Burla during the period from June 2022 to December 2024 only after getting institutional approval. A total of 61 (both male & female) clinically diagnosed Guillain-Barre syndrome patients according to Asbury and Cornblath (1990) were recruited after fulfilling inclusion criteria. In all patients, general examination, neurological examination, nerve-conduction studies (both motor & sensory) as well as serum CRP level and ESR estimation were done.

Results: We observed that raised CRP level > 6mg/dl, raised ESR, absent motor & sensory responses were positively correlated with clinical severity of GBS patients (disability score >4 by Hughes disability scale). The result was statistically significant ($p > 0.05$).

Conclusion: The results of this study reflect the impact of inflammatory responses on clinical severity of the disease and hence prognosis. So, it can be used as prognostic markers for risk assessment and help in therapeutic decision making.

Keywords*: CRP, ESR, nerve conduction study, GBS*

INTRODUCTION-

Guillain-Barre Syndrome (GBS) is an acute, immune-mediated polyradiculoneuropathy characterized by rapidly progressive, symmetrical limb weakness, areflexia, and varying degrees of sensory and autonomic dysfunction. Since the global eradication of Poliomyelitis in many parts of the world, GBS has emerged as the most common cause of acute flaccid paralysis across all age groups (Willison et al, 2016). [1]

The global incidence of GBS ranges from 1.1 to 1.8 per 100,000 population per year, with reported mortality between 3–7%. In India, the incidence varies from 0.4 to 4.0 per 100,000 population annually (Anita Mcgrogan et al,2009). [2]. Despite advances in intensive care and immunotherapy, approximately 20–30% of patients experience severe disability during the acute phase, and 10–15% are left with long-term residual disabilities. **[3].** Also it show high inflammatory response resulting in an increase in CRP and ESR (Vaishnavi et al, 2014). [4]

GBS is primarily autoimmune in origin and is frequently preceded by respiratory or gastrointestinal infections. Molecular mimicry between microbial antigens and peripheral nerve components triggers an aberrant immune response directed

against myelin or axonal membranes. This immune-mediated inflammatory cascade results in demyelination, axonal degeneration, or both, depending on the subtype of GBS. The two major electrophysiological subtypes include acute inflammatory demyelinating polyneuropathy (AIDP) and acute motor axonal neuropathy (AMAN), each demonstrating distinct pathological and prognostic characteristics.

Inflammation plays a pivotal role in the pathogenesis and progression of GBS. Elevated inflammatory markers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) reflect systemic immune activation. Increased levels of these markers have been associated with heightened inflammatory activity and may correlate with disease severity and progression. CRP, an acute-phase reactant synthesized by the liver in response to pro-inflammatory cytokines such as interleukin-6 (IL-6), serves as a sensitive indicator of systemic inflammation. Similarly, ESR is a nonspecific marker that reflects the presence of inflammatory proteins in plasma. Previous studies have suggested that elevated CRP and ESR levels may be associated with more severe clinical presentations and poorer outcomes in GBS patients. Recent advances in medical research have illuminated the role of inflammatory markers and nerve-conduction studies in the diagnosis and monitoring of GBS. Elevated inflammatory markers, such as C-reactive protein (CRP) and interleukin-6 (IL-6), are often associated with systemic inflammation and may reflect the extent of the autoimmune response driving the neuropathy.

Conversely, nerve-conduction studies (NCS) constitute a cornerstone in the diagnostic evaluation of GBS and are essential for determining the subtype and severity of nerve involvement. These electrophysiological assessments measure parameters such as conduction velocity, distal latency, amplitude of compound muscle action potentials (CMAP), and sensory nerve action potentials (SNAP). Findings may reveal demyelinating features—such as prolonged distal latencies and slowed conduction velocities—or axonal degeneration characterized by reduced amplitude or absent motor and sensory responses. Furthermore, the preferential sparing of sural sensory nerve action potential (SNAP) is seen in almost half of GBS patients.[5,6,7] The extent of abnormalities detected on NCS often correlates with clinical severity, functional impairment providing valuable insights into the extent of nerve involvement and aiding in the prediction of disease progression.

