IJBPAS, April, 2025, 14(4): 1918-1940

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)

'A Bridge Between Laboratory and Reader'

www.ijbpas.com

A CONTROLLED CASE STUDY OF INFLAMMATORY MARKERS WITH NADI TARANGINI AS A SCREENING TEST IN PRANAVAHA SROTAS DUSHTI WITH SPECIAL REFERENCE TO COPD

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Received 16th Nov. 2023; Revised 18th Dec. 2023; Accepted 2nd June 2024; Available online 1st April 2025

https://doi.org/10.31032/IJBPAS/2025/14.4.8838

ABSTRACT

Lung insults due to infections, allergens, dust, and tobacco smoking are the causative factors in lung injury and damage to the inner structures. These permanent changes in Bronchi, Bronchioles, and alveoli result in COPD **[1]**.

Ayurved look at this as Pranavaha Srotodushti janit vyadhi, especially in Shwasa and Kasa vyadhi types. High values of Cytokines (Inflammatory proteins) found in COPD result in a complex process of vascular stiffness due to changes in collagen abnormalities with loss of elastin proteins in vessels. Arterial stiffness is advanced due to an ageing factor resulting in arterial layer damage and arteriosclerosis **[2]**.

Arteries lose elasticity with bad collagen in structures that indicate stiffness. It got triggered due to periodical stress factors either. It was observed that arterial stiffness in different pathological situations and diseases can result in conflicting cardiovascular consequences than other pathophysiological factors **[2]**.

High pulse pressure, Raised Load in the Left Ventricle, and the pressure gradient that moves the coronary perfusion are the most important reasons for Arterial stiffness consequences **[3]**.

The consequences anticipate cardiovascular disease due to Arterial stiffness and both have an association with moderate to severe values of Urine Albumin Creatinine Ratio (uACR) **[4]**.

There is a relation between lower lung function with reduced myocardial perfusion and due to reducing in myocardial function ends in arterial stiffness. Though systemic inflammation doesn't take place earlier it enhances the gap between the heart in the contractile phase i.e., systolic blood pressure and the heart in the relaxing phase still exerts some pressure i.e., diastolic blood pressure is called "Pulse pressure." **[5]**

The raised large arterial stiffness in patients with Chronic obstructive pulmonary disease is associated with higher values in the peripheral systolic and diastolic blood pressures. Many etiological factors may influence arterial tone and raised blood pressure in COPD patients. Hence, it is considered that those COPD patients have raised in Dhamani Kathinya (arterial stiffness), raised Uccharaktachap (Htn.), and other systemic inflammation too **[6]**.

Increased levels of inflammatory Biomarkers, i.e., erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) are associated with cardiovascular mortality and ailments **[7]**.

'PWV' i.e., Pulse wave velocity can be assessed through 'Tonometry' of carotid and femoral arteries. It is denoted as cfPWV i.e., carotid-femoral PWV with the help of ECG as a time difference counter. The cuff-based (oscillometric) method is comparatively genuine in the measurement of the Cardio-Ankle Vascular Index (**CAVI7**) with the help of cuffs at extremities which are coupled with a microphone over the chest **[8]**.

cfPWV and **CAVI7** are both very cost-effective and not used in routine practices. Hence, we need equipment that will be cost-efficient and precise in routine practices to observe Arterial stiffness. Atreya Foundation created the Naditarangini device which is based on photoplethysmography and for prediagnostic tool in COPD for cardiovascular disease assessment with the help of some inflammatory markers common in COPD as well as arterial stiffness. Inflammatory markers and Naditarangini device in routine will save lives in those silent features of Arterial stiffness among youngsters and COPD cases. This article is a trial study for 30 cases in the pre-diagnosis parameter for cardiac blood vascular disease in COPD patients **[8]**.

When we set its normal ranges, linearity was **≤ 50 %** in the two combination groups**.** We got results for **CRP_Class** in Guru Class with Pearson Chi-Square value 4.885a and p-value 0.027 which is the most **Significant.**

But, for further accuracy and more precision we high up a little i.e., Nadi-guna normal ranges linearity were set for **≤ 60 %** in two combinations. We got **Vikriti in Tikshna** class –Value (9.630) p-value 0.039 **Significant.**

The important significance of this research work is that we got each COPD case to stand for higher values in the consistently high Urine albumin creatinine ratio. Hence, it is significant in COPD cases and cannot stand for statistical comparison significance.

