Section: Respiratory Medicine

Original Research Article

CORRELATION OF SIT TO STAND TEST AND TWO CHAIR TEST WITH SIX MINUTE WALK TEST IN THE FUNCTIONAL ASSESSMENT OF COPD PATIENTS

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Abstract

Background: The third most frequent global cause of death is chronic obstructive pulmonary disease (COPD). In terms of public health; chronic obstructive pulmonary disease (COPD) is a significant issue and the fifth-highest burden of disease by the year 2020. Therefore, the purpose of the current study is to assess functional status using the STST and two chair test with the 6MWT in patients with COPD and to compare the results of both tests based on a number of patient outcome parameters (pulmonary function, dyspnea severity, hemodynamic stress, quality of life, and peripheral muscle strength), in order to determine the utility of the STST for these patients. **Materials and Methods:** The present study was an Observational study. It involves patients presenting with symptoms suggestive of chronic obstructive pulmonary disease attending the pulmonary medicine clinic and/or admitted in pulmonary medicine clinic and/or admitted to pulmonary medicine ward in Mahatma Gandhi Medical College and Research Institute, Pondicherry. A total of 68 patients were enrolled in the study and their clinical assessment (mMrc grading, SpO2, BP), functional assessment (spirometry), and six-minute walk test, sit to stand test ad two chair test were done. **Result:** The mean ages of the patients in Groups 1 and 2 were 73.5±26.5 and 76.5±23.5 years. On comparing hemodynamic parameters before and after performing STST, 2CT and 6MWT is not statistically significant. **Conclusion:** In conclusion, STST and 2CT is less time consuming, repeatable and easy to perform. It determines the functional status of COPD patients is similar in comparison of STST and 2CT with 6MWT. The functional capacity can be assessed through STST and 2CT instead of 6MWT.

INTRODUCTION

The third most frequent global cause of death is chronic obstructive pulmonary disease (COPD).[1] In terms of public health; chronic obstructive pulmonary disease (COPD) is a significant issue and the fifth-highest burden of disease by the year 2020. Smoking cigarettes, working in environments with smoke, dust, or gas, or being exposed to biomass fuel in poorly ventilated interior spaces are all risk factors for the disease.[2] Airway obstruction and other systemic illness are linked to the chronic inflammatory disease and chronic obstructive pulmonary disease.[3] One of the key characteristics of COPD is a lack of exercise owing to peripheral muscle weakness with lung diseases.[4] Therefore, in this group of patients, functional status assessment is crucial for prescribing the right therapy and rehabilitation plans.[5]

Spirometry is a useful tool for diagnosing conditions, assessing severity, and initiating treatment. Several tests that are currently available can be used to determine functional exercise capability. The six-minute walk test (6MWT) is one such test and a good indicator of functional status.[6] In comparison to previous cardiopulmonary exercise tests, this one is well tolerated, simple to administer, and more indicative of daily activity.[7] Being able to stand up from a seated position is necessary for many other daily activities. For the same justification, STST has been accepted as a functional status indicator for aged persons. For elderly persons and those with impairments, being able to rise up from a chair is important for maintaining independence.[8] Two chair test also used to evaluate the functional status of the patients.

From an epidemiological perspective, men have historically smoked more than women, increasing their chance of acquiring COPD. However, over time and depending on the nation, smoking by women appears to be associated with a similar risk of acquiring COPD as smoking by men. There is considerable controversy surrounding the question of whether women who smoke similarly to men increase their risk of acquiring COPD.[9,10] Depending on age, infections appear to have a significant impact on the development of COPD. Childhood illness exposure may change a child's ability to breathe.[11] In adults, recurrent viral or bacterial exacerbations may also be a factor in the deterioration of lung function.[12] Early life disadvantage was associated with a persistent reduction in lung function, no catch-up in lung function decline with age, but a somewhat larger drop with time, and a significantly increased risk of COPD. The effects of childhood poverty were equivalent to those of heavy smoking.[13]

Therefore, the purpose of the current study is to assess functional status using the STST and two chair test with the 6MWT in patients with COPD and to compare the results of both tests based on several patient outcome parameters (pulmonary function, dyspnea severity, hemodynamic stress, quality of life, and peripheral muscle strength), to determine the utility of the STST and 2CT for these patients.[14]

Aims and Objectives

AIM:

To correlate sit to stand test and two chair test with six minute walk test in the functional assessment of COPD patients.

Objectives:

- To perform 6MWT, STST, and two chair test in COPD patients.
- To compare STST and two chair test with 6MWT.

