IAMJ

INTERNATIONAL AYURVEDIC MEDICAL JOURNAL

Research Article ISSN: 2320-5091 Impact Factor: 6.719

CLINICAL PROFILE IN COVID WSR VIKRUTI PAREEKSHA

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https://doi.org/10.46607/iamj1011042023

(Published Online: April 2023)

Open Access

Article Received: 31/03/2023 - **Peer Reviewed:** 14/04/2023 - **Accepted for Publication:** 19/04/2023.

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ABSTRACT

AIM: Requirement of a prior confirmation of COVID-19 and its effects through *Roga - Rogi Pareeksha* to calculate extend of sage to treat accordingly. BACKGROUND: COVID-19 is a highly infectious disease caused by SARS-COV-2 that can lead to severe respiratory distress and complications in some patients. It is considered a zoonotic disease and brought the world into the practice of isolation, quarantine, and sanitizing which is new to this 21st Century. Its way of transmission from one to another to its host invasion and how the virus is capable of entering through ACE-2(Angiotensin converting enzyme) receptors of the body. However, COVID has proved to be fatal for many with comorbidities. REVIEW RESULTS: Lab investigations like ALT, CRP, CK, Creatinine, Lactate Dehydrogenase, Ferritin, D Dimer, and HRCT to calculate extent of sage along with tests for diagnosis and treat accordingly. CONCLUSION: Assessment of *Vikruti Pareeksha* along with *Rogi Pareeksha* by lab investigations provides a better prognosis CLINICAL SIGNIFICANCE: Ayurvedic classics have given us enough evidence of what actually goes on within a body when an *Aganthuja Nidana* enters, and its vitiation caused can be explained with the help of and *Vyadhi Ghatakas* and severity from *Vikruti Pareeksha*.

Keywords: COVID-19, *Vikruti Pareeksha, Srotas, Dhatu Paka,* ALT, CRP, CK, Creatinine, Lactate Dehydrogenase, Ferritin, D Dimer

INTRODUCTION

The World Health Organization (WHO) declared the outbreak a pandemic on March 11, 2020. COVID-19 has resulted in severe morbidity and mortality, which badly affected the global economy and caused a loss of employment for millions of people. Since then, frontline warriors turned health care providers have been struggling to fight the virus, the disease, and its post-term complications. It all includes putting all efforts to find out the exact course of the disease, histopathology, and exploring various treatments including antiviral therapy, plasma therapy, vaccines, etc., for COVID-19 cases. On other hand, Ayurveda has a holistic approach to disease through *Roga Gyana* (knowledge of disease) and to look onto its *Lakshanas* (symptoms) and clearly understand *Samprapthi* (Pathogenesis) and carefully figure out *Upasaya* and *Anupasaya* (aggravating and relieving factors) and if signs observed early(poorvarupa) can be treated effectively by observing the *Avasthas* of *Vyadhi.* However, Ayurveda has always been known for ages to protect from diseases and prescribes Yoga, and Pranayama to enhance *Vyadhi BalaVirodhitva* (innate immunity).

Corona is new to the world, but *SannipataJwara, Swasa, and Kasa* are not new to *Ayurveda*. Even if the disease is not clearly understood, it can be taken as *AnuktaVyadhi* and treated based on predominant symptoms. Hence the new pandemic requires a clear understanding of *Samprapti* so that the pathogenesis can be stopped by *Samprapthi Vighatana*.

OBJECTIVES: Requirement of a prior confirmation of COVID and its effects through *Roga – Rogi Pareeksha* to calculate extent of sage to treat accordingly.

MATERIALS AND METHODS:

Authentic books of Ayurveda including Classical literatures like *CharakaSamhita, Susruta Samhita, AshtangaHrudaya, and Yogaratnakara* to understand the *Roga* and *Rogi Pareeksha* in *Anukta Vyadhi* COVID-19. Scholarly articles, google searches, and laboratory textbooks were also used to collect the data.

DISCUSSION

COVID-19, a series of acute atypical respiratory infections wrecked Wuhan city of China in December 2019. The pathogen is responsible for these atypical infections was soon discovered to be a novel coronavirus belonging to the family Corona viridae and was named the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). It was seen to be highly homologous to the SARS corona virus (SARS-CoV), which was responsible for the respiratory pandemic during the 2002–2003 period. The respiratory illness caused by this virus was termed coronavirus disease 2019 by the WHO, and the outbreak was considered to have started via a zoonotic spread. Subsequently, the human-to-human transmission was recognised to be responsible for the community spread of the disease.