Keywords: COPD, Pranavaha Srotas vyadhi, ESR, CRP, UACR, Naditarangini, Nadi-guna, Sira & Dhamani Kathinya

INTRODUCTION

World death statistics in 2022 show that in **Chronic diseases**, Cancer is in 1st place, stroke/ cardiovascular diseases, and chronic kidney diseases are in equal percentage in 2nd place, while septicemia is in 3rd place. The COPD-induced death rate in chronic diseases is in the 4th place after the post-pandemic years **[9]**.

In Ayurveda, COPD relates to Pranavaha Srotas, with their origin and location explained by Charak Samhita as the "Hridaya and Maha-srotas." Gangadhar commentators clear it as "Hridaya and Vaksha" (Chest), i.e., 'Hridaya' means Heart, and 'Phupphusa' means lungs.

In his commentary, he clarifies that the chest-embedded respiratory system and its supportive muscles are the biggest srotas or Maha-srotas. While Chakrapani said that "Vayu" or air passage through "Pranavahanam" throughout the body through a system is called Pranavaha srotas **[10]**.

Tobacco chewing, Gutkha, and Smoking in the form of 'Bidi & Cigarettes' with severe disclosure to dust work, working in congested and non-ventilated kitchens, use of firewood for kitchen stove, dried cow dung cakes in smoky traditional to cook food **[11]**. Allergy to some spices at the time of tempering, some chemicals for cleaning purposes, and other outdoor pollution is going to change alveolar function and cause temporary or permanent damage i.e., COPD **[11]**.

Indoor pollution, biomass fuel gases, and cooking gases due to the improperly vented dwelling are the factors for COPD especially seen in women. Some types of moulds, pollen grains, bacteria, and work farming like feed, bedding particles, fumes, animal hairs, droppings, and feathers can cause organic as well as inorganic inhalation may cause occupational factors for COPD. A childhood history of severe pulmonary infections for a long time or untreated has a strong relationship with adulthood-raised pulmonary symptoms and reduced lung functioning **[12]**.

"Pra" is to carry throughout the body. The end word is "Na" which means through nasal **[13]**. Thus, we can say that "Prana" enters through nasal cavities and spreads throughout the body for life.

"Prana" is the Vayu that passes through Pranavaha srotas. In the Sanskrit dictionary, "Prana" is derived from "**Pra**" as a prefix with "**An**" which means breath, and liveliness **[14]**.

Acharya Sushruta has explained Dwadash Pranas as Vayu, Pitta, Sleshma, and the 03 Manas guna as Sattva, Rajas, and Tamas, with 05 sense organs as Granendriya (Smell), Chakshu and Prana itself contributed term under "Prana". **[15]**

Location of Prana Vayu & its karma: - Prana Vayu which is carried out by Pranavaha Srotas running through Nasika (nose), Jihva (tongue), Murdha (Soft palate), Asya (mouth), Kantha (throat), Urah (chest). It accomplishes like Sthivana (spitting or salivation), Kshvathu (sneezing), Udgara(eructation), Shwasa (respiration), Ahara Praveshkrita (deglutition of food), etc. **[16]**

According to Sushrut Samhita, the origin of Pranavaha srotas is Hridaya (heart) and Rasavahini Dhamanis. Acharya Sharangdhara described Cardiopulmonary blood circulation and its purifications as swasankriya and moolasthana as "Hridaya" in the Pranavaha srotas. According to Sharangadhara, "Prana-Pavana" is located in Nabhi, after leaving "Hritt-Kamala", it comes out through Kantha and mixes with Vishnu-padamruta **[17]**.

Pathophysiology in COPD: - COPD is a continuous process with time along with causative factors of chronic inflammatory illness of the airways which affects alveolar sacs, and small airways. The oxidative stress is due to the presence of permanent changes in the Alveoli and Bronchioles and vascular blood supply due to inflammatory changes and oxidant imbalance.

Neutrophils, macrophages, and lymphocytes release injuries and reveal inflammatory compounds like cytokines, chemokines, and chemoattractants like IL-8 with leukotriene B4 (LTB4). proteinase enzymes such as elastase, proteinase-3, cathepsin G, cathepsin B, and matrix metalloproteinases (MMP) released by polymorphs cause damage to lung tissues with loss of its connective tissue's smoothness **[18]**.

A connective tissue-related large phagocytic cells release cytokines and chemokines such as IL-8, IL-6, IL-10 TNFα, LTB4, etc., and the provoking cells get attracted by reactive oxygen species and activate various.

Proteolytic enzymes in series that breakdown the protein of elastic tissues and elastinolytic thiol proteinases such as cathepsin K, L, and S with mucous membrane pemphigoids like MMP-2, MMP-9, MMP-12, and MMP-14 too **[19, 20]**.