REVIEW OF LITERATURE

Patients with COPD develop exercise intolerance as a result of their dyspnea, in addition to prolonged respiratory symptoms. In order to improve the direction towards pulmonary rehabilitation, measurement of exercise tolerance is an essential component of the diagnosis and management of this disease.[15,16] Various testing techniques are used globally at various health centers, ranging from sophisticated equipment with limited accessibility, such as lung function laboratory, to more accessible and less sophisticated instruments referred to as "field-testing".[17,18]

EPIDEMIOLOGY OF COPD:

GENETIC FACTORS:

Since not all smokers who smoke acquire COPD in their lifetimes, even though smoking cigarettes is the biggest risk factor, it is possible that hereditary variables are also in effect. 19 Typically, it would seem that a child's respiratory system is influenced by the respiratory condition of both of its parents. Thus, 37% of children whose parents have a slightly low respiratory function will also have a low respiratory function. On the other hand, 41% of children whose parents have normal or high respiratory function will have a normal function.[20]

Currently, the only known genetic causative factor for the PiZZ phenotype is a significant deficiency in a1-antitrypsin. This deficiency affects 1-3% of COPD patients and manifests as pan lobular emphysema in the clinical picture.[21] The SERPINA 1 gene, which regulates the production of a1-antitrypsin, appears to contain simple nucleotide polymorphisms in six of its haplotypes. As a result, this gene is regarded as a significant contributing factor to COPD.[22]

Occupational Exposure

According to estimates, the risk of COPD associated with occupational exposure is 19% for smokers and 31% for nonsmokers. The main exposure sites for non-smokers are the rural environment, where individuals are exposed to a high level of organic particles (vegetable dust, bacterial or fungal toxins), and the textile industry, where people are exposed to a high level of plant dust (such as cotton dust), and the industrial environment (mining, smelter plants, the iron and steel industry, the wood industry, and the building trade).[23] It is still unappreciated how occupational exposures, and in particular how these factors may interact with cigarette smoking, cause COPD. The risk of COPD has been observed to significantly rise when occupational variables and smoking are combined.[24]

Air Pollution

Due to exposure to smoke when cooking or the way of heating in inadequately ventilated buildings, particularly for females, there is a significant risk of getting COPD (risk accounts for 35% of cases). This risk is particularly high in nations that are developing. In comparison to females living in urban environments who are not thus exposed, the prevalence of COPD is considered to be three times greater in rural places in China.[25] Uncertainty surrounding the contribution of air pollution to risk factors. Its effect as an exacerbating factor has been demonstrated in people with the most severe types of COPD during air pollution peaks.[26-28] An extensive multi-city research of elderly people who left the hospital alive after receiving treatment for COPD recently found a significant impact of long-term exposure to airborne particles on the probability of death.[29] In epidemiology investigations, it is essential that environmental measurements be accurate, and reconstructing individual exposure using traffic-related air pollution dispersion models is a significant problem.[30]

AGE:

The prevalence of COPD increases with age.[31] A physiological deterioration in respiratory performance throughout the life of an individual starts to occur around the age of 30 to 40.

Bronchial hyperactivity:

Smokers with asthma are more likely to experience deterioration in respiratory function than non-asthmatic patients.[32]

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Social and economic factors: After accounting for smoking, populations in underprivileged social and economic circumstances had a higher chance of acquiring COPD.[33]

Diagnosis of COPD: Patients who have a history of exposure to risk factors for COPD, dyspnea, persistent cough or sputum production should be evaluated for the disease. A post – bronchodilator FEV1/FVC < 0.70 indicates the presence of persistent airflow limitation and consequently, COPD in patients with relevant symptoms and substantial exposures to noxious stimuli. Spirometry is necessary to make the diagnosis in this clinical situation. The most accurate and consistent way to detect airflow limitation is by spirometry.[34] Peak expiratory flow measurement provides a relatively high sensitivity, but its specificity is sufficiently low that it cannot be used as a standalone diagnostic test.[35,36] For the diagnosis of COPD in primary care, the WHO has established a limited number of interventions.[37]

Grades	Intensity	Airflow limitation
GOLD 1	Mild	FEV1 ≥80% predicted38
GOLD 2	Moderate	50% ≤FEV1 <80% predicted
GOLD 3	Severe	30% ≤FEV1 <50% predicted
GOLD 4	Very severe	FEV1 <30% predicted

When deciding on a treatment strategy for COPD patients it is important to consider the degree of airflow obstruction, intensity of symptoms, any past exacerbations, and the existence of co-morbidities.[38]

Pathogenesis: Chronic inflammation in the parenchyma, pulmonary vasculature, and airways is a characteristic of COPD. There is an increase in macrophages, neutrophils, and T lymphocytes (mainly CD81) in different areas of the lung. Numerous mediators, which can damage lung structures or maintain neutrophilic inflammation, are released by activated inflammatory cells. These mediators include leukotriene B4 (LTB4), interleukin-8 (IL-8), tumor necrosis factor-a (TNF-a), and others. Two other mechanisms that are considered to possess a significant role in the pathophysiology of COPD, in addition to inflammation, are an imbalance between the proteins and antiproteinases in the lung and oxidative stress. Exposure to inhaled toxic particles and gases results in lung inflammation. Smoking cigarettes can directly harm the lungs and cause inflammation.[39-44] Even though there is less evidence, it's likely that additional risk factors for COPD start an identical inflammatory process.[45-49] It is considered that COPD may then result from this inflammation.