Currently, there is no single definitive biomarker for GBS, and diagnosis relies on a combination of clinical evaluation, cerebrospinal fluid (CSF) analysis demonstrating albuminocytological dissociation, and electrophysiological studies. Given the variability in clinical course—from mild weakness to respiratory failure requiring mechanical ventilation—early identification of prognostic indicators is crucial for risk stratification and therapeutic decision-making. Therefore, this research aims to evaluate the relationship between clinical severity of GBS and inflammatory markers (CRP and ESR), along with nerve-conduction study findings. By correlating biochemical and electrophysiological parameters with disability grading, this research seeks to improve prognostic assessment and contribute to more targeted and timely management strategies in patients with GBS.

AIM & OBJECTIVES: -

AIM: The aim of this study is to evaluate the clinical severity of Guillain–Barré Syndrome (GBS) in relation to serum inflammatory markers (CRP and ESR) and findings from nerve conduction studies.

OBJECTIVES:

1. To measure serum inflammatory markers, specifically **C-reactive protein (CRP)** and **erythrocyte sedimentation rate (ESR)**, in patients diagnosed with GBS.
2. To assess **motor and sensory nerve conduction study (NCS) parameters** in these patients.
3. To determine the **clinical severity of GBS** using the Hughes disability scale.
4. To analyse the **correlation between inflammatory marker levels and NCS findings with the clinical severity** of GBS.

MATERIALS & METHODS-

This was a cross-sectional study carried out in the Department of Neurology, in collaboration with the Department of Physiology and the Department of Medicine at VIMSAR, Burla. The study was conducted from June 2022 to December 2024. The study was conducted after obtaining approval from the Institutional Ethics Committee, and written informed consent was obtained from each participant prior to inclusion in the study. A total of 61 patients of both sexes who were clinically diagnosed with Guillain–Barré Syndrome (GBS) were included in the study. The sample size was calculated using the Rosoft sample size calculator.

Patients presenting with progressive weakness of both limbs, relative symmetry of symptoms, decreased or absent tendon reflexes, sensory symptoms or signs, and bilateral cranial nerve involvement (mostly facial muscle weakness) were included in the study according to the diagnostic criteria described as per as per Asbury and Cornblath (1990) [8] .

Patients were excluded if they had other neurological illnesses that could affect nerve conduction studies (such as diabetes mellitus, drug- or toxin-induced neuropathy), pregnant women, patients with tumors or those receiving immunomodulatory therapy, patients with systemic failure (respiratory, renal, hepatic, or cardiac), and patients with severe psychiatric illness. After inclusion, all patients underwent laboratory and electrophysiological evaluation. Serum C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were measured in each patient. Nerve conduction studies (NCS), including both

motor and sensory nerve conduction as well as F-wave study of the median nerve, were performed. The study tools used included the turbidimetry method for estimation of CRP, the Westergren method for ESR measurement, and the Medicaid Neurostim system for nerve conduction studies. Clinical severity of GBS was assessed using the Hughes and Rees disability scale.

CLINICAL SEVERITY- (Hughes & Rees scale (1997) [9]
0. healthy
1. minor symptoms, capable of running
2. able to walk without assistance, unable to run
3. able to walk with help
4. Chair bound/bedridden
5.requiring ventilation for at least part of the day
6. Dead

All the relevant DATA were collected in a predesigned proforma. Statistical analysis was done by using SPSS software. ANOVA test was applied and p-value <0.05 was considered significant

RESULTS:
In the present study, total number of patients included were sixty (60) out of which 41 were male and 19 were female belonging to the age group 15 to 45 years. The table-1 shows the demographic data of all the study participants. In this study, we also observed that 34 patients (56.6%) presented with URTI as the most common predisposing factor. Also all the participants presented with neurological symptoms (both sensory & motor). However only 6 patients (10% cases) required mechanical ventilation.
Table-1