Killer T cell (CD8+) lymphocytes secrete enzymes such as perforin and granzyme B which apoptosis of the alveolar epithelial cells and T helper cell Lymphocytes (CD4) which react to autoimmune reactions to the lung tissue **[21]**.

The change in proteolytic enzyme action on elastin tissue pathologically in loss of lung tissue's elastin flinching pressure. Damage to the Elastin tissue in COPD can end up in stenosis of the airway with a reduction of airflow and retention of air in the lungs. The air retention in good volume in the lungs in the equi-pressure stage is known as 'functional Residual Capacity' (FRC). Pulmonary plethysmography is the option to

measure functional over-breathing due to Functional residual capacity ultimately rising in 'Complementary air' into raising exertion of inhalation helping muscles like the diaphragm and external intercostal muscles work **[22]**.

Brainstem chemo receptor apparatus will reduce in sensitivity due to the power of retaining chronic Carbon dioxide and no more feedback mechanism to the complementary air. Vasoconstriction due to low oxygen levels results in altering the path of blood away from ventilated alveoli **[23]**.

It was hypothesized that an inflammatory spillage from the lungs through blood to other Systemic swelling involvement via thin layered pulmonary vasculature. This pulmonary spillage has a swelling effect on different systemic organs of the body **[24]**.

The level of inflammatory proteins like CRP has been elevated in COPD cases and has been shown a strong relation in various studies [22] with pro-inflammatory cytokines like (TNF)-alpha and IL-6 **[25]**.

Acharya Sharangdhara supports the view in the term "Pranvahe-dve." For that, Prof. Dr B. G. Ghanekar explained that both Phupphusa in the chest cavity are linked to the Hridaya with the help of Pulmonary arteries. It is called as 'Hridayam moolam' where the arteries originating from the heart carry blood toward the lungs through the Lung arteries. In this way, Rasavahini dhamani was also explained regarding towards part of Dhamani where "Pranavahini Dhamani" should be considered as Trachea and Bronchi **[26]**.

Acharya Dr. B.G. Ghanekar further explained that bronchi of the respiratory tract and Kantha nadi as Prana Dhamani should be considered as "Shwasa-vahinis." The part of blood carried out by pulmonary arteries towards bronchioles so that de-oxygenated blood from the heart to the lungs gets mixed with oxygen and goes back to the heart getting oxygenated with "Prana Vayu" by pulmonary veins. This elaboration includes the deoxygenated to oxygenation circulatory action of "Prana Vayu" from the cardiopulmonary cycle. Hence, Charakacharya and Sushrutacharya, both accepted the common factor "Hridaya" as the origin and centre-situated part of Pranavaha Maha-srotas **[27]**.

Prana Origin: - *"Pranamahurmatar Ashwinam Vato ha Prana Uchyate I Prane ha Bhootam*

Bhavya cha Prane Sarva Pratishtitam II Atarvaveda Jeevitaadhaarah II" C. Su. 29/03.

In 'Vedic literature' Prana was explained as liveliness and the base of life in each creature. It is the potential empowerment for each life. The life before breath is Prana, that cause of breath.

Nirukti: - *"Pranaayati iti Pranah iti Pranashabdasya Niruktihi I"* Satik Charak.

"Prana" is acquired from a word of "An" with pre-suffix *"Pra"* and *"ac"* and *"Ghan." Sometimes its basis is as the root "Pr" means "Pranati" or* filling it means *"Piparti" to nurture* by the of "Purna" i.e., fulfil **[28]**.

Definition: - "*Pranavaayoho Jeevitadharatvam Pradhanatayaa Drushtavaa evam Visheshaajjeevitam Pranah etyuktam Vagbhaten I"* A. Hri. Ni. 16/56.

Here, Acharya Vagbhat explained in Sutrasthana chapter 16 that the "Prana-Vayu" is a priority in a specific base for liveliness. No life without *"Prana."*

"Sushrutacharya" in Nidan sthana Chpt. 01 / 13 explained that Vayu moving through Kantha (Throat), Nasika (Nasopharynx) and Murdha (Soft palate) is a Prana. This prana enters through the respiratory system to give empowerment to Hridaya and Agni **[29]**.

While Acharya Gayadasa also explained in his commentary that Prana creates "Avalambanam" i.e., the root of death is the support of life, Pranadhar Hridaya supports **[30]**.