Pathology: The parenchyma of the lung, peripheral airways, central airways, and pulmonary vasculature all exhibit pathologic alterations that are typical of COPD. Inflammatory cells penetrate the surface epithelium of the central airways, including the trachea, bronchi, and bronchioles, with an interior diameter of more than 2 to 4 mm.[50,51] Mucus hypersecretion is associated with enlarged mucus-secreting glands and an increase in goblet cells. Chronic inflammation causes the airway wall to repeatedly be damaged and repaired in the peripheral airways, which are small bronchi and bronchioles with an internal diameter of less than 2 mm.[52] Due to increased collagen content and the development of scar tissue, the repair process causes the airway wall to structurally restructure, narrowing the lumen and resulting in permanent obstruction of the airways.[53] In patients with COPD, lung parenchymal destruction usually manifests as centrilobular emphysema. The respiratory bronchioles are dilated and destroyed in this process.[54] These lesions are more common in the upper lung regions in milder cases, but in more severe stages of the disease, they can also entail the loss of the pulmonary capillary bed and show diffusely throughout the entire lung. One of the main hypothesized mechanisms behind emphysematous lung damage is an imbalance of endogenous proteinases and antiproteinases in the lung caused by hereditary factors or the action of inflammatory cells and mediators. Furthermore, another effect of inflammation could be oxidative stress.[55] A thickening of the artery wall that starts early in the disease's natural course is a characteristic of pulmonary vascular alterations associated with COPD. The initial anatomical alteration is the thickening of the intima, which is followed by an augmentation of smooth muscle and the infiltration of inflammatory cells into the artery wall.[56,57] The vessel wall becomes thicker as COPD develops due to increased levels of smooth muscle, proteoglycan, and collagen.[58]

Pathophysiology: The disease's pathologic changes in the lungs cause associated physiological changes, including mucus hypersecretion, ciliary dysfunction, airflow limitation, pulmonary hyperinflation, abnormalities in gas exchange, pulmonary hypertension, and cor pulmonale. Throughout the course of the disease, they frequently manifest in this order. Sputum production and a persistent cough are caused by ciliary dysfunction and mucus hypersecretion. Before further signs or physiologic abnormalities appear, these symptoms may persist for a long time. Before any other signs or physiologic problems develop, these symptoms may persist for years. Spirometry is the most accurate method for measuring expiratory airflow limitation, which is the primary physiologic alteration associated with COPD and is essential for making a diagnosis. The main cause of it is persistent blockage of the airways, which raises airway resistance. The loss of alveolar attachments, which prevents small airways from staying open, is less significant.

Advanced COPD results in reduced lung capacity for gas exchange, which leads to hypoxemia and eventually hypercapnia. These factors include peripheral airway obstruction, parenchymal damage, and pulmonary vascular anomalies. The primary cardiovascular consequence of COPD is pulmonary hypertension, which generally appears delay in the

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course of the disease (Stage III: Severe COPD). It is linked to the development of cor pulmonale and an adverse prognosis.[59] It is currently unclear how often cor pulmonale is in COPD patients and how it develops naturally.

In 2006, S. Ozalevli et al conducted a study where Fifty-three patients with stable COPD (mean forced expiratory volume in 1 s (FEV1) 46±9% predicted, mean age 71±12 year) and 15 healthy individuals (mean FEV1 101±13% predicted and mean age 63±8) were included. Similar to 6MWT, STST is capable of accurately determining the functioning state. Additionally, compared to the 6MWT, it generates less hemodynamic stress. In conclusion, patients with COPD may substitute the STST for the 6MWT.[60]

In 2019, Sayid Tabish Rehman et al conducted a cross-sectional study of 100 mild to severe COPD patients. The COPD Assessment Test (CAT), the STST, and the 6MW Test were all administered and compared. When the association Coefficient Test was used, a moderately positive association between the Sit to Stand Test and the 6-Minute Walk Test Distance was discovered(r=0.71, p=0.0005). The Sit to Stand Test and the Chronic Obstructive Pulmonary Disease Assessment Test score showed a slightly positive connection in these patients (STST and CAT r=0.46, p=0.011). The distance covered in the 6-minute walk test and the score on the Chronic Obstructive Pulmonary Disease Assessment Test also showed a moderately positive correlation(r=0.58, p=0.001). The functional capacity can be evaluated in COPD patients using STST rather than the 6 MW Test, with the same outcomes, has been found.[61]