WHY IS IT FATAL

The spike protein on the surface of the virus and the central nucleocapsid protein, which holds the viral genome are more virulent. The insertions in the spike protein appear to help the viruses penetrate human cells. The researchers determined that the spike insertions may improve the viruses' ability to interact with the receptor on human cells that the viruses use to gain entry. The role of the insertions in the nucleocapsid protein is this protein has several functions.

TRANSMISSION OF CORONA VIRUS :

People release respiratory fluids during exhalation (e.g., quiet breathing, speaking, singing, exercise, coughing, sneezing) in the form of droplets across a spectrum of sizes. These droplets carry the virus and transmit infection.

- The largest droplets settle out of the air rapidly, within seconds to minutes.
- The smallest very fine droplets and aerosol particles formed when these fine droplets rapidly dry, are small enough that they can remain suspended in the air for minutes to hours. [(1)]

Infectious exposures to respiratory fluids carrying SARS-CoV-2 occur in three principal ways (not mutually exclusive):

1. **Inhalation** of air carrying very small fine droplets and aerosol particles that contain the infectious virus. The risk of transmission is greatest within three to six feet of an infectious source where the concentration of these very fine droplets and particles is greatest.
2. **Deposition** of virus carried in exhaled droplets and particles onto exposed mucous membranes (i.e., "splashes and sprays", such as being coughed on). The risk of transmission is likewise greatest close to an infectious source where the concentration of these exhaled droplets and particles is greatest.
3. **Touching** mucous membranes with hands soiled by exhaled respiratory fluids containing a virus or from touching inanimate surfaces contaminated with the virus. [(2)]

VIRAL CYCLE AND HOST INVASION :

The virus is transmitted via respiratory droplets and aerosols from person to person. Once inside the body, the virus binds to host receptors and enters host cells through endocytosis or membrane fusion. Corona viruses are made up of four structural proteins, namely, the spike (S), membrane (M), envelop (E), and nucleocapsid (N) proteins. The S protein is seen to be protruding from the viral surface and is the most important one for host attachment and penetration. This protein is composed of two functional subunits (S_1 and S_2), among which S_1 is responsible for binding to the host cell receptor and the S_2 subunit plays a role in the fusion of viral and host cellular membranes. ACE-2 has been identified as a functional receptor for SARS-CoV and is highly expressed in pulmonary epithelial cells. It is through this host receptor that the S protein binds initially to start the host cell invasion by the virus.

After binding of SARS-CoV-2 to the ACE-2, the S protein undergoes activation via a two-step protease cleavage: the first one for priming at the S1/S2 cleavage site and the second cleavage for activation at a position adjacent to a fusion peptide within the S_2 subunit. The initial cleavage stabilises the S2 subunit at the attachment site and the subsequent cleavage presumably activates the S protein causing conformational changes leading to viral and host cell membrane fusion.

Post membrane fusion, the virus enters the pulmonary alveolar epithelial cells, and the viral contents are released inside. Now inside the host cell, the virus undergoes replication and formation of a negative-strand RNA by the pre-existing single-strand positive RNA through RNA polymerase activity (transcription).

This newly formed negative strand RNA serves to produce new strands of positive RNAs which then go on to synthesis new proteins in the cell cytoplasm (translation). The viral N protein binds the new genomic RNA, and the M protein facilitates integration into the cellular endoplasmic reticulum. These newly formed Nucleocapsids are then enclosed in the ER membrane and transported to the lumen, from where they are transported via golgi vesicles to the cell membrane and then via exocytosis to the extracellular space. The new viral particles are now ready to invade the adjacent epithelial cells as well as for providing fresh infective material for community transmission via respiratory droplets.

INVOLVEMENT OF LOWER RESPIRATORY TRACT

The virus invades and enters the type 2 alveolar epithelial cells via the host receptor ACE-2 and starts to undergo replication to produce more viral Nucleocapsids. The virus-laden pneumocytes now release many different cytokines and inflammatory markers

such as interleukins (IL-1, IL-6, IL-8, IL-120, and IL-12), tumor necrosis factor-α (TNF-α), IFN-λ and IFN-β, CXCL-10, monocyte chemoattractant protein-1 (MCP-1) and macrophage inflammatory protein-1α (MIP-1α). This 'cytokine storm' acts as a chemoattractant for neutrophils, CD4 helper T cells, and CD8 cytotoxic T cells, which then begin to get sequestered in the lung tissue. These cells are responsible for fighting off the virus, but in doing so are responsible for the subsequent inflammation and lung injury. The host cell undergoes apoptosis with the release of new viral particles.