Prana – Rakta Relation: - The inflammatory spillage from the lungs field to other systemic inflammation through blood is a relation of Prana or air inspiration as a (Causative air factor, Polluted air, fumes and Smoke) – through Pranavaha Srotas (Respiratory system) – through blood (Rakta) – Changes in collagenopathy with loss of elastin in inner layers of Vessels (Tunica intima) and to other systemic involvement especially Cardiovascular Circulation through Arteries, vessels get stiffness (Rasa-Raktavahi Dhamanya Kathinya) – involving Hridaya (Cardiac diseases – Valvular &Vascular).

Acharya Charaka in numerous texts has explained 'Vata' [1], 'Anna' [2] and 'Rakta' [3] as nothing but Prana **[31]**. Nyayachandrika Vyakhyakar, Commentator Gayadasa explained in the same chapter (Su. Ni. 01/43.) that an "Agitated Vata" gets prone to a speedily agitate the Rakta / Blood. An aggressive with inner restless Prana/Vayu is going to create pathophysiology of the diseases through the aggravation of Rakta dhatu with systemic involvement **[32]**.

Acharya Charak also explained "Dashapranayatana" or "Dasha Jeevitdhamani" in Sutrasthana Adhyaya 29/03. Where the 2 Shankha marmas, Tri-Marmas as Shira, Hridaya and Basti, Kantha (Vocal box), Rakta (Blood), Shukra (Essence of all dhatu), Oja (Supreme part after Shukra), Guda (Rectal region), but in Sharira sthana he replaced Two Shankha marma by Nabhi (Umbilicus), Mamsa Dhatu (Muscles). Here in Sutra sthana& Sharira sthana "Rakta" itself influence Prana **[33]**.

Sushrutacharya clearly explained the origin of Raktavaha srotas is Raktavahi

Dhamanya. Hence, we can say that Prana is associated with Hridaya and Rasavahi, Raktavahi Dhamanya and Sira too **[34]**.

At the time of the foetal organs developing phase, Lungs are developed from the influence of the foam part of that foetal blood. Here blood and Prana (air) are expected to create a foam of blood but not as vata dosha. A commentary on the same phrase by Dalhanacharya is that smaller round-shaped sacs holding air i.e., alveoli are occupied at the bronchioles terminals of the lungs and are connected to the heart with blood-carrying vessels **[35]**.

"रक्तफेनात् फुप्फुसम् ||१||" A. Sam. Sha. 05/46 (01) (Vriddha Vagbhata) - The same part is explained by "Vriddha Vagbhata" in Ashtanga Sangraha Sharir sthana – Chapter number 05 after 46 phrases followed in 1st phrase that the Lungs are generated by "Rakta phena" i.e., a combination of blood & Prana into small bubbles **[36]**.

In addition, in the same chapter from Ashtanga Sangraha, Rakta (Blood) combines with Prana Vayu (Anilah) to create an iron mix of the reddish-black colour of foetal lungs **[37]**. "रक्तस्यानिलयोगात् कालीयम् ||१||" A. Sam. Sha. 05/46 (01) (Vriddha Vagbhata.)

In Shabdakalpadrum, "Kaliyakam" is a kind of Brick red colour Sandalwood.[38] And Kālīyam कालीयम् [काल-छ] **"A kind of sandal-wood"**; also, Kālīyak कालीयक **[39]**.

Hridaya – Prana – Rakta – Relation: - Acharya Sharangdhara explains the respiration function in association with Prana vayu as "**Pavana**" that goes out to carry **"Ambar Piyush"** as a part of circulation from the lungs to the heart **[40]**. Hridaya is formed from Shonita (Blood) Kapha prasad and it is the ashraya for "Pranavaha-Dhamani." **[41]**

Vriddha Vagbhatacharya explained in Sharira sthana 5th Chpt. that hridaya is made up of the supreme part of 'Rakta' (Blood) and Shlesha (Kapha) where it is hollow in a structure like hanging lotus bud-like shape **[42]**.

Sushrutacharya explained that Hridaya is made up of the supreme part of Kapha dosha and Rakta dhatu in foetal phases. Dhamanis (Arteries) are associated with hridaya. Hridaya is associated with Pranavaha avayava (Prana-Phupphusa-Prana Dhamani). It is shaped like a lotus bud tilted towards the left side of the chest with its apex facing downward and relates to Kloma. Acharya Gananath Sen ji explained this **"Kloma"** as Trachea i.e., heart direct relation with Pranavahi nadi & Raktavahi Dhamanya **[43]**.