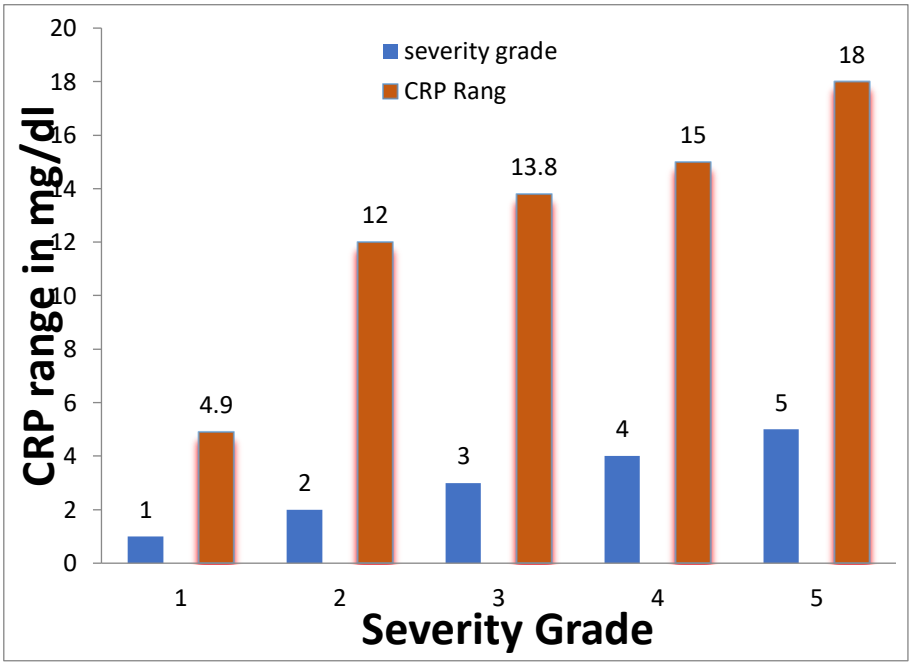
Demographic Data & Clinical presentation of GBS patients		
Variables	**No. (n=60)**	**Mean/ Percentage**
Age	15-45 Yr (Range)	**32.55 (±7.96)**
Sex M	41	**68.3%**
F	19	**31.6%**
Preceding Infection		
URTI	34	**56.6%**
GIT	21	35%
No	5	8.3%
Clinical presentation		
Motor symptoms	**60**	**100%**
Sensory symptoms (B/L)	**60**	**100%**
Cranial nerve involvement		
Facial	37	61.6%
Bulbar	12	23.3%
Autonomic symptoms	13	21.66%
Mechanical ventilation	**6**	**10%**

In this study, all the study participants were categorized into 5 grades based on clinical severity as per the Hughes & Rees scale. Out of 60 participants, 4 patients belonged to grade-1, 9 patients grade-2, 17 patients grade-3, 25 patients grade-4 and only 5 patients belonged to grade-5.

When we compared serum CRP level in different grades of patients as shown in figure -1, we found that the level was significantly raised in grade 4 and grade 5 group of patients that is 15mg/dl and 18mg/dl respectively ($p < 0.001$). However, when we compared ESR level with clinical severity grades as shown in figure-2, we observed that grade -4 patients had high ESR level i.e 31.33mm in 1st hour which was significant ($p < 0.001$) .

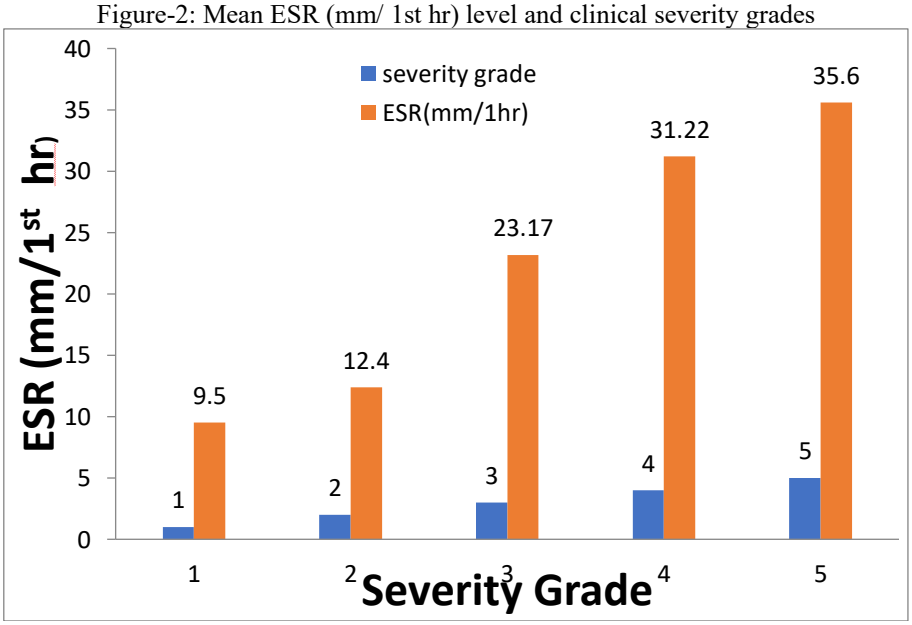
Figure-1: Mean serum CRP level (mg/dl) and clinical severity grades