In 2015, Mijid Meriem, et al conducted a study of 49 patients with stable COPD (mean age 67.06 ± 8.4 years, mean forced expiratory volume in the first second 46.25% ± 19.64%), 6MWT and STST were correlated with each other (r = 0.47, P = 0.001). Similar to the 6MWT, the STST can assess functional status in COPD patients. In comparison to 6MWT, it also takes less time and results in less hemodynamic stress. In patients with COPD, STST may be utilized as an alternative to 6MWT.[62]

In 2013, Sarah E Jones et al conducted a study of 50 COPD patients, the test-retest and inter observer reliability of the 5STS were evaluated. The estimated MCID of 1.7 s for the 5STS indicates that it is dependable, valid, and responsive in COPD patients. It is a useful functional outcome measure that may be applied in the majority of healthcare settings.[63]

In 2008, C.G. Cote et al conducted a study of 1379 COPD patients and evaluated the baseline 6MWD in meters for its ability to predict outcomes, and the 6MWD work as a percentage of predicted values the 6MWD in meters according to two reference equations. The threshold values were 350 m for the 6MWD, 25,000 kg m¹for the 6MWD work, and 67 and 54% predicted for the two reference equations, respectively. All testing modalities were comparable in predicting COPD mortality and had strong correlations with the 6MWD test. In conclusion, mortality in chronic obstructive pulmonary disease is predicted by all testing modalities equally well. A value of 350 m in the 6-minute walk distance test is linked to an increased risk of mortality and should be considered abnormal.[64]

In 2018, Qin Shang et al conducted a study of 128 patients with COPD. The 5STS is comparable to the 30STS in terms of sensitivity and specificity as a main screening test for predicting poor 6MWD, but the 5STS provides a better patient experience.[65]

MATERIALS AND METHODS

The present study was an observational study of 80 patients. The study involved patients presenting with symptoms suggestive of COPD attending the outpatient department of pulmonary medicine and/or admitted to pulmonary medicine in Mahatma Gandhi Medical College and Research Institute, Pondicherry.

Sample size: The effectiveness between the two procedures was assumed a moderate value of 0.7, alpha =0.05, power =80%, 68 was taken as the sample size for the study.34 samples for each group.

Here,

Z1-α/2 = 1.96,

Z1-β = 0.84,

d = 0.7 then,

Using the sample size formula,

$$n=\left(\frac{1+r}{r}\right)\frac{\left(Z\left(1-\frac{\alpha}{2}\right)+Z\beta\right)^2}{d^2}+\frac{Z(1-\alpha/2)}{2(1+r)}$$

Statistical analysis: Quantitative variables were described with mean and SD between the two groups will be compared using an unpaired t-test. Qualitative variables were described with frequency and percentage, comparison between the two groups was done by a chi-square.

Study duration: 6 months

Inclusion criteria:

- Spirometry – Post FEV1/FVC <0.7 with no reversibility
- Age more than 18 years
- Consented to participate in the study

Exclusion Criteria

- Patients with other respiratory diseases (Bronchial asthma, Active pulmonary Tuberculosis, Bronchiectasis, Interstitial lung Disease)
- Recent myocardial infarction / Cor Pulmonale
- Resting heart rate >120/ min
- Patients with Systolic blood pressure more than 180mm of Hg and Diastolic blood pressure more Than 100mm of Hg
- Patients with respiratory failure
- Patients with oxygen support or ventilatory support.
- Orthopedic pathology with difficulty in walking
- Not consented to participate in the study

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Study Procedure

Sit to stand test

The patient was instructed to stand up and sit in a chair for 1 minute as many times as they could manage within the 2CT, with their arms crossed and their feet parallel 2. The participant was instructed to sit with his or her feet flat on the floor and their upper limbs folded across their chest. They were then instructed to fully stand up and sit down without using their arms for 1 minute. Sensations of breathlessness were scored using the Borg scale before starting the STSTs, and also after the end of the test. SpO2 and heart rate were measured throughout the test 6. Fig 1 - STST test among study participants shows the conductance of STST test among study participants

Figure 1: STST test among study participants

Six minute walk test: The 6 MWT was a simple practical test that only needed a 100-foot corridor and didn't require any specialized equipment or training for technicians. All patients, except those who had significant impairments, walked on a daily basis. The 6MWT examined the patient's ability to cover a certain distance quickly on a flat, hard surface over six Minutes.[65] Following the European Respiratory Society/American Thoracic Society (ERS/ATS) guidelines, the 6MWT was performed by moving back and forth between two cones spaced 30 meters apart. Patients were instructed to walk for as long as they could for six minutes, with the use of a cane or walker permitted if required, as seen in [Figure 2]: The six-minute walk test. During the test, the lowest saturation, blood pressure, and Respiratory rate were noted.[1]

