A Comparative Study on effects of Conventional Smoking Versus E-Cigarettes Smoking Assessed by Inflammatory Markers

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ABSTRACT

Introduction: Electronic cigarettes (e-cigarettes) are marketed and advertised as a new method and a better alternative than conventional smoking. It is promoted by various companies that there are lesser incidence of smoking-related illnesses which is doubtful. However, concerns about their safety and toxicity have to be considered due to its widespread use and limited number of studies which enlightens on their effects on the respiratory system. Our aim was to study whether the e-ecigrattes are safer than conventional cigarettes smoking.

Material and methods: A total of 105 patients were considered for the present study, 35 patients constituted the control group who had no history of smoking, rest 70 patients were smokers with h/o smoking more than 5 years, smokers were further grouped based on type of cigarettes used into Group A which included 35 patients using conventional smoking and 35 patients who used ecigrattes constituted Group B. Patients in the age group 30 - 70 yrs were enrolled for the study

Results: Continuous variables in three groups were compared using ANOVA and no categorical variables were compared using chi square test. The mean age of study population was 52.28 ± 11.13 , Mean age in group A was 51.29 ± 10.67 compared to group B 53.54 \pm 11.79 and controls 52 \pm 10.9 p (>0.05). Case group comprised of 88.6% males and 11.4% females in both group A and Group B compared to 82.9% males and 13.3% females in controls p (>0.05), patients presented with history of HTN as follows Group A 60%, group B 40% and controls 37.1% p (>0.05). Group A 28.6%, group B 25.7% and controls 31.4% p (>0.05) had history of diabetes. Conclusion: Present study demonstrates that effects of conventional cigarettes on inflammatory markers are significantly higher than when compared to e cigarettes. this doesn't rule out the safety with e cigarettes as the incidence of inflammatory markers with e cigarettes was observed to be significantly higher than controls.

Keywords: Conventional Smoking, E-Cigarettes Smoking, Inflammatory Markers

INTRODUCTION

Electronic cigarettes (e-cigarettes) are marketed and advertised as a new method and a better alternative than conventional smoking. It is promoted by various companies that there are lesser incidence of smoking-related illnesses which is doubtful. However, concerns about their safety and toxicity have to be considered due to its widespread use and limited number of studies which enlightens on their effects on the respiratory system. There have been various controversies in relation to the risks and benefits of e-cigarettes, which has resulted in confusion among health care practitioners and the general population. 1-2 In 2015, the prevalence of newer-

smokers using e-cigs among the youth were nearly 19% compared to 10% adults as newer smokers.3 About 5% of college students who have never smoked are nowadays using e-cigs due its promotions as safer alternative. More than Fifty percent of adult smokers in the US have already tried e-cigs, and near about 23% of adults are using both cigarettes and e-cigs. The reasons for which adults have started using e-cig were in hope of quitting smoking, health concerns, and convenience. Various marketing tools are used to promotes and popularize the use of e-cigs among young and adults alike with various add on's such as e-liquid flavors, which make them more attractive to a smokers and non-smokers.³⁻⁵ E cigarettes are battery powered with electronic heating elements that aerosolize carrier liquids that usually contain nicotine they come with various models. The carriers are vegetable glycerol (VG) and/or propylene glycol (PG).6-7 The use of e-cigs and similar products is rapidly rising, with sales totaling more than \$3.7 billion per year. All of the major tobacco manufacturers are marketing these products. The rates of e-cig use among youth are now higher than cigarette use, although the estimate of use may vary depending on the method of survey.

Riker, et al. study has shown that e-cigarette aerosol exhaled can pose a risk exposure to the bystanders as similar to that of environmental tobacco smoke (ETS) from conventional cigarettes. Our aim was to study whether the e-ecigrattes are safer than conventional cigarettes smoking.

MATERIAL AND METHODS

A total of 105 patients were considered for the present study, 35 patients constituted the control group who had no history of smoking, rest 70 patients were smokers with h/o smoking more than 5 years, smokers were further grouped based on type of cigarettes used into Group A which included 35 patients using conventional smoking and 35 patients who used ecigrattes constituted Group B. Patients in the age group 30-70 yrs were enrolled for the study.

Inclusion criteria

1. Males and female between age group 30 - 70 yrs.

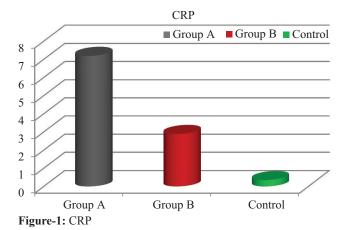
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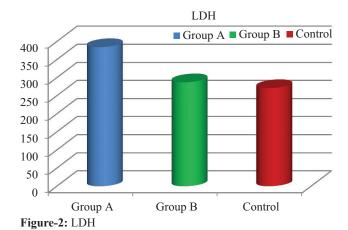
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	Group A	Group B	Control	p value	
Age	51.29 ± 10.67	53.54 ± 11.79	52 ± 10.9	0.691	
BMI	20.31 ± 1.74	21.37 ± 2.4	21.69 ± 2.7	0.053	
CRP	7.19 ± 1.66	2.89 ± 2.5	$0.35 \pm .24$	< 0.001	
LDH	384.4 ± 125.7	287.1 ± 106.6	271.09 ± 73.5	< 0.001	
WBC	9294.28 ± 1049.3	8242.8 ± 990.1	6740 ± 1209.8	< 0.001	
Table 1: Comparison of both group with control and its statically significance					