Acharya Charak explained in 8th Chpt. of Nidana sthana that *"Nidanarthkaro Rogo Rogasya apyuplabhyate |"* i.e., one disease can cause as an etiological to other diseases. Due to the Santap laxana of Jwara (fever), it may manifest as Raktapitta (Bleeding

disorder), Raktapitta with Jwara can cause Shosha (Tuberculosis). "Shwasaschapup Jayate|" – In the same verse, Chakrapani Datta commentator explained that this shisha should be considered as **Shwasa** vyadhi instead of "Shosha" vyadhi **[44]**.

Metabolic Syndrome (A bunch of any three conditions from): -1. Central obesity, 2. Hypertension, 3. Hyperglycemia, 4. Hypertriglyceridemia, 5. HDL low level in the serum with Specific cytokine patterns in association with Arterial ageing, Raised Arterial stiffness, and Inflammation on the Arterial wall. The Arterial wall thickness will be greater when both Metabolic Syndrome and High levels of cytokine are present **[45, 46]**.

Here, Rasavahini Dhamanya in Rasavaha srotas and Raktavahi Dhamanya in Raktavaha srotas get deranged with Vayu, as we discussed earlier. It creates discolouration, twitching, dryness and numbness-like sensation symptoms, as well as a pricking sensation and cracks over the skin when it enters Rasagata in skin symptoms. But this vitiated prana, when deranged in Raktavahi Dhamanya, results in ulcerations. This dominant vata enters Sira and then creates severe pain, sira spasms, stiffness and constriction with varicosity. It is all because of Dhatugata prana vitiation explained by Sushrut in 1st chapter of nidanasthana **[47]**.

Gayadasa commentator explained in the same chapter in phrase 30, that the Prana-vayu gets vitiated and upset to unbalance the dhatu formation basic principles process in the body progressively from the lower and upper limbs, head and spread all over the body in the form of dysfunctions. Those comorbidities can be identified as numbed (paralysis), spasmodic straining of the limbs (Aakshepa), and numbness. The whole body shows intermittent pains (Shula), and tenderness (Shotha) too **[48]**.

In Ayurveda, Shwasa vyadhi is a disease of **Pranavaha srotas due to Kapha dosha vitiation** and the **Premature arterial stiffness is the same due to Kapha dosha dominancy** induced. That will be a prior state or start with Shwasa Vyadhi Upadrava **[49]**.

"Gayadasacharyastevam mandate Prananam Agnishomadinamvalmban vachachnenaadhar Bhoota Hridayavalambanen maran moolatvamuchyate, Eten Pranadhar hridayadhaaranen pranadharan mevoktam | Ata eva prananamavalambanen maranmulatvamuchyate |" Nibandha Sangraha Satika Su. Ni. 1 - 13 page 259.

Acharya Gayadas explained that Prana is a liveliness of Agni dependency and it is an adaptation of "Cardiac function." Prana is life-supporting and holding the heart. Hence, the Prana influencing through the

heart and its pathology may result in death **[50]**.

Shwasa is a disease of Kapha-Vatatmaka. Its doshas vitiation is associated with Pitta dosha in Aamashaya (Stomach). This pitta in shwasa vyadhi tries to dry out cardiac-related Rasadi Dhatus and Hridaya itself. Hence, in Ayurved text, it is mentioned that the phase of dryness will create Shwasa, and Hridroga in an early phase but it is Khariya-bhotatva with the end in the mature phase of Arterial stiffness. This one is an incurable phase and if there is delay or improper treatment will end up in sudden death like a poisonous snake bite **[51]**.

"Kaphavatatmakaavetau Pittasthansamudbhavau I Hridayasya Rasadeenam Dhatunam chopshoshanau II8II Tasmaat Saadhaaranaavetau matau Paramdurjayau I Mithyopacharitau Kruddhau hatt Aasheevishaviva II9II C. Chi. 17/8 -9.

According to Charaka Samhita, Vaivarnyata (Discoloration), Moorcha (Fainting), Jwara (Fever), Kasa (Cough), Hikka (Hiccup), Shwasa (Exacerbation), Asyavairasya (Change in taste), Trishna (Thirst), Pramoha (Stupor ness), Chardi, Kaphotklesha (Mucous production), Ruja and Aruchi is the Samanya Lakshanas of Hridroga **[52]**.

"Arthe [1] Dasha Mahaamoolah Samaasaktaa [2] Mahaphalah I Mahacchaarthashchya Hrudayam Paryayairuchyate Budhaih II" C. Su. 30/3-4. Satika.