Grade -1 (n=4)	Grade -2 (n=9)	Grade-3 (n=17)	Grade -4 (n=25)	Grade-5 (n=5)



Grade 4	15	p<0.001
Grade 5	18	P<0.001

Figure-2: Mean ESR (mm/ 1st hr) level and clinical severity grades



Grade 4	31.22	p<0.001

When motor nerve conduction study was performed and its result was compared with CRP level as shown in figure-3 and figure-4, raised CRP level was positively associated with nerve conduction abnormalities in median nerve and common peroneal nerve of GBS patients respectively. Also 83.34% (n=50) cases showed F-wave abnormality.

Figure-3: CRP level and Results of Motor NCS (Median nerve) in patients with GBS

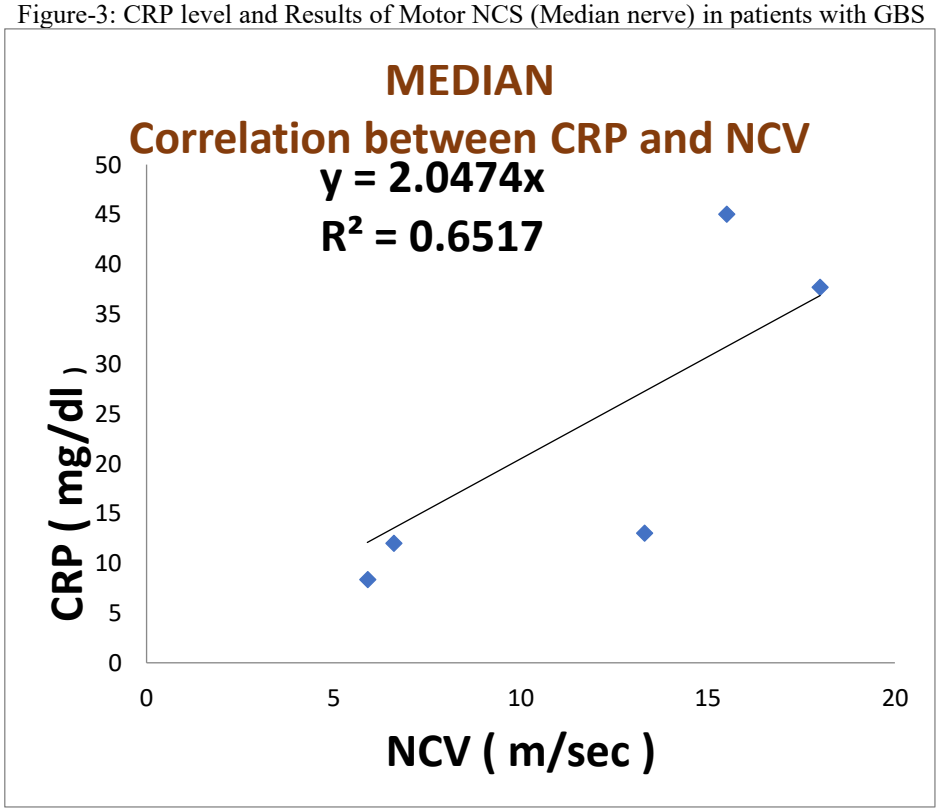
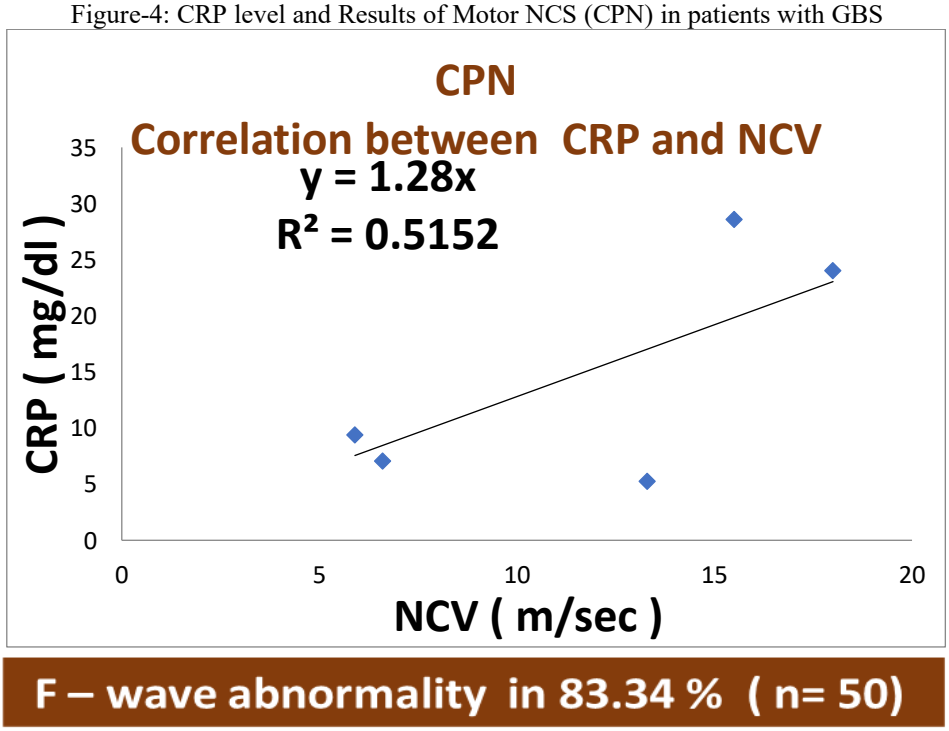