	Group A	Group B	Control			
Gender						
Males	31 (88.6)	31 (88.6)	29(82.9)	0.719		
Females	4 (11.4)	4 (11.4)	6 (17.1)			
HTN	21(60)	14 (40)	13 (37.1)	0.112		
DM	10 (28.6)	9 (25.7)	11 (31.4)	0.869		
Table-2: Descriptive						





known cigarettes and e-cigarettes smokers with coexisting HTN and DM

Exclusion Criteria

- 1. Patients with airway disease other than COPD
- 2. Patients with Fbs > 150mg/dl, PPBS >250mg/dl, and Ketosis
- 3. Patients with Uncontrolled HTN
- 4. History of asthma
- 5. History of collagen vascular/autoimmune diseases,
- 6. History of malignancy,
- 7. History of pulmonary embolism,
- 8. History of renal insufficiency,

9. History of cirrhosis and other serious liver diseases The BMI was then calculated by dividing the weight in kilograms by height in meter square. Serum CRP, WBC and LDH was estimated in both groups.

STATISTICAL ANALYSIS

The data obtained was analyzed using SPSS v 17. Continuous variables in three groups were compared using ANOVA and no categorical variables were compared using chi square test.

RESULTS

The mean age of study population was 52.28 ± 11.13 , Mean age in group A was 51.29 ± 10.67 compared to group B 53.54 ± 11.79 and controls 52 ± 10.9 p (>0.05). Case group comprised of 88.6% males and 11.4% females in both group A and Group B compared to 82.9% males and 13.3% females in controls p (>0.05). patients presented with history of HTN as follows Group A 60%, group B 40% and controls 37.1% p (>0.05). Group A 28.6%, group B 25.7% and controls 31.4% p (>0.05) had history of diabetes (table-1,2).

Mean BMI in Group A was 20.31 ± 1.74 compared to Group B was 21.37 ± 2.4 and controls 21.69 ± 2.7 , there was no statistically significant difference observed in the mean BMI between controls and case groups p >0.05. Mean CRP in Group A was 7.19 ± 1.66 which was significantly higher than Group B 2.89 ± 2.5 and controls $0.35 \pm .24$ (p <0.05). Mean CRP in group B was also significantly higher than when compared to controls (figure-1).

Mean LDH in Group A was 384.4 ± 125.7 which was significantly higher than Group B 287.1 ± 106.6 and controls 271.09 ± 73.5 (p <0.05). Mean LDH in group B was not significantly higher than when compared to controls p >0.05 (figure-2).

Mean WBC in Group A was 9294.28 \pm 1049.3 which was significantly higher than Group B 8242.8 \pm 990.1 and controls 6740 \pm 1209.8 (p <0.05). Mean WBC in group B was also significantly higher than when compared to controls p <0.05.

DISCUSSION

In majority of cases of Chronic Obstructive pulmonary disease and Carcinoma lung it is clear that more than 90% of patient have predisposing cause i.e. Cigarette smoking. It is very clear by multiple studies over decades that smoke emitted from smoking have toxicants which results in inflammatory response in lung. This inflammation is considered a hallmark of cancer and COPD. In healthy smokers before the onset of disease it is observed that they have impact of pro-inflammatory effects on lung.

People with smoking of cigarette activates alveolar macrophages and airway epithelial cells which releases proinflammatory cytokines which results in infiltration of inflammatory cells into lung via blood. It is clearly explained by many studies that people with smoking have upregulation of proinflammatory markers and down-regulate anti-inflammatory markers.

Smoking can results in chronic inflammation which can promote unregulated proliferation of cells, cell invasion, angiogenesis and instability of genome.