In Charak Sutrasthana chapter number 30-Arthe -dashmahamooliya adhyaya, where Charakacharya explained **"Arthe"** means Hridaya with its origin in ten great vessels. 'Mahat' and 'Arthe' are synonyms for Hridaya.

"Dhmaanaaddhamanyah Sravanaatsrotaansi Saranaatsirah II12II" C. Su. 30/12.

In the same chapter, Charakacharya differentiates between Sira and Dhamani. The pulsatile vessel is called Dhamani while outflowing due to the pulsatile heart i.e., **"Sravana"** conducting are Srotas while the vessels that carry the contents forward (Sarana), are called Sirah **[53]**.

According to Sushruta, 'sira' originates in the embryogenic phase of life. It spread from the nabhi (umbilical) and then spread upward, downward and in an oblique fashion from the nabhi **[54]**.

Hence, Dhamanis as well as Siras hardening is a strong anticipator for the heart (cardio) and the blood vessel-related events of MI that cause mortality **[55, 56]**.

A rise in the heart disease and its circulatory blood vessels related to the stiffness – in a relative phase and death has an association with a rise in Biomarkers for inflammation such as ESR, CRP, and IL-6 **[57]**.

Aim – Pranavaha Srotojanit Vyadhi study to rule out cardiovascular disease with the help

of Nadi-Guna and inflammatory markers in COPD.

Null hypothesis – Pranavaha srotas dushti-related cardiovascular diseases cannot be screened out with the help of a pulse acquisition system and laboratory inflammatory markers for the vitality and futurity in Pranavaha moola sthana vikriti as Phuffusa – Hridaya.

Alternative Hypothesis – Pranavaha srotas dushti-related cardiovascular diseases can be screened out with the help of a pulse acquisition system and laboratory inflammatory markers for the vitality and futurity in Pranavaha moola sthana vikriti as Phuffusa – Hridaya.

Sample size & Formulae – Sample Size is = 144., Prevalence rate is = 32%.

The formula i $n = \frac{z^2 \cdot p \cdot q}{E^2}$ ara, Gujarat, India especially. (2yrs cases frequency conducted at CTRI-mentioned hospitals.)

Inclusion Criteria – 1. There will be no consideration of the gender or religion of the patients. 2. Age from 18 to 70 years. will be included as a standard parameter. 3. Emphysema, Chronic Bronchitis, Tuberculosis, post-COVID-19 complications, Chronic Cigarette smoking, Pulmonary fibrosis (Scarring and thickening of lung tissues), and Occupational lung disease are included under one term COPD i.e., chronic obstructive pulmonary disease.

Exclusion criteria – 1. Patients under 18 years, and above 70 years shall be excluded from the study. 2. Lung cancer, Pulmonary hypertension.3. Lung Transplant, Trauma. 4. Lungs Aspirate (Vomits). 5. Cystic fibrosis (Genetical disease). 6. Pleural effusion (Non-Infectious, Malignancy). 7. Depression-induced ARDS.

Objective parameters– Primary investigations will be assessed in diagnosed COPD cases with exacerbation and low Oxygen Saturation on Pulse-Ox meter as symptoms.

1. ESR (By Westergren method) with normal value M≤15mm/hr., F≤20mm/hr. By using an EDTA/K_2EDTA bulb for blood sample collection.

2. CRP Quantitative method. The normal range is ≤3mg/L in both genders, by using a plane bulb for blood sample collection.

3. A Pulse Acquisition system based on Photoplethysmography will be used for the Radial pulse (Arterial)based on the Nadi-Guna record. Normal range ≤ 60% By using Atreya Naditarangini Yantra.

4. UACR (Urine Micro-Macro Albumin in mg. /Urine creatinine in gm.) test for significance in Nadi Guna changes in respective to Arterial stiffness. In all age groups after calculation. By using a dipstick urinalysis test.

Category 1 Normal = In M ≤ 2-20 mg/Gm. In F ≤ 2.8-28 mg/Gm.

Category 2 Moderate Raised = 30 to 299 mg/Gm.

Category 3 Severe Raised = ≥ 300 mg/Gm. As an Inflammatory marker for Arterial stiffness too.

The urine albumin-to-creatinine ratio (UACR) is associated with arterial stiffness and can be predicted with photoplethysmography for its endothelial dysfunctions in vessel walls to rule out cardiovascular diseases in any age group apart from gender **[58-62]**.

coronary artery disease (CAD), chronic heart failure (CHF), or chronic obstructive pulmonary disease (COPD) occur commonly in the presence of each other and are associated with similar systemic inflammatory reactions under the term "Cardiopulmonary continuum". **[63]**

Plan of Study – 1. Chronic obstructive pulmonary disease (COPD) diagnosed patients under the ayurved term Pranavahasrotasa impaired have the most important symptom of "Exacerbation" with the need for oxygen correction will be considered for case data **[1]**.