Figure-4: CRP level and Results of Motor NCS (CPN) in patients with GBS



In this study when we corelated the sensory nerve conduction study with CRP level, we observed that the result of sensory nerve conduction abnormalities in median nerve was positively associated with raised CRP level as shown in figure-5. However the sural nerve conduction study was negatively associated with raised CRP level as shown in figure-6. Sural sparing was observed in 70% (n= 42) cases.

Figure-5: CRP level and Results of Sensory NCS (median nerve) in patients with GBS

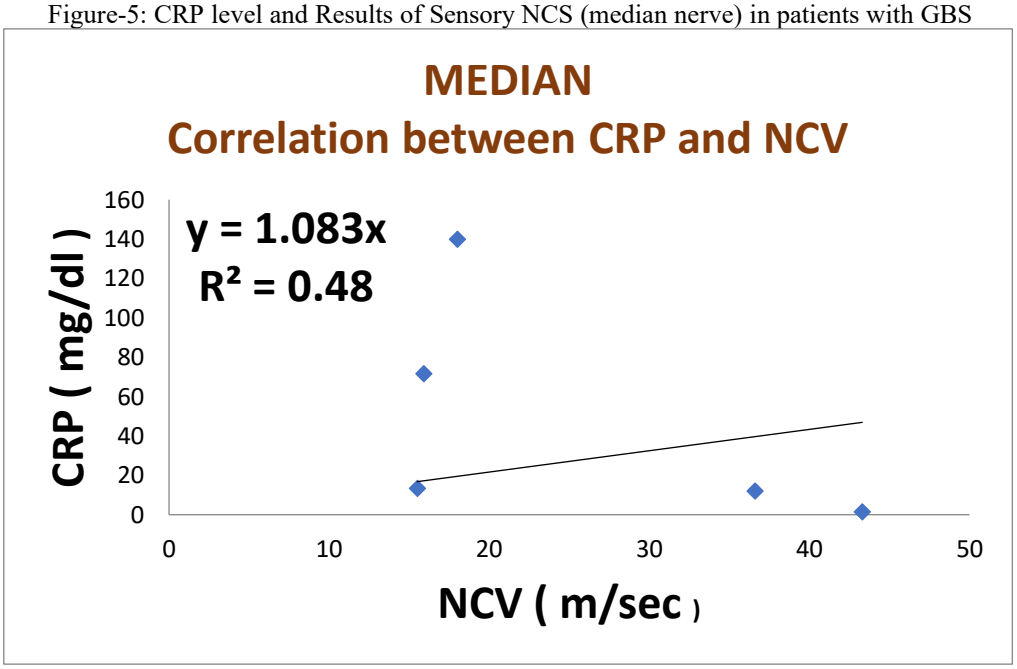
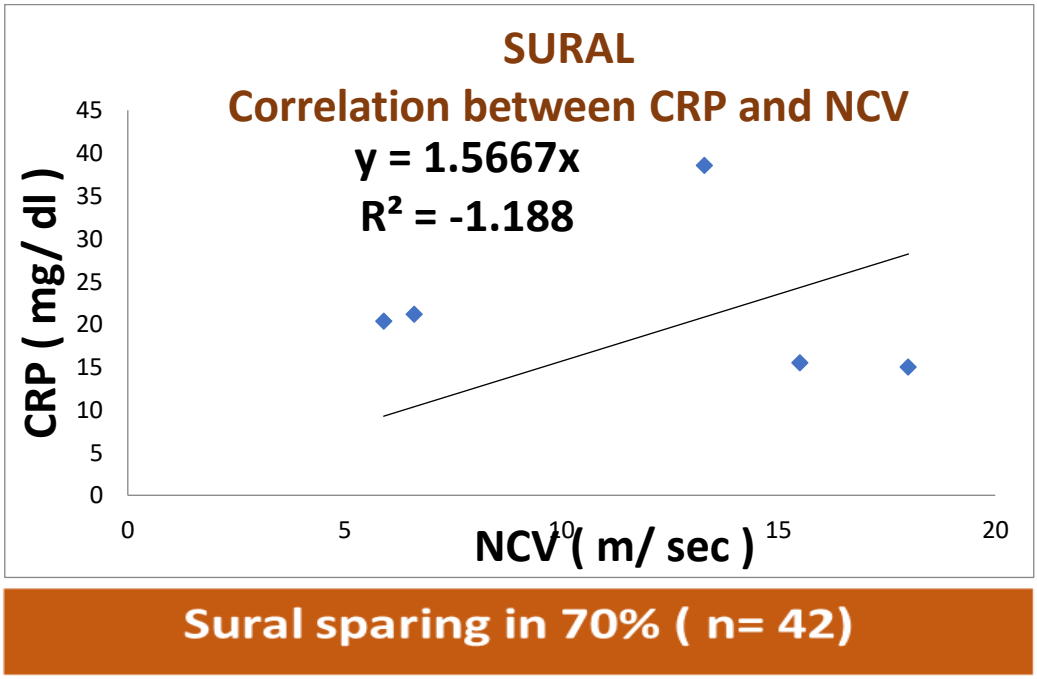
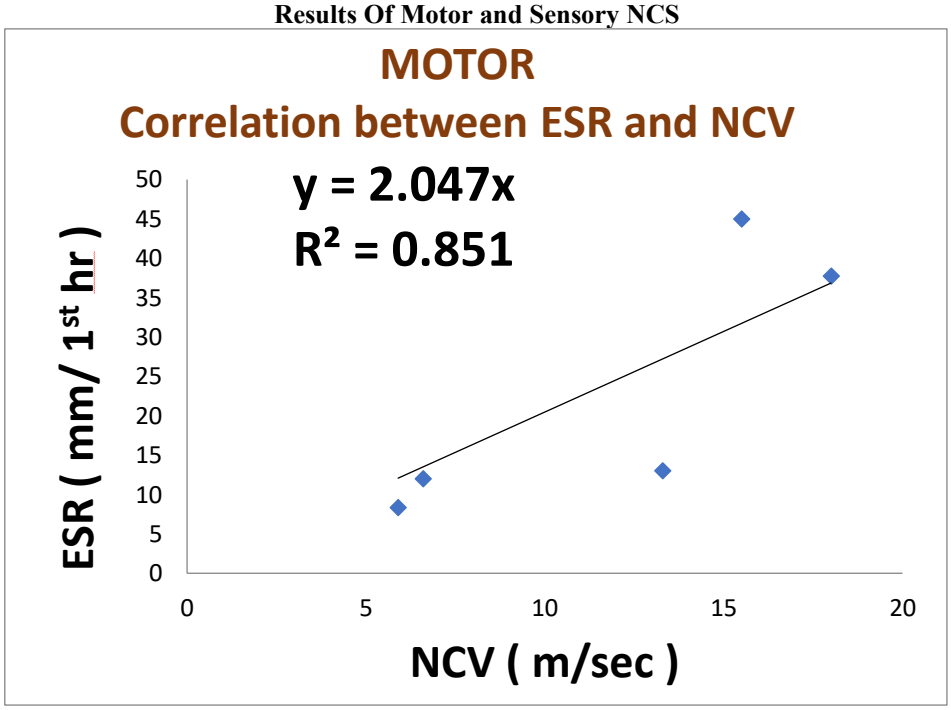