SIGNIFICANCE OF ACE

Angiotensin-converting enzyme-2 (ACE2) has been found to be the SARS-CoV-2 cell entry receptor while TMPRSS2, a cellular transmembrane serine protease, is employed by the virus for S protein priming. Upon viral entry, the spike proteins of both SARS-CoV and SARS-CoV-2 cause the internalization and degradation of ACE2 that critically contribute to lung damage. A decrease in ACE2 activity exacerbates the severity of lung injuries and inflammatory lung. Type II alveolar cells are not the only ones to express ACE2: indeed, it has been detected in the myocardium, kidney, urothelial, ileum, colon, esophagus, and oral mucosa cells. This may in part explain the multiple systemic presentations of COVID-19. Recent evidence suggests that while younger subjects may be more prone to get infected**,** lower levels of ACE2 in older patients may prompt more severe clinical behavior of COVID-19. Furthermore, patients with more aggressive COVID-19 clinical behavior are more often in older age groups and may progress toward ARDS.

IMPORTANCE OF LAB INVESTIGATION :

"Prevention is better than cure"- In accordance with this saying, medical laboratory testing plays a crucial role in the early detection, diagnosis, and treatment of disease in patients. Leukocytosis, elevated ALT, lactate dehydrogenase, high-sensitivity cardiac troponin I, creatine kinase, d-dimer, prothrombin time, serum ferritin, procalcitonin, and IL-6 have been associated with death in a cohort of 191 patients infected with SARS-CoV.[(3)]

ALT TEST

Elevated levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were reported, ranging from 14% to 53%. Additionally, a pathological study of liver biopsy specimens from a patient who died from COVID-19 showed moderate micro vesicular steatosis and mild lobular and portal activity, indicating that SARS-CoV-2 may have led to this liver damage. ACE-2 in cholangiocytes was similar to that of type 2 pneumocytes, indicating that the liver is a potential target organ for SARS-CoV-2. These include direct damage of the virus penetrating by ACE-2 into the liver tissue, an uncontrolled inflammatory/immune reaction causing fibrosis and liver dysfunction, or a liver lesion caused by anti-COVID-19 drug therapy. *Yakrut* being *raktava-hasrotomula* gets affected and shows signs of affliction in blood.

LACTASE HYDROGENASE

The enzyme is composed of two major subunits (i.e., A and B), and is present in humans in five separate isozymes (LDH-1 in cardiomyocytes, LDH-2 in reticuloendothelial system, LDH-3 in pneumocytes, LDH-4 in kidneys and pancreas, and LDH-5 in liver and striated muscle). The acidic extracellular pH due to increased lactate from infection and tissue injury triggers the activation of metalloproteases and enhances macrophage-mediated angiogenesis. Severe infections may cause cytokine-mediated tissue damage and LDH release. Since LDH is present in lung tissue (isozyme 3), patients with severe COVID-19 infections can be expected to release greater amounts of LDH in the circulation, as a severe form of interstitial pneumonia, often evolving into acute respiratory distress syndrome, is the hallmark of the disease. [(4)]

CREATININE TEST

The exact mechanism of kidney involvement is unclear and likely multi-factorial. Kidney disease may be caused by SARS-CoV-2 binding to the ACE2 receptor on kidney cells that allows the virus to enter. Diffuse damage in proximal tubules with the loss of brush border, vacuolar degeneration, and even necrosis was observed. Diffuse erythrocyte aggrega-

tion with endothelial damage, and obstruction without fibrin thrombi or distinct fragmentation of erythrocytes or platelets[5]

FERRITIN

Elevated forms of this iron storage indicate the presence of any kind of virus or bacteria in the human body. Ferritin may be not only a marker of the inflammatory milieu but also an active player in the "cytokine storm" scenario that characterizes severe COVID-19. Complex feedback mechanisms between ferritin and cytokines in the control of pro-inflammatory and anti-inflammatory mediators might exist as cytokines can induce ferritin expression, but ferritin can induce the expression of pro- and anti-inflammatory cytokines as well. [6,7]