The physical activity required a person to sit between two chairs and walk between them, and the physiological effects were quantified by counting the post-exercise changes in arterial oxygen saturation (SpO2) and pulse rate (PR). Two regular wooden chairs were set place side by side, 5 feet apart (from the front to Front ends), facing each other, with a flat backrest, no hand rest, and a sitting area that is 48 cm above the ground. The patient had to first relax for a few minutes while sitting on one of them until his PR and SpO2 were stable for at least 30s as seen in [Figure 3]: The Two chair test. The patient was then instructed to get up, walk to the other chair, sit down, then get up and walk back to the first chair after the baseline PR and SpO2 measurements were taken.[67]

Figure 2: The six minute walk test

Figure 3: Two Chair test

RESULTS

GROUP 1: Patients who performed 6MWT

GROUP 2: Patients who performed STST and 2CT.

The [Figure 4] represents the distribution of participants in the study, Group 1 and Group 2, each with a sample size of 34. The percentages indicate the distribution within each group, with both groups contributing to a total sample size of 68. Group 1

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represents 50% of the total, and Group 2 also represents 50% [Figure 4].

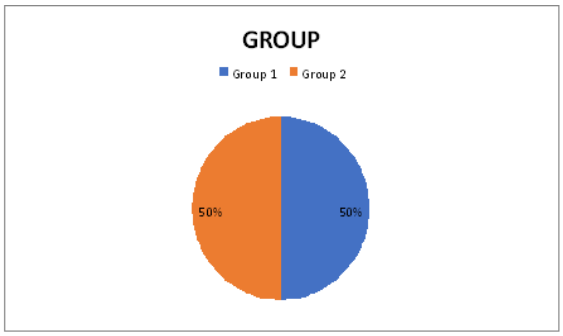

Figure 4: Distribution of study participants in both groups (n=68)

[Table 1] represents the distribution of participants in the study based on biological sex, smoking history, and biomass fuel exposure.
In group 1, 73.5% of the participants were male, and 26.5% were female. For Group 2, 76.5% were male, and 23.5% were female as seen in [Figure 5]: Gender-wise distribution of study participants in Group 1 and Fig 6: Gender-wise distribution of study participants in Group 2. Regarding smoking history, 52.9% of group 1 and 64.7% of group 2 had a smoking history. Additionally, 26.4% in Group 1 had biomass fuel exposure, while 23.5% had the same exposure in Group 2 as seen in [Table 1]: Distribution of study participants according to Age, Smoking history & Biomass fuel exposure.

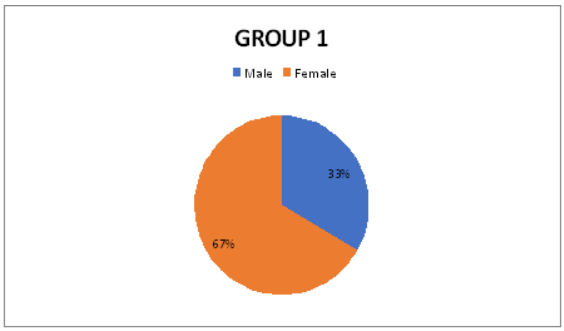

Figure 5: Gender wise distribution of study participants in Group 1. (n=34)

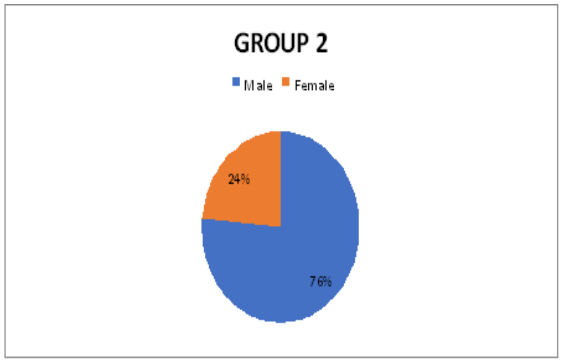

Figure 6: Gender-wise distribution of study participants in Group 2. (n=34)

Severity between the groups: The [Table 2] represents the distribution of participants in the study based on the severity of COPD.
In group 1, 5(14.7%) exhibited mild COPD, 14(41.1%) showed moderate COPD, and 12(35.2%) presented with severe COPD. In group 2, 8(23.5%) had mild COPD, 13(38.2%) had moderate COPD, and 11(23.3%) experienced severe COPD. Moreover, 3(8.8%) of group 1 participants and 2(5.8%) of group 2 participants demonstrated very severe COPD as seen in Fig 7: Distribution of study participants based on severity of COPD.