2. The patient's case proforma with other Srotas dushti (Systemic involvement) was recorded with all vital parameters.

3. Nadi Guna was assessed properly as per the Photoplethysmography Instrument's manufacturer's instruction.

4. Patients' data to rule out "Deha prakriti" was filled in inbuilt software of the purchased Photoplethysmography Instrument.

3. Patients' laboratories investigation for inflammatory markers like CRP, ESR & Urine micro albumin and urine creatinine (for uACR value) was derived.

Statistical study for trial cases: - We have conducted 32 COPD cases with the above-mentioned inclusion and exclusion criteria with symptoms of exacerbation category at the time of hospitalisation. Pearson correlation (Pc), p-value (p-v) and results among significant Nadi guna in **normal range ≤ 60 %** in inflammatory markers as…

CRP mg/L – 1. Guru Nadi guna as Pc (0.166), p-v (0.372) result (NS), 2. Kathina Pc (0.130) p-v (0.485) result (NS), 3. Sthula Pc (0.210), p-v (0.256) result (NS), 4. Tikshna Pc (-0.156), p-v (0.401) result (NS).

UACR mg/gm – 1. Guru Nadi guna as Pc (-0.085), p-v (0.651), 2. Kathina Pc (-0.164) p-v (0.377), 3. Sthula Pc (-0.111), p-v (0.552) 4. Tikshna Pc (-0.008), p-v (0.968), For all result (NS).

ESR mm/hr – 1. Guru Nadi Value (0.030a) p-value (0.863), p-v 2side (1.000), p-v 1side (0.589), 2. Kathina Nadi guna Value (0.194a) p-value (0.660), p-v 2side (1.000), p-v 1side (0.563),3. Sthula Nadi guna Value (1.288a) p-value (0.256), p-v 2side (0.386) p-v 1side (0.256), 4. Tikshna Nadi guna Value (2.358a) p-value (0.125), p-v 2side (0.235) p-v 1side (0.127) For all result (NS).

Tridosha in the body in a state of equilibrium is considered as Prakriti or "Swastha Avastha." Where the Vata, Pitta, and Kapha in the raised or lowered phase in the body can be considered as abnormal state i.e. vikriti Avastha **[64, 65]**.

The Vikruti in the body can be analysed with the help of its current status without any more Pathological investigation. Pulse or Nadi can assess it for recent changes in Dosha, Dhatu, and Upadhatu **[65]**.

Prakriti in different Nadi Guna & Inflammatory classes with Fisher's Exact Test value & Significance (Sig.): -

Prakriti in Guru Nadi Guna class–Value (5.491), Exact Sig. (2-sided)0.370 (NS),

Prakriti in Kathina Guna Class –Value (7.161) Exact Sig. (2-sided)0.146(NS),

Prakriti in Sthula Class –Value (3.278) Exact Sig. (2-sided)0.736(NS),

Prakriti in Tikshna class–Value (2.989) Exact Sig. (2-sided)0.864(NS),

Prakriti in UACR Class –Value (7.431) Exact Sig. (2-sided)0.344(NS),

Prakriti in CRP Class –Value (2.095) Exact Sig. (2-sided)0.964(NS),

Prakriti in ESR Class –Value (3.298) Exact Sig. (2-sided)0.738(NS),

Vikriti in different Nadi Guna & Inflammatory class with Fisher's Exact Test value & Significance (Sig.): -

Vikriti in Guru Nadi Guna class –Value (5.111) p-value1.000(NS),

Vikriti in Kathina Guna Class –Value (6.511) p-value 0.657(NS),

Vikriti in Sthula Class –Value (9.286) p-value 0.160(NS),

Vikriti in Tikshna class –Value (9.630) p-value 0.039 **Significant.**

Vikriti in UACR Class –Value (11.222) Exact Sig. (2-sided) 1.000(NS),

Vikriti in CRP Class –Value (4.884) Exact Sig. (2-sided) 0.807(NS),

Vikriti in ESR Class –Value (5.363) Exact Sig. (2-sided) 0.866(NS),

We lower the Nadi Guna normal range ≤ 50 % in inflammatory markers in Prakriti & Vikriti as in Pearson correlation (Pc), p-value (p-v).