D DIMER

D-dimer is an indirect marker of active coagulation and thrombin formation. It is released when plasmin, a fibrinolytic enzyme, cleaves fibrin to degrade clots and represents a mirror of the endovascular thrombotic processes. [8]

IL- 6 (INTERLEUKIN 6)

IL 6 is an interleukin that controls the immune response in addition to cell proliferation and differentiation. After viral infection, viral products enhance the transcription or translation of IL-6 from cells such as fibroblast, mesenchymal, endothelial, and many other cells which are an indication of pulmonary inflammation and severe lung damage. The targets of IL-6 are B cells, T cells, basophils, eosinophils, and neutrophils. The functions of IL-6 on B cells are differentiation of the B cells as well as IgM, IgE, and IgA production. In addition, IL-6 also controls the activation, differentiation, and survival of the T cells. IL triggers the activation of leukocytes. Therefore, after infection, cytokine storm causes T and B cell activation and differentiation. Increased levels of IL-6 are demonstrated. IL-6 secretion causes antibody production from B cells, and it enhances auto-antibody hypergammaglobulinemia. As a result of the cytokine storm. IL-6 levels can be seen elevated.

C- REACTIVE PROTEIN

CRP is a type of protein produced by the liver that serves as an early marker of infection and inflammation. In blood, the normal concentration of CRP is less than 10 mg/L; however, it rises rapidly within 6 to 8 hours and gives the highest peak within 48 hours from the disease onset. Its half-life is about 19 hours, and its concentration decreases when the inflammatory stages end and the patient is healing. CRP preferably binds to phosphocholine expressed highly on the surface of damaged cells. This binding makes active the classical complement pathway of the immune system and modulates the phagocytic activity to clear microbes and damaged cells from the organism. When the inflammation or tissue damage is resolved, CRP concentration falls, making it a useful marker for monitoring disease severity. [9]

N/L RATIO:

Neutrophils are primarily responsible for activating the immune system, and systemic inflammation destroys CD4+ T lymphocytes and increases suppressor CD8+ T lymphocytes, thereby leading to an increased neutrophil-to-lymphocyte ratio (NLR). An increase in the apoptosis of lymphocytes leads to lymphopenia and elevated thrombopoietin (THPO) promotes megakaryocyte production. The reflections of these inflammatory changes can be vital in gauging the progression of the disease. It is an important marker in the initial phase of the disease to study the inflammatory changes in the body.

OTHER IMPORTANT PARAMETERS :

RT-PCR to confirm the infection and Ag test for random checking. Ab tests however are less used, and it shows positive if affected long back also. Findings of HRCT are valuable in corona-negative cases and also to check for cytokine storms. A home kit with a thermometer pulse oximeter is essential to check the vitals like temperature, oxygen saturation, and heart rate at home. Respiratory rate can be checked by assessing the abdominal or chest movement of a patient lying in bed. Awareness of patients regarding checking vitals is the duty of doctors in discharged patients, in isolation or who are of low-risk category.

ASSESSMENT OF VIKRUTI PAREEKSHA IN COVID :

It's all in wake of the Covid Pandemic made us realize the practicality of *Janapadodwamsa*[10] *Yuge Yuge Dharma pada kramena Anena Heeyathe*…) illuminating the drooping of dharma, the quality of plants, our immune system. COVID is primarily a *Pranavahasroto Vikara* affecting the lungs and their adjacent organs. As per *Charaka Acharya*, "*Pranavahanam Srotasam Hridayam Moolam Mahasrotas Cha*[11] elucidates that the whole of the cardiopulmonary system is disrupted in COVID. In a similar manner, *Hrdaya* is also *Rasahavaha Srotomula*, *Ojo Sthana*[12] **(**seat of the essence) that takes about to affect the blood that circulates in the body. The target site of this virus is the *Kapha Sthana(Urah Kanda Shira Kloma Kaphasya Sutharam Uraha)*[13] as mentioned by *Vagbhata Acharya* and hence all signs of *Kapha Dushti* and *Avruta Vāta* aggravation, thus dry cough (*Vataja Kasa*) [14] dyspnoea, fatigue, heaviness on the chest can be observed thereby leading to ARDS (acute respiratory distress syndrome) subsequent *Pranaavrodha* leading to *Ojokshaya* and *OjoVisramsha*.[15] . Among the *Pancha Kapha* present, the *Avalambaka Kapha*[16] has its seat in H*ridaya* whose afflict ion leads to disturbance in fluid balance of pericardium and pleura. The *Rasnadasamoola* includes the liver and other *Koshtangas* extend the *Kapha Dushti* to *Raktavaha Srotas* causes *Pichila*(slimy), *Tantumata*(thready) and *Ghanata*(clot)nature of blood[17] make them susceptible to clotting diathesis. Assessing *Vikruthi Pariksha* gives an idea about *Vyadhi Ghataka*, the same guna creates a "*Daruna Vyadhi*" a Strong disease whose *Vyadhi Bala* is high. This is best explained in the case of a diabetic patient who is already suffering from *Kapha Dusti* and increased *Kledatha* in the body. The *Rakta Dusti* causes coagulation in a patient who is already suffering from Peripheral vascular disease(PVD), bleeding disorders, and heart disease because the properties of *Dosha-Dusya* in the patient are the same as the COVID making it a *Balavan Vyadhi*. [18]