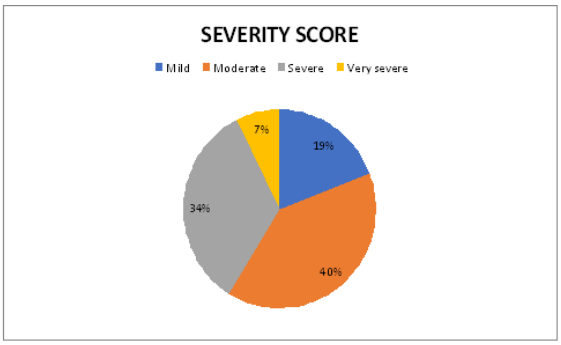

Figure 7: Distribution of study participants based on the severity of COPD (n=68)

The [Table 3] represents the comparison of symptoms between the groups.
The comparison of symptoms shows statistically significant (p =.000) between the groups 1 and 2 as seen in [Table 3] Distribution of study participants based on symptoms.

FOR HR,

Before the intervention, in Group 1, the mean HR was 87.50 with a standard deviation of 13.837 and in Group 2 the mean HR was 88.79 with a standard deviation of 15.227. The difference in HR between the two groups before the intervention was not statistically significant (p=0.715).
After the intervention, in group 1, the mean HR was 97.18 with a standard deviation of 18.008 and in group 2 the mean HR was 100.74 with a standard deviation of 16.497. The difference in HR between the two groups after the intervention was not statistically significant (p=0.399) as seen in Table 4: Distribution of study participants comparison of HR, SPO2, DBP, and SBP for both groups 1 (6MWT) and 2 (STST).

FOR SPO2,

Before the intervention, in group 1, the mean SPO2 was 96.35 with a standard deviation of 18.008 and in group 2 the mean SPO2 was 97.26 with a standard deviation of 2.287. The difference in SPO2 between the two groups before the intervention was not statistically significant (p=0.091).
After the intervention in group 1, the mean SPO2 was 94.85 with a standard deviation of 3.026, and in group 2 the mean SPO2 was 95.53 with a standard deviation of 4.237. The difference in SPO2 between the two groups after the intervention was not statistically significant (p=0.451) as seen in [Table 4]

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Distribution of study participants comparison of HR, SPO2, DBP, and SBP for both groups 1 (6MWT) and 2 (STST).

FOR SBP,

Before the intervention, in group 1, the mean SBP was 122.06 with a standard deviation of 14.095 and in group 2 the mean SBP was 124.56 with a standard deviation of 14.687. The difference in SBP between two groups before the intervention was not statistically significant (p=0.476).

After the intervention, in group 1, the mean SBP was 127.06 with a standard deviation of 15.081 and in group 2 the mean SBP was 132.35 with a standard deviation of 18.267. The difference in SBP between the two groups after the intervention was not statistically significant (p=0.197) as seen in [Table 4]: Distribution of study participants comparison of HR, SPO2, DBP, and SBP for both groups 1 (6MWT) and 2 (STST).

FOR DBP,

Before the intervention, in group 1, the mean DBP was 81.76 with a standard deviation of 12.178 and in group 2 the mean DBP was 76.76 with a standard deviation of 10.652. The difference in DBP between the two groups before the intervention was not statistically significant (p=0.076).

After the intervention, in group 1, the mean DBP was 85.59 with a standard deviation of 11.597 and in group 2 the mean DBP was 81.91 with a standard deviation of 11.011. The difference in DBP between the two groups after the intervention was not statistically significant (p=0.185) as seen in [Table 4]: Distribution of study participants comparison of HR, SPO2, DBP, and SBP for both groups 1 (6MWT) and 2 (STST).

FOR HR,

Before the intervention, in group 1, the mean HR was 87.50 with a standard deviation of 13.837 and in group 2 the mean HR was 89.24 with a standard deviation of 12.395. The difference in HR between the groups before the intervention was not statistically significant (p=0.588).

After the intervention, in group 1, the mean HR was 97.18 with a standard deviation of 18.008 and in group 2 the mean HR was 102.47 with a standard deviation of 14.463. The difference in HR between the groups after the intervention was not statistically significant (p=0.186) as seen in [Table 5]: Distribution of study participants comparison of HR, SPO2, DBP, and SBP for both groups 1 (6MWT) and 2 (STST).

FOR SPO2,

Before the intervention, in group 1, the mean SPO2 was 96.35 with a standard deviation of 2.087 and in group 2 the mean SPO2 was 97.21 with a standard deviation of 2.115. The difference in SPO2 between the groups before the intervention was not statistically significant (p=0.099)

After the intervention in group 1, the mean SPO2 was 94.85 with standard deviation of 3.026 and in group 2 the mean SPO2 was 96.09 with a standard deviation of 2.756. The difference in SPO2 between the two groups after the intervention was not statistically significant (p=0.083) as seen in Table 5: Distribution of study participants comparison of HR, SPO2, DBP, and SBP for both groups 1 (6MWT) and 2 (STST).

FOR SBP,

Before the intervention, in group 1, the mean SBP was 122.06 with a standard deviation of 14.095 and in group 2 the mean SBP was 122.06 with a standard deviation of 11.222. The difference in SBP between the two groups before the intervention was statistically significant (p=1.000).

