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# **Reliability of Anxiety Stress Irritability Behavioral Changes (Asib) – Neuro Psychosomatic Scale in the Post - Covid 19 Population**

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# ABSTRACT

BACKGROUND AND PURPOSE: The Corona Virus Disease (COVID-19) has lead to universal psychosocial impact by causing mass hysteria, economic burden and financial losses. Mass fear of COVID-19, also called as "coronaphobia", has generated a plethora of psychiatric problems all over the society. Disease itself multiplied by forced quarantine to combat COVID-19 applied by nationwide lockdowns can produce acute panic, anxiety, obsessive behaviors, hoarding, paranoia, and depression, and post-traumatic stress disorder (PTSD) in the long run. The ASIB neuro psychosomatic scale for post COVID - 19 patients is basically designed to evaluate the level of neuro psychosomatic impairments caused by COVID – 19.

**DESIGN:** A cross – sectional study.

# SUBJECTS: 500

**METHOD**: An informed consent form (approved by the institution) was taken from the subjects prior to the initiation of testing. For test-retest reliability Subjects were tested twice with ASIB - NEURO PSYCHOSOMATIC SCALE by the same rater, with 15 minutes gap between tests for testretest reliability. ASIB - NEURO PSYCHOSOMATIC SCALE score was taken on 1st occasion and at the same day, it was taken on the 2<sup>nd</sup> occasion with the time gap of 15 minutes by the same rater. Scores of retest was kept in an envelope. For determining intra-rater reliability subjects were tested twice with the ASIB – Neuro psychosomatic Scale by the same rater, on two different occasions (with the time gap of 15 minutes).

**RESULT:** The present study established the intra-rater reliability of ASIB – neuro psychosomatic scale for post COVID – 19 patients. The result demonstrated that there is excellent intra-rater reliability of ASIB - neuro psychosomatic scale for post COVID - 19 patients.

**CONCLUSION:** The present study established the intra-rater reliability of ASIB – neuro psychosomatic scale in post COVID 19 population. The result demonstrated that there is excellent intra-rater reliability of ASIB – neuro psychosomatic scale in post COVID 19 population.

Based on the finding we can conclude that ASIB- Neuro psychosomatic scale is reliable to check anxiety, stress, irritability and behavioral changes in post covid - 19.

**KEYWORDS:** Post covid patients, anxiety, stress, irritability, behavioral changes



# INTRODUCTION

The first COVID-19 pandemic was located in Wuhan, Shin, and spread rapidly of all the states. In people who develop clinical disease in reservoir to SARS-COV-2, the respiratory system is the most affected.<sup>[1]</sup> Coronaviruses get their name from the way they look under a microscope. The virus has a genetic component surrounded by an envelope containing anti-protein proteins. This makes it look like a crown. The word Corona means "crown" in Latin.

Coronaviruses are zoonotic<sup>[12]</sup>, meaning that viruses are transmitted between animals and humans. It was determined that MERS-CoV was transmitted from camel camels to humans and SARS-CoV from civet cats to humans<sup>[11]</sup>. The source of SARS-CoV-2 (COVID-19) is not yet known, but research is still ongoing to identify the source of the zoonotic outbreak<sup>[13]</sup>.

#### Clinical Introduction

Coronaviruses usually have respiratory symptoms. Among those who will be infected, some will not show any symptoms. Those who develop symptoms may develop a mild, but independent disease with symptoms similar to seasonal flu<sup>[14]</sup>.

