combustion products as observed in earlier reports [20 and 21].

BMI is one of the major risk factor for COPD [22]. Results of the present study also showed that BMI values in COPD patients (19.66±4.98) were significantly low when compared to their respective controls (28.35 ± 8.04) (p=0.001). This may be due to malnutrition or intake of low calorie diet or due to complete utilization of calories leading to heavy weight loss. This is in accordance with a previous study in Indian population where COPD patients had decreased BMI [23].

In the present study, among 250 COPD patients, 197 (68%) were smokers, 45 (29.6%), ex-smokers and 8(1.6%) were non-smokers. Studies from India have shown tobacco smoking to be responsible for over 82% of COPD cases [24]. Rani et al., [25] reported that the prevalence of smoking in South Indian males was significantly high (35.4%) compared to North Indians (23.9%). In smokers the risk for COPD is based upon the age, intake of cigarettes, pack years of smoking, present status of smoking. But not all smokers develop COPD which indicates that there is a possible risk of associated genetic factors for the susceptibility of the disease. [26]. The emerging consensus that pack years of smoking is the key predictive factor in the development of COPD is well supported [27]. This study also collected the data from patients regarding number of cigarettes smoked per day along with duration of cigarette smoking from the smokers and ex-smokers with COPD. The information obtained from the proforma of COPD non smokers showed that the patients were industrial workers, farmers, welders, washer-men and painters who were suffering with COPD for more than 2 years.

There are three main themes within the pathogenesis of COPD. The oxidant-antioxidant theory states that disparity between levels of harmful oxidants and protective antioxidants leads to oxidative stress, which in turn influences the actions of anti-proteases, and expression of proinflammatory mediators [28]. The protease-anti-protease theory suggests that there is an imbalance between proteases that digest elastin together with other components of the extra-cellular matrix and anti-proteases that protect against it [29]. Both of the above theories link to the third theory about the importance of inflammation in the pathogenesis of COPD [30,31]. Polymorphisms in genes relating to oxidants, proteases, antioxidants and inflammation have been found to relate to the presence of features of COPD suggesting reasons for the heterogeneity observed in clinical phenotype.

Imbalance of oxidants-antioxidants results in oxidative stress and release number of oxidants that destroy the antioxidant capacity. Various studies have proved the existence of oxidative burden in blood, breath, lung airspaces and in urine of smokers suffering with COPD. The oxidants released from cigarette smoke increase the oxidative stress along with increase in leukocytes with ROS in the blood and in airways of lungs. Smoking leads to oxidative stress that causes chronic inflammation of lungs [32]. Antioxidant enzymes increase the activity of cells and other antioxidant defences to reduce oxidative burden under physiological conditions. This is achieved by a variety of antioxidant defense systems that activate enzymes and the synthesis of antioxidants. ROS and RNS increase the oxidative stress and decrease the antioxidant levels [33]. Thus, decline in levels of antioxidant enzyme activity could lead either to higher oxidative stress or low levels of defense. Hence the present study was aimed at evaluating the role of antioxidant genes (GSTM1 & HMOX-1) that are shown to be associated with COPD in different population and to see whether these genes are involved in COPD from Indian population. So here we report the association of GSTM1 null and HMOX-1 GC in our population as the genetic risk factors in developing COPD.

Various studies have shown the involvement of Glutathione S-transferase μ (GSTM1) null genotype to be associated with COPD and lung cancer [34 & 35]. Few studies have reported that the function of GSTM1 is lost in null genotype leading to non-detoxification of aromatic hydrocarbons released from cigarette smoke, resulting in increase of inflammatory cells which damage the tissue of lungs with decline in lung function [36]. The frequency of mutant GSTM1- null genotype has also been reported to be significantly higher in mild/moderate types of COPD . GSTM1 polymorphism which is in accordance with earlier studies [37, 38, 39 & 40]. The evaluation of GSTT1 does not show any significance with the genotypes of COPD and the observations were similar to the studies of Yim et.al [41].

In the present study the frequency of GC genotype of HMOX-1 was found to be significantly associated in COPD patients when compared to control. This is the first study from India to show the HMOX-1 -19/GC (rs2071747) variant to be associated with COPD. Among the antioxidative enzymes HMOX1 is considered as protective gene for lungs from oxidative stress. Recently the role of microsatellite polymorphism of HMOX1 gene promoter has been reported for some human diseases. It was shown that longer (GT)n repeat was associated with angiographic restenosis after coronary stunting, lung adenocarcinoma and pneumonia [42, 43, 44]. There are very few studies that investigated on the role of antioxidant gene polymorphisms in COPD and one study had shown the nominal association (p = 0.015) between one intronic HMOX-1 SNP (rs2071749) and lung function decline [45]. Some studies did not show the association of HMOX-1 gene in lung associated diseases [46]. However the studies on HMOX-1 in relation with COPD from other populations are required to clarify these results.

6. Conclusion

This is the intial study carried out in Telangana region with COPD population. The demographic feature like age and BMI was found to be the risks factors associated with disease. This study also shows the male gender is more prone to COPD as smoking remains the main risk factor of the disease where as female gender are not habituated towards smoking in our country when compared with most western and European countries. This study reveals the relation of GSTM1 and HMOX-1 genes were found to be significantly associated with COPD. Hence it is clear that the role of genes also play an important role in the pathology of the disease. It is essential to have knowledge on genetic

contribution to COPD development and especially interaction of different candidate genes and GWAS (Genome wide association studies) from India would provide in better understanding the pathways involved in COPD pathogenesis.

7. Future Study

Further research will be focused on the role of oxidative genes involved in COPD. This study will help in early diagnosis and to discover new drugs to eradicate the habit of smoking and Exacerbations. Studies are also needed on a large sample size to bring about the knowledge and awareness of COPD among people living in the rural areas with Confirmational studies in other prospective cohorts will be of great importance.

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