After the intervention, in group 1, the mean SBP was 127.06 with a standard deviation of 15.081 and in group 2 the mean SBP was 131.18 with a standard deviation of 14.515. The difference in SBP between the two groups after the intervention was not statistically significant (p=0.256) as seen in Table 5: Distribution of study participants comparison of HR, SPO2, DBP, and SBP for both groups 1 (6MWT) and 2 (2CT).

FOR DBP,

Before the intervention, in group 1, the mean DBP was 81.76 with a standard deviation of 12.178 and in group 2 the mean DBP was 80.29 with a standard deviation of 8.699. The difference in DBP between the two groups before the intervention was not statistically significant (p=0.569).

After the intervention, in group 1, the mean DBP was 85.59 with a standard deviation of 11.597 and in group 2 the mean DBP was 85.29 with a standard deviation of 9.919. The difference in DBP between two groups after the intervention was not statistically significant (p=0.911) as seen in [Table 5]: Distribution of study participants comparison of HR, SPO2, DBP, and SBP for both groups 1 (6MWT) and 2 (2CT).

Table 1: Distribution of study participants according to Age, Smoking history & Biomass fuel exposure (n=68)

Variables		**Group 1 (n=34)**	**Group 2 (n=34)**
Sex	Male	25 (73.5%)	26 (76.5%)
	Female	9 (26.5%)	8 (23.5%)
	Total	34 (100%)	34 (100%)
Smoking history		18 (52.9%)	22 (64.7%)
Biomass fuel exposure		9 (26.4%)	8 (23.5%)

Table 2: Distribution of study participants based on severity of COPD. (n=68)

Grades	**Intensity**	**Airflow limitation**	**No.of.patients**	
GOLD 1	Mild	FEV1 ≥ 80% predicted	13 (19.11%)	Group 1 -5 (14.7%)
				Group 2 -8 (23.5%)
GOLD 2	Moderate	50% ≤ FEV1 80% Predicted	27 (39.7%)	Group 1 -14 (41.1%)

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				Group 2 -13 (38.2%)
GOLD 3	Severe	30 ≤ FEV1 50 % Predicted	23 (33.8%)	Group 1 -12 (35.2%)
				Group 2 -11 (23.3%)
GOLD 4	Very severe	FEV1 < 30% predicted	05 (7.3%)	Group1 -3 (8.8%) Group 2 - 2(5.8%)

Table 3: Distribution of study participants based on symptoms (n=68)

Group	Symptoms						Total	P value
	No symptoms	Dyspnea	Palpitation	Lower limb pain	Dyspnea & palpitation	Dyspnea & lower limb pain		
GROUP 1	6 (17.6%)	3 (8.8%)	14 (41.2%)	10 (29.4%)	1 (2.9%)	0 (0%)	34 (100.0%)	.000
GROUP 2	8 (23.5%)	15 (44.1%)	6 (17.6%)	1 (2.9%)	3 (8.8%)	1 (2.9%)	34 (100.0%)	
TOTAL	14 (20.6%)	18 (26.5%)	20 (29.4%)	11 (16.2%)	4 (5.9%)	1 (1.5%)	68 (100.0%)	

Table 4: Distribution of study participants comparison of HR, SPO2, DBP, and SBP for both groups 1 (6MWT) and 2 (STST) (n=68)

GROUP		N =34	MEAN	SD	P VALUE
HR BASELINE	1	34	87.50	13.837	0.715
	2	34	88.79	15.227	
HR END OF THE TEST	1	34	97.18	18.008	0.399
	2	34	100.74	16.497	
SPO2 BASELINE 2	1	34	96.35	2.087	0.091
	2	34	97.26	2.287	
SPO2 END OF THE TEST	1	34	94.85	3.026	0.451
	2	34	95.53	4.237	
SBP BASELINE	1	34	122.06	14.095	0.476
	2	34	124.56	14.687	
SBP END OF THE TEST	1	34	127.06	15.081	0.197
	2	34	132.35	18.267	
DBP BASELINE	2	34	81.76	12.178	0.076
	1	34	76.76	10.652	
DBP END OF THE TEST	1	34	85.59	11.597	0.185
	2	34	81.91	11.011	

Table 5: Distribution of study participants comparison of HR, SPO2, DBP, and SBP for both groups 1 (6MWT) and 2 (2CT) (n=68)