Symbols can include:

- Respiratory symptoms
- Fever
- Cough
- Shortness of breath
- Respiratory problems
- Fatigue
- Sore throat

A small group of people will show more severe symptoms and will need hospitalization, usually with pneumonia, and in some cases, the illness may include ARDS, sepsis and septic shock <sup>[14] [15]</sup>. Signs of emergency warning for immediate medical attention <sup>[16]</sup> include:

- Shortness of breath or difficulty breathing
- Persistent pain or pressure in the chest
- New confusion or inability to wake up

#### Lips or blue face

However, the virus can be present when body is found. In the most diseased parts, multirle organs at often affected. This virus binds to the enzyme 2 (ACE2) to replace the enzymes 2 (ACE2) of vascular endothelial cells, lungs, heart, brain, kidneys, intestines, liver, pharynx and other body tissues. <sup>[1]</sup>

It can directly damage these organs (lungs, kidneys, intestines, throat, etc.). In addition, systemic problems caused by a virus can eventually cause malfunctions. When treating a patient it is important to check for injuries to many organs. Disruption of coagulation and vasot endothelium is common but may not lead to symptoms at first. They come with injuries to many organs. Heart and kidney failure is common in patients who die. Injuries to the limbs can be seen long after the severe infection is gone. Different organs can be affected at different times. Long-term chronic injuries are possible. Recovery and recovery can be long and difficult. <sup>[2]</sup>

#### Inflammation and endotheliitis

Compared with other health conditions, COVID-19 can lead to a much higher production of cytokines by white blood cells <sup>[2]</sup>. Catecholamine deficiency can precede and contribute to cytokine storm, also called hypercytokinemia or cytokine release syndrome. This negative response can lead to systemic



inflammatory reaction syndrome (SIRS), acute respiratory stress syndrome (ARDS), trauma, shock and multiple deaths. The inflammatory response can continue to increase even if the viral load decreases.

SARS-CoV-2 infects endothelial cells in many organs and causes the spread of lymphocytic endotheliitis, leading to vasoconstriction <sup>[3]</sup>. Accompanying inflammation, hypercoagulability, and edema causes hypoperfusion leading to ischemia. However, patients with pre-existing inflammatory bowel disease treated with anticytokine biologics and other immunosuppressive therapies are not at increased risk due to COVID-19<sup>[4]</sup>.

# **Effect on integration**

Bleeding is uncommon in COVID-19. Deep vein thrombosis (DVT), venous thromboembolism, pulmonary embolism (PE) and cor pulmonale, systemic and pulmonary arterial thrombosis and embolism, ischemic and myocardial infarction (MI) reported <sup>[5 - 10]</sup>. DVT and PE are common among the dead <sup>[7]</sup>. These are caused by inflammation, platelet function, hypercoagulability, endothelial dysfunction, vascular stiffness, stasis, hypoxia, muscle paralysis, and intravascular coagulation (DIC) <sup>[5 - 10]</sup>.

Fever and inflammation cause hypercoagulability and disrupt fibrinolysis. Cytokine interleukin-6 (IL-6) levels are associated with hypercoagulability and severity of the disease. Elevated antiphospholipid antibodies are associated with thrombosis <sup>[11]</sup>. The liver increases the production of procoagulant substances. Prothrombin time and duration of prescribed thromboplastin are moderately extended. Central thrombocytopenia is seen. C-protein proteins are elevated. Cytokine storm and systemic inflammation are associated with lymphocytopenia, higher D-dimer, higher fibrin degradation products (FDPs), and DIC. D-dimer and DIC levels are informative.

The guidelines recommend thromboprophylaxis <sup>[12-13]</sup>. Prophylaxis with low molecular weight or regular heparin, fondaparinux, or a specific anticoagulant such as apixaban or rivaroxaban should be considered. Heparins bind tightly to COVID-19 spike proteins that block the entry of viruses into cells. Heparins also lower IL-6 and reduce body function. Negative studies suggest that among patients requiring mechanical respiration, systemic anticoagulation may be associated with reduced mortality without increasing excessive bleeding <sup>[14]</sup>. However, systemic anticoagulation has not been shown to be beneficial in ARDS due to certain etiologies. After discharge from the hospital an extended prophylaxis may be helpful.