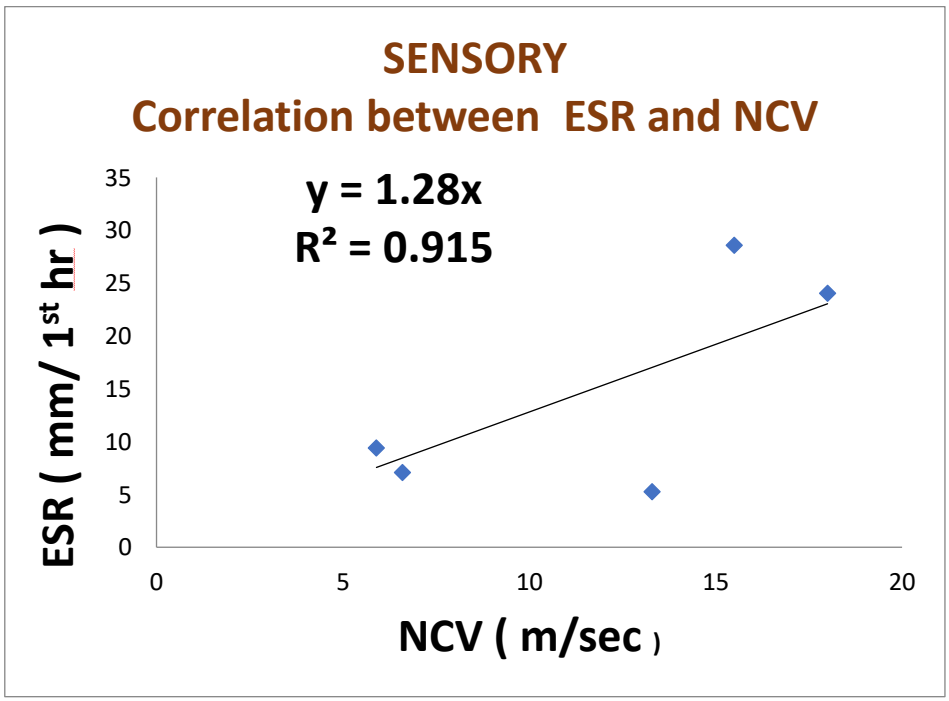
Figure-6
CRP level and Results Of Sensory NCS (sural nerve) in patients with GBS



Corelation study as also done between the ESR level and result of nerve conduction study as shown in figure-7 and figure-8.