1. Creatinine- kidney damage
2. ALT – Liver Damage
3. Creatine kinase – Muscle damage including heart muscle.
4. D Dimer – Cross-linking fibres to rule out a blood clot
5. LDH – Multi-system damage as seen in the liver, kidney, skeletal muscles, blood cells, etc.
6. Inflammatory markers – CRP, IL – 6, Ferritin, NL ratio – clear *Dhatupaka* markers

All the above-mentioned markers give the early idea before developing *Vyakta Avastha*(complete manifestation) of *Paka* in individual *Dhatu* which further progresses to cause *Nidranasha*(loss of sleep), *Hrudi Sthambha* (heart-related morbidities), *Vishtambha*(sluggishness) in circulation, heaviness, irritability, loss of taste and generalized weakness[19]. On *Vikalpa Samprapti* (cross-sectional study) the major affected *Srotas* are *Pranavaha*, *Rasavaha*, and *Raktavaha*, but based on *Khavaigunya*, more *Srotas* involvement is observed like *Mamsavaha*, *Medavaha*, *Annavaha*, and *Udakavaha* makes condition grave. It is understood from *Kasa Samprapti* [20] that *Prana Vayu* along with *Udana Vayu* is getting affected in *Kasa*, which is the common symptom of COVID. *Udana* controls the normal functions of areas like the nose throat and abdomen[21] thus setting back. This results in temporary loss of taste and smell. Thus, major *Kapha Sthana Ura*(thorax) is affected by causing *Avarana* to *Vata Dosha* resulting in its functional abnormality causing running nose, thorn pricking sensation of throat cough dyspnea, loss of smell and taste. Based on the *Dosha* dominance, this can also affect the *Vishesha Sthana*[22]of *Tridoshas* causing back ache, diarrhea, loss of appetite, fever, headache chest pain, etc. Excessive *Kapha Dusht*i always produces *Rasa Dushti* and obstruction of *Srotas* which is evident from *Rasa Vrudhi Lakshana* and this *Srotorodha* (obstruction) results in an increase in *Sthanika Ushma* generating severe *Dhatupaka*. Those with less mental strength get depressed and debilitated by the diagnosis (*Vishadho Roga Vardhananam*) (*Soka Soshananam*), who maintained the inferior type of food habits (*Ekarasabyaso Dourbalyakaranam*), old age (*Vridho*

Yapyanam)[23] with comorbidities develops complications.

Thus, this gives us clear evidence of why COVID makes a person with the above-said factors fatal and those with dissimilarity experience only lighter affliction.

CONCLUSION

The COVID pandemic has come from somewhere and now everywhere shaking all levels of life and causing damage to livelihood. It is all left to us to handle the pandemic with the best of all our efforts by keeping up our immunity by following *Dinacharya, Rtucharya, and Sadvrita* judiciously and foremost giving no opportunity for the virus to enter the body. Also, the best use of classics for *Lakshanas* and prevention of further progression of disease helps us grow a better world.

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Source of Support: Nil

Conflict of Interest: None Declared

How to cite this URL: Anisha Nabi & Devi S Nair: Clinical Profile in Covid Wsr Vikruti Pareeksha. International Ayurvedic Medical Journal {online} 2023 {cited April 2023} Available from: http://www.iamj.in/posts/images/upload/827_833.pdf