# Lung effects

Autopsy studies show that in the critical stage patients have normal alveolar damage other than association with fibrosis <sup>[15 - 16]</sup>. It is caused by disruption of endothelial and alveolar cells. This leads to fluid and cell formation and the formation of hyaline membranes. Severe and systemic pneumonia is also seen <sup>[17]</sup>. It contains alveolar fibrin synthesis. Airway inflammation is present. Increased capillary permeability causes alveolar and internal edema. Vascular angiogenesis is a characteristic that separates COVID-19 <sup>[18 - 19]</sup>.

Chest CT, subcutaneous and extracellular findings of ground glass opacity and integration are available in patients with COVID-19. The majority of patients are still hospitalized in both countries. On chest radiographs, small incisions are seen that can be evenly distributed.

Several methods are available to treat respiratory failure <sup>[20 - 23]</sup>. Oxygen through the canal of nasal flow, as well as improper ventilation are among the drugs used in these patients. Normal posture can promote oxygenation <sup>[24 - 28]</sup>.

# Heart effects

In COVID-19, heart problems can precede and occur in the absence of pulmonary and other complications <sup>[29-30]</sup>. Ischemic heart damage can occur in patients with coronary artery disease (CAD), those with latent CAD, and those without CAD. The main cause of what was previously the case is plaque rupture and



thrombosis.

The latter is due to insufficient oxygen supply and mimics MI. With acne coronary syndrome due to plaque cracking, antiplatelet treatment and anticoagulation may be helpful. Fibrinolytic treatment and coronary percutaneous interventions can be considered. However, reported cases of acute MI decreased during COVID-19<sup>[31]</sup>.

Myocyte attacks of the virus are seen in some patients. A systemic inflammatory response such as a cytokine storm can cause myocarditis without direct entry of the virus. It can cause heart failure and arrhythmias. This can happen even after the critical stage of infection has been resolved and when there is no lung injury.

About half the survivors have serious heart attacks and heart failure. Respiratory failure is prevalent in the early stages of the disease and heart damage is worse in later stages. The risk factors for diabetes, obesity, age, and high blood pressure are more closely related to death than respiratory disease. In Britain one quarter of all COVID-19 deaths occur among diabetics, with 15% occurring in patients with chronic obstructive pulmonary disease.

Heart failure and elevated brain type natriuretic peptide (BNP) are evident. High levels of troponin and BNP are associated with death. PE can cause elevated troponin and BNP levels. In older patients with existing CAD or high blood pressure, heart failure can be caused by an increased demand for relationships. Myocarditis may be a cause in young patients. Arrhythmias include tachycardia, bradycardia, and asystole. They can be due to inflammation, myocarditis, hypoxemia, allergies, or medications.

Heart problems can occur long after the virus is approved and restored. Inflammation can persist and change silently. For example, dyslipidemia, pulmonary fibrosis, and avascular necrosis have long emerged in many survivors of acute acute respiratory syndrome (SARS), which is closely related to COVID-19.

Commonly used drugs including angiotensin converting enzyme inhibitors and angiotensin II receptor blockers have not been shown to increase the risk of COVID-19 infection or its complications and should not be discontinued <sup>[32]</sup>.

#### **Kidney effects**

COVID-19 suppresses patient management in dialysis and kidney transplants <sup>[33]</sup>. In Britain about 15% of overweight patients have chronic kidney disease. ACE2 receptors are present in the kidneys <sup>[34]</sup>. The virus is found in glomerular cells, tubular epithelium, and kidney podocytes. Acute kidney injury (AKI) often follows systemic abnormalities including diabetes, high blood pressure, chronic kidney disease, hypoxemia and coagulopathy. Cytokine storms can cause severe hypoperfusion with AKI.

Severe kidney damage is also caused by rhabdomyolysis due to hyperventilation or medications that include medications such as remdesivir. In New York, about 90% of patients who were on a ventilator developed AKI <sup>[35].</sup> AKI occurs through temporary contact with respiratory failure.