GROUP		N	MEAN	SD	P VALUE
HR BASELINE	1	34	87.50	13.837	0.588
	2	34	89.24	12.395	
HR END OF THE TEST	1	34	97.18	18.008	0.186
	2	34	102.47	14.463	
SPO2 BASELINE	1	34	96.35	2.087	0.099
	2	34	97.21	2.115	
SPO2 END OF THE TEST	1	34	94.85	3.026	0.083
	2	34	96.09	2.756	
SBP BASE LINE	1	34	122.06	14.095	1.000
	2	34	122.06	11.222	
SBP END OF THE TEST	1	34	127.06	15.081	0.256
	2	34	131.18	14.515	
DBP BASELINE	1	34	81.76	12.178	0.569
	2	34	80.29	8.699	
DBP END OF THE TEST	1	34	85.59	11.597	0.911
	2	34	85.29	9.919	

DISCUSSION

The daily social and physical activities of an individual are used to evaluate the effectiveness of treatment for COPD patients.[68] The most accurate, standardized, well-tolerated, and simple test for predicting survival in COPD patients is the 6- minute walk test (6MWT).[69] However, the primary requirement for this test is qualified personnel and space, all of which are typically not available in any setting. Therefore exploring other tests altered to 6MWT like STST, to assess physical ability is essential, It was initially used in elderly patients with orthopedic diseases but later on, it was found suitable in patients with COPD.[70]

In this study, we recruited 68 COPD patients which were divided into 2 groups, A and B. In this study, comparison is based on the mMRC grading, heart rate, spo2, and blood pressure and also correlates the difference between the 6MWT with STST and 2CT in the functional assessment of COPD patients.

Moreover, the strong positive correlation between the Sit-to-Stand test, the Two Chair Test, and the Six-Minute Walk Test implies that these assessments collectively capture various dimensions of physical function in COPD patients. This multifaceted

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approach enhances the sensitivity of the evaluation, allowing for a nuanced understanding of different aspects of functional performance, such as lower limb strength, and balance. The emphasis on low-cost preferences and ease of administration in employing the Sit-to-Stand test, Two Chair Test, and Six-Minute Walk Test is pivotal in enhancing the feasibility and accessibility of functional assessments for COPD patients. Firstly, the affordability of these tests makes them particularly advantageous, especially in resource-constrained healthcare settings. The minimal requirement for specialized equipment ensures that healthcare facilities, even those with limited budgets, can readily incorporate these assessments into their routine practice. This cost-effectiveness is crucial for widespread adoption and ensures that the benefits of functional assessments are accessible across various healthcare settings, regardless of financial constraints. Secondly, the simplicity and ease of administration of these tests contribute to their practical utility. Minimal training is required for healthcare professionals to conduct these assessments, reducing the burden on resources and time. Additionally, the straightforward instructions and low complexity make it feasible for non-specialized personnel, such as nurses or respiratory therapists, to administer the tests effectively. This simplicity enhances the scalability of functional assessments, allowing them to be implemented in diverse healthcare settings, including primary care facilities and community health centers. Furthermore, the ease of these assessments makes them patient-friendly, fostering better cooperation and compliance. Patients are more likely to participate actively in assessments that are simple to understand and perform, which, in turn, improves the reliability and accuracy of the gathered data. This patient-centered approach aligns with the broader goal of enhancing patient engagement and promoting regular monitoring of functional status in COPD management. In conclusion, the integration of low-cost and easily administered functional assessments, such as the Sit-to-Stand test, Two Chair Test, and Six-Minute Walk Test, not only optimizes resource utilization but also ensures their applicability across diverse healthcare settings, ultimately benefiting COPD patients through improved accessibility and patient engagement in functional evaluation.

Furthermore, the positive correlation serves as a foundation for establishing these tests as reliable outcome measures in interventions and clinical trials aimed at improving COPD patients' functional capacity. The shared variance among these assessments enhances the precision of tracking changes over time, thereby facilitating the evaluation of the effectiveness of therapeutic interventions and rehabilitation programs. This positive discussion reinforces the clinical significance and applicability of the chosen functional assessment tools in the context of COPD management, and endurance.

In conclusion, the positive correlation between the Sit-to-Stand test, Two Chair Test, and Six-Minute Walk Test not only validates their concurrent measurement but also opens avenues for future research. Exploring the underlying mechanisms driving these correlations and their implications for tailoring individualized interventions could contribute to advancing our understanding of functional limitations in COPD patients and optimizing their care.

There were similar correlations of the STST and 2CT with 6MWT which means there were no significant differences in the capacity of these tests. Our study indicates that the results of the 6MWT are very similar to the STST and two-chair test. It also proved that the STST and two chair test develop similar hemodynamic stress, better tolerated, easy to perform, simple, cheap, less time-consuming, requiring only basic equipment but at the same time valid, reliable, and repeatable.

CONCLUSION

This study aimed to correlate the functional assessment of COPD patients using the STST and 2CT with 6MWT. The results indicate that no statistically significant difference in heart rate, spo2, mMRC grading, and blood pressure. To conclude STST and 2CT show similar interventions with 6MWT. As it is low cost, reliable, repeatable, easy to perform, less time-consuming when compared to the other tests.

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