Results Of Motor and Sensory NCS





DISCUSSION:

Guillain-Barre syndrome is a severe autonomic disorder in which the immune system forms autoantibodies against healthy nerve cells of the peripheral nervous system and can be clinically diagnosed. Although exact cause is not known, it is triggered by many infectious illness like respiratory tract infection and gastroenteritis. [10] Nerve conduction study can help to diagnose and discriminate between axonal and demyelinating subtypes that could relate to prognosis. [11] C-reactive protein is one of the acute phase protein, the level of which rises in response to a number of nonspecific wide variety of disease including autoimmune diseases.[12]

In the present study, the mean age (yrs)of the GBS patients was 32.55(± 7.96) years. Similar type of result was reported by Mohamed *et al*, 2016 i.e. mean age was 38.74yrs.[13] But another previous study has reported higher mean of the GBS patients i.e. 43.7 years.[14]

Regarding sex distribution, we found GBS was more frequent among male (41) than the female (n=19). This result was in accordance with the result of a previous study by Dhadke et al, 2013 who reported 5.3% male and 41.77% female .[15]But another previous study reported no gender difference among GBS patients .[14]

Concerning triggering infection in GBS patients, 56.6% of patients were suffering from upper respiratory tract infection. This data was in agreement with results obtained by Willson et al., 2005 who reported 62.5% cases were history of URTI. [16] Another study by Newswagner et al.,2004 reported that most common preceding infection in 47.25% cases was Campylobacter jejuni. [17]. Another previous study in the year 2015, has reported that the most common antecedent events associated with GBS was gastrointestinal infection.[18]

Regarding CRP and ESR level in GBS patients in relation to disease severity, we found significantly raised level of both inflammatory markers in grade 4 and grade 5 patients also positive association between the CRP level as well as the ESR level in grade 4 and 5 GBS patients. Similar type of result was reported by a previous study by Vaishnavi et al in 2014 i.e 24.4% cases had raised ESR AND CRP level with bad prognosis.[3]

Patients of Guillain- Barre syndrome differ from each other regarding the degree as well as involvement of the motor and sensory nerves. The nerve conduction study result in majority of our patients (91.4%) shows decreased conduction velocity, decreased CMAP AND SNAP and F- wave abnormalities (83.34%). Parmer et al study in 2013 also reported F-wave abnormalities in 90% case, prolonged distal latency in 0% cases, delayed conduction(73%) and abnormal SNAP(28%).[19] Our result is in agreement with the findings of a previous study conducted in the year 2012.[20]

Mean Age (yr) **32.55 (± 7.96)**	**Mean Age 38.74 yrs (Mohamed *et al.*, 2016)** **Higher mean age 43.7yrs (Gonzalez *et al.*,2013)**
SEX **M (41) > F (19)**	58.3% Male Vs 41.7% Female (Dhadke *et al.*,2013) No difference between both gender (Gonzalez *et al.*,2013)
URTI in 56.6%	62.5% cases of URTI (Willson *et al.*, 2005) 47.25% Campylobacter jejuni most common (Newswagner *et al.*,2004)
CRP and ESR **positively associated** with severity	Raised ESR & CRP in 24.4% cases with bad prognosis (Vaishnavi *et al.*,2014)
F-wave abnormality (83.34%) Decreased CMAP and SNAP **Decreased conduction velocity (91.4%** Prolonged Latency Sural sparing (70%)	F-wave abnormality (90%), Prolonged distal latency(80%), Delayed conduction (73%) and 28% Abnormal SNAP (Parmer *et al.*, 2013) In aggrement with Rajbally *et al.*, 2012

CONCLUSION: -

The findings of this study suggest that inflammatory markers such as CRP and ESR, along with motor and sensory nerve conduction responses, are significantly associated with the clinical severity of Guillain–Barré Syndrome. Therefore, these parameters may serve as useful diagnostic as well as prognostic biomarkers in assessing disease severity and progression. The results may help clinicians in early risk assessment and in making timely therapeutic decisions, which could ultimately improve patient management and clinical outcomes.

However, despite the strengths of this study, it has certain limitations. As this was a cross-sectional study, causal relationships could not be established. Additionally, no attempt was made to differentiate between axonal and demyelinating subtypes of Guillain–Barré Syndrome, which may influence disease severity and prognosis. The ultimate outcome of this research is to provide supportive evidence that combining inflammatory markers with nerve conduction study findings can aid in better evaluation of disease severity and may contribute to improved prognostic assessment in patients with Guillain–Barré Syndrome.

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