Due to the lack of continuous medication for kidney replacement and other hemodialysis equipment and supplies, there is widespread use of peritoneal dialysis. The latter is less common in patients in hospitals, especially if they are unstable. A peritoneal dialysis catheter is usually inserted into the abdomen. It is less effective in patients who are beaten because of shortness of breath. Placing a catheter on the side of the abdomen reduces the problem.

Among kidney transplant recipients, the first fever is found in about one part and diarrhea is present in about one half of the patients <sup>[36]</sup>. Compared with the corresponding group they have a faster increase in morbidity and higher mortality.



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#### Effect on the brain

ACE2 receptors are present in the cerebral cortex and brain stem. Some patients have meningitis and encephalitis that indicate an infection of the central nervous system (CNS). There is a depression of stem stem thinking including one that feels oxygen starvation. Neurological manifestations may be the only visual or may be associated with respiratory symptoms or other symptoms <sup>[37 - 39]</sup>. Neurological manifestations are more common in people with a more serious illness. Altered levels of oxygen and carbon dioxide levels may contribute to them. Includes dizziness, headache, paralysis that can include confusion, delirium, and inability to wake up. Delirium is common and can lead to chronic mental retardation including memory impairment. Due to the shortage of widely used sedatives such as propofol and dexmedetomidine, benzodiazepines are used for sedation. They can improve the delirium. The brains of dead patients show hypoxic changes but encephalitis or other changes due to the virus are rare <sup>[40]</sup>.

Cytokine depletion can cause brain inflammation and edema. Some patients have a sensitive storm that can cause symptoms such as fainting due to closure of the cerebral artery may also occur in young patients with no previous history <sup>[41]</sup>. This is in part due to hypercoagulability and endothelial damage. Bleeding in the brain is also evident. Ataxia and fainting may be present. Cranial nerves may be involved. Anosmia and dysgeusia, i.e., a bad sense of taste, are reported <sup>[42]</sup>. Emotional pain, muscle weakness and pain, numbness or numbness in the hands and feet are also seen. Rhabdomyolysis can cause elevated serum creatine kinase. Neurological features among ICU patients with ARDS include encephalopathy, agitation, and confusion <sup>[37]</sup>. Symptoms of the Corticospinal tract with advanced cord stiffness, ankle clonus, and bilateral extensor plantar reflexes are present in most patients.

#### Eye effect

Both the ACE2 receptors and TMPRSS2 proteases required for SARS-CoV-2 infection are found in the upper cells of the cornea, within the eyelids and in the white of the eye <sup>[43]</sup>.

About one-third of hospitalized patients develop ocular abnormalities including conjunctivitis <sup>[44]</sup>. Conjunctivitis is more common in sick patients. Ocular involvement is possible at first. The surface cells are the entry points and repositories of the virus. Ocular degeneration is a source of infection. The infection can persist in the eye for up to three weeks <sup>[45]</sup>.

#### **Intestinal effect**

Intestinal (GI) symptoms include loss of appetite, nausea, vomiting, diarrhea, and abdominal pain or discomfort <sup>[46 - 47]</sup>. These symptoms may start before or appear with or without other symptoms such as fever, myalgias, and cough. The Lower GI tract is rich in ACE2 receptors. The stool of some patients contains a soft infection virus or only RNA and protein fragments of the virus. Patients with a cell infection take longer to remove it. Although a small percentage of patients had GI symptoms, up to half of the virus was shed in the cell <sup>[47]</sup>. Virus protein shell is also found in cells in the stomach, duodenal. More than half of COVID-19 patients in hospitals have elevated lactate dehydrogenase and other liver enzymes that indicate damage to the liver or bile ducts. This may be due to an overactive immune system or to drugs that cause liver damage. Effect on the skin

The skin manifestations of COVID-19 are similar to those of other viruses and chronic inflammatory diseases such as acne, eczema, psoriasis, and rosacea. Vascular disorders associated with skin manifestations can be neurogenic, microthrombotic, or mediated immune complex. In patients with skin manifestations, the majority have patchy erythematous rash [48 - 49]. Some have urticaria or a common cold. A few have fluid-filled vesicles — like fluid-filled water or bubbles. They can have a rash like measles. The most affected area is the trunk. The itch is mild or not. Some patients develop skin rashes at



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the onset of symptoms, and others after hospitalization. The sores usually heal in a few days. Skin expression does not correspond to the complexity of COVID-19.