We got no significance in another test except **CRP_Class** in Guru Class with the Pearson Chi-Square value 4.885a and p-value 0.027 which is the most **Significant.**

A "Urine Albumin Creatinine Ratio" (UACR) value in each COPD case stands for moderate to severe changes i.e., continuous abnormal values, none have a normal range hence statisticians cannot compare its significance in other comparison groups. Hence, UACR cannot stand for statistical comparison significance but is still very significant in COPD short-trial cases. But in actual prevalence study in comparison with the controlled group may stand for its relations.

DISCUSSION:

Collagen and elastin are the two main scaffold proteins in the vessel wall that maintain its elasticity and are well-functioning. These protein molecules are in the dynamic process of degradation with lung insults, infections, tobacco smoking, lifestyle-induced dyslipidaemia, and metabolic disorders. All these etiological factors in the body create an inflammatory chain and the atmosphere in the body may start from any part of the body, especially the lungs. That triggers the overproduction of abnormal collagen which may alter the qualities of the body's normal collagen and its normal elastin properties may end up in vascular stiffness all over the body **[66]**.

Acharya Vaghbata explained in Ashtanga Hridayam in Sutrasthana chapter - 01 for Ayushkamiya Adhyaya in "Gurvadi Guna." Where the guru stands for heaviness, Manda is slowness, Hima is a chilling effect, Snighdha for oleaginous, Slakshna for sliminess, Sandra for denseness, Mridu for a soft feel, Sthiratva for steadiness, Sukshma for the fineness, Vishada for clearness and non-slimy, and its antonyms are Laghu for delights, Tikshna for sharp and penetrating, Ushna for warmness, Rukshatva for skimmed, Khara for coarseness, Dravata for fluidity, Kathinya for hardness or solidity, Chala for motion and restlessness, Sthula for bulkiness, and Piscchila for stickiness. These twenty qualities or gunas play a crucial role in the assessment of health or diseased conditions and qualities of doshas **[67]**.

This study is also associated with raised arterial stiffness in COPD patients who have a tobacco smoking history though the patient gets managed for the age, mean BP and BMI. The study reports showed mild COPD is not associated with metabolic secondary diseases or cardiovascular disease. As the study raised the Nadi Guna **normal range ≤ 60 %** we got **"Tikshna Nadi Guna"** more significant with **Vikriti Dosha** relation in COPD **[68]**.

From Ayurveda's point of view, Shwasa vyadhi is a disease of **Pranavaha srotas due to Kapha dosha vitiation** and the **Premature arterial stiffness is the same due to Kapha dosha dominancy** induced Upadrava.[69] Hence, Shwasa vyadhi in the Primary phase without any other srotas involvement (no comorbidities) can be stated in the phase of normal range calculation for Nadi Guna ≤ **50 %**of Significance in **"Guru Nadi Guna"** findings in small data with inflammatory markers related to "**CRP.**" **[69]**

The UACR values raised with those COPD cases group that have severity in symptoms in time durations. There is a significant difference in UACR among different groups of COPD especially with C-reactive protein (CRP), and Nadi "Guru" and "Tikshna" guna **[70]**.

CONCLUSION:

This trial study, though it has a small sample size. Each COPD case shows a moderate to severe Urine albumin creatinine ratio. Hence, it cannot be compared and showed statistically significant. Many cases showed **Nadi Kathinya** with a higher or lesser percentage than other Nadi Guna.

Shwasa vyadhi is a disease of **Pranavaha srotas due to Kapha dosha vitiation** and the **Premature arterial stiffness is the same due to Kapha dosha dominancy** induced as Upadrava. In early COPD cases with no other srotas involvement (Systemic involvement) having kapha dominancy in Nadi Guna in hetus of Shwasa vyadhi, and its main samprapti (Pathophysiology) component Nadi Guna **≤ 50 %** of Significance in the CRP_Class in **Guru Nadi Guna** Class with Pearson Chi-Square value 4.885a and p-value 0.027 which was the most **Significance** inflammatory markers related to **"CRP."**

But when **Pitta** dosha dominancy takes place with Kapha dosha in a little more period in those COPD with its causative factors vitiating pitta gets a little faster and sharp in Nadi Guna recorded statistically as – Vikriti in **Tikshna Nadi Guna** class – Value (9.630) p-value 0.039 **Significant.**

We hope in a large sample size, we will find furthermore pitta dosha activity in association with vata dosha, which may turn Kaphakar samprapti components into further pakatva to Kharatva of Sira-Dhamani Kathinya will be a screening diagnostic part in COPD cases.

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