Various studies have shown that patients who are smoking for long period of time are prone to lung cancer. It is proposed that these people have KRAS oncogenesis which is frequently mutated by many factors such as NF- κ B and STAT3

Bullen C et.al. 2010, Etter JF 2011 et.al have shown that in smokers there is higher inflammatory cell content than non-smokers.⁹

Vansickel AR et.al 2013, Etter JF et.al 2011, Dawkins Let.al 2012,2013, have done studies on smokers versus nonsmokers were they interesting found that when the patients are exposed to smoking they have higher neutrophil counts in sputum than compared to nonsmokers. Apart from this when patient had quitted smoking they observed that the sputum neutrophil counts started to decrease after 6 weeks in these patients. Not only neutrophils but they have a decrease of macrophages as early as 1 week.¹⁰⁻¹³

Mc Auley TR et al (2012) enrolled 28 known smokers and ex-smokers who quitted smoking for nearly 1 year and these patients underwent bronchoscopy. It was observed that patient who quitted smoking had significant reduction of inflammatory cells than smokers. It is seen that patients who haved reduced smoking of cigarettes by 50% each day have decrease BAL macrophages and neutrophils.¹⁴

Romagna G et.al 2013 the study compared the aerosol causing cytotoxicity from various flavored brands of e-cigarettes liquid. It was found that aerosol from coffee-flavored e-liquid produced a cytotoxic effect.¹⁵

Flouris et al has assessed the impact of e-cigarette use on pulmonary function in 15 cigarette smokers who has been given the puff of e-cigarette apart from conventional cigarette. It was seen that conventional cigarette people had significant decrease in expired lung volume whereas in e-cigarette group of patients it was not seen. In addition in conventional cigarette group of patients it was seen that w.b.c count was increased whereas in e-cigarette group of patients it was not significantly increased.¹⁶

CONCLUSION

Present study demonstrates that effects of conventional cigarettes on inflammatory markers are significantly higher than when compared to e-cigarettes. This doesn't rule out the safety with e cigarettes as the incidence of inflammatory markers with e cigarettes was observed to be significantly higher than controls.

REFERENCES

1. Caponnetto P, Campagna D, Papale G, et al. The emerging

- phenomenon of electronic cigarettes. Expert Rev Respir Med 2012;6:63–74.
- 2. Etter JF, Bullen C, Flouris AD, et al. Electronic nicotine delivery systems: a research agenda. Tob Control 2011;20:243–8.
- 3. Choi K, Forster JL. Beliefs and experimentation with electronic cigarettes: a prospective analysis among young adults. Am J Prev Med. 2014;46:175–178.
- Farsalinos KE, Romagna G, Allifranchini E, Ripamonti E, Bocchietto E, Todeschi S, Tsiapras D, Kyrzopoulos S, Voudris V. Comparison of the cytotoxic potential of cigarette smoke and electronic cigarette vapour extract on cultured myocardial cells. Int J Environ Res Public Health. 2013;10:5146–5162.
- Etter JF, Bullen C. Electronic cigarette: users profile, utilization, satisfaction and perceived efficacy. Addiction. 2011;106: 2017–2028.
- Pellegrino R.M., Tinghino B., Mangiaracina G., Marani A., Vitali M., Protano C., Osborn J.F., Cattaruzza M.S. Electronic cigarettes: An evaluation of exposure to chemicals and fine particulate matter (PM) Ann. Ig. 2012;24:279–288.
- 7. Regan A.K., Promoff G., Dube S.R., Arrazola R. Electronic nicotine delivery systems: Adult use and awareness of the "e-cigarette" in the USA. Tob. Control. 2013;22:19–23.
- Riker C.A., Lee K., Darville A., Hahn E. E-cigarettes: Promise or peril? J. Nurs. Clin. North Am. 2012;47:159– 171.
- 9. Bullen C, McRobbie H, Thornley S, Glover M, Lin R, Laugesen M. Effect of an electronic nicotine delivery device (e cigarette) on desire to smoke and withdrawal, user preferences and nicotine delivery: randomised crossover trial. Tob Control. 2010;19: 98–103.
- Vansickel AR, Eissenberg T. Electronic cigarettes: effective nicotine delivery after acute administration. Nicotine Tob Res. 2013;15:267–270.
- Etter JF, Bullen C. Saliva cotinine levels in users of electronic cigarettes. Eur Respir J. 2011;38: 1219–1220.
- Dawkins L, Corcoran O. Acute electronic cigarette use: nicotine delivery and subjective effects in regular users. Psychopharmacology. 2013:1–7.
- Dawkins L, Turner J, Hasna S, Soar K. The electroniccigarette: effects on desire to smoke, withdrawal symptoms and cognition. Addict Behav. 2012;37:970– 973
- McAuley TR, Hopke PK, Zhao J, Babaian S. Comparison of the effects of e-cigarette vapor and cigarette smoke on indoor air quality. Inhal Toxicol. 2012; 24:850–857.
- 15. Romagna G, Allifranchini E, Bocchietto E, Todeschi S, Esposito M, Farsalinos KE. Cytotoxicity evaluation of electronic cigarette vapor extract on cultured mammalian fibroblasts (ClearStream-LIFE): comparison with tobacco cigarette smoke extract. Inhal Toxicol. 2013;25:354–361.
- Flouris AD, Chorti MS, Poulianiti KP, Jamurtas AZ, Kostikas K, Tzatzarakis MN, Wallace Hayes A, Tsatsakis AM, Koutedakis Y. Acute impact of active and passive electronic cigarette smoking on serum cotinine and lung function. Inhal Toxicol. 2013;25:91–101.

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