Patients can develop livedo reticularis. It is a net-like purification of the skin, often the result of abnormal blood pressure. Lacy, a dusky rash, including dead skin cells is visible on the arms, legs and buttocks. They are associated with hypercoagulability. Petechiae exists. Outbreaks of nonpruritic blanching livedoid arteries, possibly due to vaso-occlusion may occur. They appear as black, red as net, or pink spots. And there are chilblains, which are purplish, slightly strong and tend to be soft. COVID's toes and fingers have ice-like areas with red or purple bumps or nest-like eruptions.

# **Psychological effects**

Due to financial difficulties and social isolation due to COVID-19, many psychological problems can arise. They can be delayed for months. There is an increase in "death from despair" from drug abuse or suicide. The risk is very high for people with dementia, mental illness, and autism. Personal communication and online communication with friends and support professionals is helpful.

When ICU is discharged, one-third of patients have dysexecutive syndrome which involves inattention, disturbance, or irregular movements in response to a command <sup>[37]</sup>. Some patients recovering from COVID-19 experience mental health problems <sup>[50]</sup>. These include anxiety, depression, and post-traumatic stress disorder (PTSD). Long-term side effects may include the development of Alzheimer's disease or Parkinson's disease.

# People at High Risk

The virus that causes COVID-19 infects people of all ages. However, evidence to date suggests that two groups of people are at high risk of developing the severe COVID-19<sup>[17]</sup>:

- Older people (people over 70 years of age)
- People with serious illnesses such as:
- Diabetes
- Heart disease
- Chronic respiratory disease
- Cancer
- High blood pressure
- Chronic liver disease

WHO has issued and published the advice of these high-risk groups and the public support. This is done to ensure that these high-risk individuals are protected from COVID-19 without discrimination, stigma, high risk of being left out or having limited access to basic services and social care. WHO recommendation for high-risk people <sup>[17]</sup>:

- If you have guests in your home, extend a "1 meter greeting", such as a wave, a handshake or a bow.
- Ask guests and their roommates to wash their hands.
- Clean and disinfect your area (especially those that people are most affected by) regularly.
- Reduce shared space if your roommate is unwell (especially with COVID-19 symptoms).
- If you show signs of COVID-19 illness, contact your healthcare provider by telephone, before visiting your health facility.
- Have an app to prepare for the COVID-19 outbreak in your community.
- If you are in public, follow the same prevention guidelines as you would at home.
- Keep up-to-date information on COVID-19 by accessing information from reliable sources.



# **COVID-19 transfer**

The evidence is still there, but current data shows that transfers from one person to another are possible. The mechanisms of transmission of COVID-19 remain unclear at present, but evidence from other coronaviruses and respiratory diseases suggests that the disease may be transmitted by large respiratory droplets and directly or indirectly affect the infected secretion <sup>[18]</sup>. Air transfers can be done in crowded places and indoor air-conditioned rooms, especially infected people who spend a lot of time with others, such as a shopping mall, a restaurant, etc. Also air transfers are performed in medical care facilities while performing medical care procedures Production procedures).<sup>[18] [19]</sup>

Transmission can easily occur in "Three C's" (the risk of spreading COVID-19 is high in areas where these "3Cs" meet:

Crowded places with many people nearby;

Social networking settings, especially when people are chatting a lot;

Closed and closed spaces that allow airflow<sup>[19]</sup>.

The incubation period of COVID-19 is currently understood to be between two and 14 days <sup>[16]</sup>. This means that if a person is healthy within 14 days of contact with a person with COVID-19 certified, he or she is not infected.

Recent studies of COVID-19 outbreaks and studies conducted in previous epidemics underscore that major outbreaks appear to have far-reaching psychological effects. Among these psychological and psychological effects, symptoms such as depression, irritability, anxiety, fear, complaints of depression and post-traumatic stress disorder are common.

Biological, psychological and social factors are considered to play an important role in the aetiology of psychiatric symptoms and related disorders. <sup>[8]</sup>

In particular, life events and stress appear to be closely related to various physical ailments with major symptoms such as chronic pain, gastrointestinal disorders (GI) and headache.<sup>[9]</sup>

Biological, psychological and social factors are considered to play an important role in the aetiology of psychiatric symptoms and related disorders <sup>[18]</sup>. In particular, life events and stress appear to be closely related to various physical ailments with major somatic symptoms such as chronic pain, gastrointestinal disorders (GI) and headache <sup>[19,20]</sup>. Psychosomatic complaints can develop after traumatic events. For example, high levels of somatic symptoms have been reported among civilians exposed to war <sup>[21,22]</sup> as well as military veterans and emergency personnel <sup>[23]</sup>.

With the COVID epidemic - 19, practices such as sleep, diet, daily activities (sports, etc.) and social activities have been banned due to changes in working conditions, as well as forced separation or timing. In addition, reduced sleep quality and efficiency reduce sleep quality and disrupt daytime function. It is reported that all of these components can directly cause somatic complaints <sup>[25]</sup>. Similarly, there are studies that promote the relationship between circadian rhythm and natural rhythm and psychological complaints among university students <sup>[26,27]</sup>. In this context, in the present study, we focused on sleep problems and examined natural rhythms of varying sizes (sleep, activities, social rhythm and food pattern). The current study is designed to study psychological complaints after the development of the COVID-19 Epidemic. Related to psychological complaints. In addition, current research illuminates the necessary psychological interventions in identifying areas that are most effective in post-epidemic rehabilitation. Prior to the start of the study, we hypothesized that

1. increased frequency of symptoms following COVID-19 outbreaks,

2. psychological symptoms would be related to biological rhythm, intolerance and incomprehensibility,



and apparent threat of COVID-19 from schools, and biological rhythm will play a mediator role in the relationship between the visual threat of COVID-19 from many schools and psychiatric symptoms. Various scales have been developed for the evaluation of COVID 19 cardio pulmonary effects and psychological effects.

#### METHODOLOGY

STUDY DESIGN: Cross-sectional study

**SAMPLING METHOD:** Purposive sampling

**STUDY POPULATION:** Post covid – 19 population

SAMPLE SIZE: 500

**STUDY SETTING:** hospitals and societies

#### STUDY DURATION: 6 month

#### SAMPLING CRITERIA

#### **Inclusion criteria**

- 1. Male and female patient post covid infection.
- 2. Patient between 35 to 55 years of age.
- 3. Patient must be post covid at least 1 month back.

#### **Exclusion criteria**

- 1. Patient with any diagnosed neurological impairment.
- 2. Patient with any diagnosed psychiatric impairment.
- 3. Person who cannot understand and follow the commands.

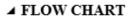
#### Procedure of data collection :

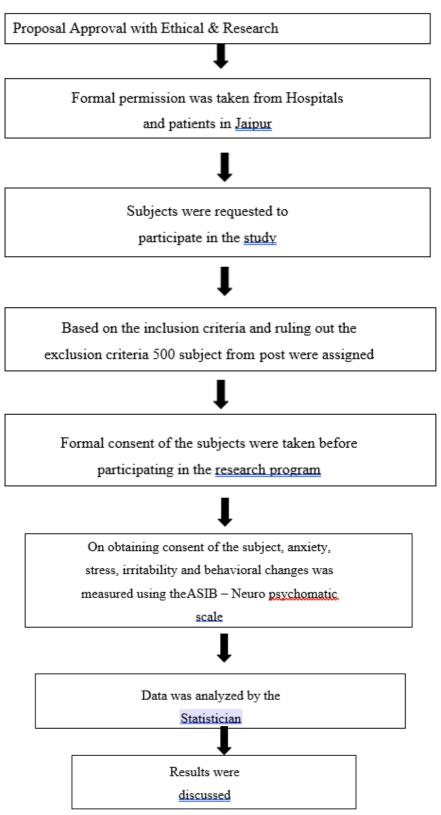
- The participants who meet the inclusion and exclusion criteria were explained about the procedure and were asked to sign an informed consent form (approved by the institution) prior to the initiation of analysis.
- For test-retest reliability Subjects were tested twice with ASIB Neuro psychosomatic scale by the same rater, with 15 minutes of gap between tests for test-retest reliability.
- ASIB score was taken on 1st occasion, date and time will be noted, and was kept in an envelope. Only the time and date of the first occasion was provided at the administration of occasion-2.
- On the next occasion after 15 minute, retest was done by the same rater.
- Scores of retest was kept in an envelope.
- For determining intra-rater reliability subjects were tested twice with ASIB Neuro -psychosomatic scale by the same rater, with 15 minutes of gap.



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# RESULT

The data analysis was done by using the statistical software SPSS (version 16) for windows and Descriptive statistics were calculated for baseline characteristics are age, gender, anxiety, irritability, stress, behavioral changes in the post Covid – 19 patients. The mean age of all the participants was 44.03 years with a standard deviation of 6.235 years for 500 post-covid subjects were selected for this study in which 264 were males and 236 were females subjects.

In this study ASIB – NEURO PSYCHOSOMATIC SCALE was applied on the subjects twice by the same rater with 15 minutes of gap between two tests for intrarater reliability and to find the intrarater reliability between 1<sup>st</sup> occasion and 2<sup>nd</sup> occasion score of ASIB – NEURO PSYCHOSOMATIC SCALE, intraclass Correlation Coefficient (ICC) was used in this study.

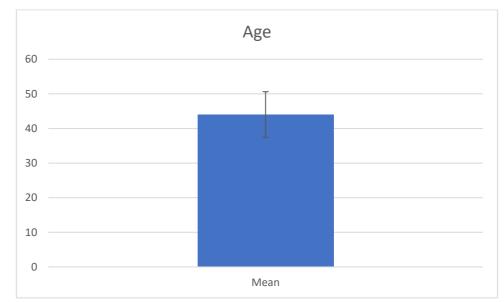
This study is proven to be significantly reliable in its test and re test. The reliability measures testing the interclass correlation coefficient for the study came out to be 0.999 and the cronbach alpha score is 1.000

# **GRAPH OF THE GENDER DISTRIBUTION**

#### 1. DEMOGRAPHIC DATA

	Mean	S.D
Age	44.03	6.235

Table 1 : The table is showing the mean of age and standard deviation of subjects taken for the<br/>study.



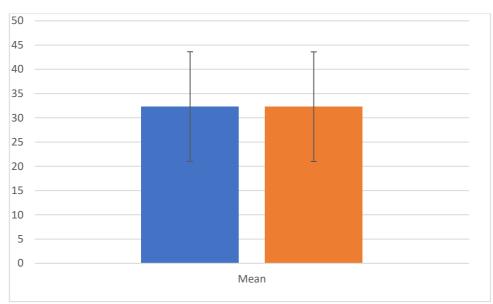
Graph 1: The graph is showing the mean age of subjects taken for the study.

#### 2. <u>TEST RE-TEST RELIABILITY</u>

	Mean	S.D.
Test	32.32	11.666
Re-Test	32.30	11.670

 Table 2 : It represent the test re-test reliability of ASIB Neuro psychosomatic scale with mean & standard deviation.





Graph 2 : It represent the test re-test reliability of ASIB Neuro psychosomatic scale with mean & standarddeviation.

# 3. <u>INTERCLASS CORRELATION COEFFICIENT & CRONBACH α SCORE OF</u> <u>INTERARATER RELIABILITY</u>

Cronbach Alpha Score	1.000
Interclass Correlation Coefficient	.999

 Table 3: It represent the ICC & Cronbach α score of interarater reliability for ASIB Neuro

 psychosomatic scale in post COVID 19 patients.

#### DISCUSSION

A variety of scales have been developed to study the different effects of SARS 2 in post Covid patients. For studying anxiety, depression, stress and irritability in psycho somatic patients there are various scales present. ASIB Neuro psycho somatic scale specifically focuses on post Covid patients and is here used to study there effects.

500 post covid subjects with a mean age of is 44.03 years with were selected for this study in which 264 were males and 236 were females subjects.

In this study ASIB Neuro psychosomatic scale was applied on the subjects twice by the same rater with 15 minutes gap between two tests for intrarater reliability. For ASIB Neuro psychosomatic maximum score is 65.

To find the intrarater reliability between 1<sup>st</sup> day and 2<sup>nd</sup> occasion score of ASIB Neuro psychosomatic scale, intraclass Correlation Coefficient (ICC) was used in this study.

For intrarater reliability, the results of the present study showed that occasion 1 and day 2 ASIB Neuro psychosomatic scale scores are closely related with each other. It shows the ICC = 0.999 (Table no..).

The Anxiety Stress Irritability and Behavioral changes (ASIB) Neuro psychosomatic scale consists of 4 components anxiety, stress, irritability and behavioral changes and the grading is done in the sequence of: 0-25 NORMAL

25-50 MILD 50-75 MODERATE



#### 75-100 SEVERE

**Noam matalon , et.al, april 2021** conducted a study on Trajectories of post – traumatic stress symptoms, anxiety, and depression in hospitalized Covid 19 patients : A one month follow up. From this study they concluded that 20 % of Covid 19 hospitalized patients develop significant PTSS a month after hospitalization.

**Lu yang, et.al 2021** conducted a study on Analysis of psychological state and clinical psychological intervention model of patients with Covid 19. They concluded that patients those who diagnosed with COVID 19 in the isolation ward or general pneumonia in observation ward have different degrees of anxiety, depression and sleep problems

**Qian Gua, et.al 2020** conducted a study on Immediate psychological distress in quarantined patients with Covid 19 and its association with peripheral inflammation: a mixed method study. This study showed that significant psychological distress was experienced by hospitalized Covid 19 patients and that levels of depressive features may be related to the inflammation markers in these patients.

**Jinzhi Li, et.al 2020** performed a study on The effects of cognitive behavioral therapy on depression, anxiety, and stress in patients with Covid 19 : A Randomized controlled trial. The study showed that the patients with Covid 19 experienced high levels of anxiety, depression and stress.

**Lioudmila V Karnatovskais, et.al 2020** conducted a study on Stress and fear : clinical implication for providers and patients (in the Time of Covid 19 and beyond). This study concluded that the physiological and psychological consequences of the worldwide Covid 19 pandemic on both patients and caregivers are well documented, but the link between physiology, pathophysiology and psychology in this milieu is underappreciated.

# CONCLUSION

The present study established the intra-rater reliability of ASIB- Neuro psychosomatic scale in post Covid – 19 patients. The result demonstrated that there is excellent intra-rater reliability of ASIB- Neuro psychosomatic scale.

By accepting the Experimental Hypothesis and rejecting the Null Hypothesis we can conclude that the ASIB – Neuro psychosomatic scale is reliable to measure the anxiety, stress, irritability and behavioral changes in post COVID – 19 patients.

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