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Evaluation of hSCRP and microalbumin levels in smokers

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Abstract--Background: Smoking is associated increase in morbidity and mortality from various diseases. Increasing evidence suggests that chronic smoking adversely affects vascular and hormonal systems. Smoking plays a significant role in the development of atherosclerosis, thrombogenesis and vascular occlusion, which further adversely affects the prognosis of nephropathies. Aim and. Objectives: to estimate and compare the levels of hSCRP and microalbumin levels in smokers and non-smokers. Materials and Methods: Under aseptic precautions random venous blood sample of 2 mL was drawn from ante-cubital vein and collected in red top tube and serum was separated and analysed for high sensitivity C reactive protein (hSCRP). Early morning mid-stream urine was used for urine microalbumin estimation and the same sample was used for urine microscopy to exclude patients with urinary tract infection. Statistical Analysis: All statistical tests was performed using SPSS software. For comparisons of different variables student's t-test and Chi-square test were be used. Pearson coefficient of correlation was used for assessment of relationship between variables. A p value <0.05 was considered statistically significant. Results and Discussion: In our study we observed that mean levels of urine microalbumin, hsCRP were increased in non-diabetic, normotensive smokers, this increase was directly related to the amount and duration of smoking in smokers.

Conclusion: Non-Diabetic, normotensive smokers have significantly higher levels of hsCRP and urine spot microalbumin levels in our smoker population. This increase is proportional to the duration of smoking in pack-years. This study has a large impact on the early detection of renal damage in subjects with smoking which could be prevented with early intervention. Early intervention can prevent further progression of the renal disease. There was limited data regarding effect of smoking on microalbuminuria and hsCRP levels in non-diabetic and normotensive subjects, hence study will add to the new information to the existing limited data.

Keywords---high sensitivity, C reactive protein, urine microalbumin, smoking, creatinine.

Introduction

Smoking is associated increase in morbidity and mortality from various diseases. Increasing evidence suggests that chronic smoking adversely affects vascular and hormonal systems. Smoking plays a significant role in the development of atherosclerosis, thrombogenesis and vascular occlusion, which further adversely affects the prognosis of nephropathies [1,2]. Several studies conducted in the past have documented that, smoking is an independent determinant of microalbuminuria in all participants, i.e., non-diabetic and diabetic patients with a high cardiovascular risk profile. Microalbuminuria is defined as “excretion of albumin in the range of 30-300 mg /day or >20 mg/L (early morning midstream urine)”. Microalbuminuria is a predictor of cardiovascular mortality, predicts the future risk of mortality and end stage renal disease. Chronic Smoking increases the risk of microalbuminuria and accelerates the rate of progression from microalbuminuria to macroalbuminuria and subsequent renal failure in diabetic population [3,4,5,6,7,8].

Serum hs-CRP, the main acute phase protein, is a sensitive marker for systemic inflammation in humans. It is produced mainly by the liver and to some extent by the adipose tissue in response to proinflammatory cytokine induced by inflammatory stimuli. Among apparently healthy men and women, the currently recommended serum hs-CRP cut-off points are <1.0 mg/L for low risk, 1–3 mg/L for average risk and >3.0 mg/L for high risk of future CVD¹¹. Cigarette smoking is a classical and a major risk factor for development of inflammatory condition which can be assessed by serum high sensitive C reactive protein (hs-CRP) level. Low grade inflammation indicated by the serum hs-CRP level, which may act as risk factor to develop future atherosclerosis. On the contrary some authors found no change of hs-CRP level in smokers. Several studies have done abroad to observe the association of hs-CRP level with smoking but the relationships are still debatable. [9,10,11,12,13,14]. Hence we have taken up this study to evaluate the levels of hsCRP and urine spot microalbumin levels in our population.

Aim and Objectives

- To estimate the levels of hSCRp and microalbumin levels in smokers

- To compare the levels of hsCRP and microalbumin levels in smokers and non-smokers

Materials and Methods

Source of Data

A prospective Hospital Based Prospective Cross-Sectional Cohort Study on “Evaluation of hscrp and microalbumin levels in Smokers” An Hospital Based Prospective Cross-Sectional Cohort Study” was conducted at our Hospital as per the inclusion criteria at TRR Institute of Medical Sciences from January 2021 to December 2021.

Inclusion Criteria

50 subjects with history of smoking in the age group of 25-70 years who are non-diabetic (fasting serum glucose <125 mg/dL), normotensive (Blood Pressure <139/<89 mmHg), with normal lipid profile, no history of premature vascular disease and normal serum urea (<40 mg/dL) and creatinine (<1 mg/dL) with the history of smoking, who were attending the general medicine outpatient clinic were enrolled in the study. The non-smokers will be age matched and taken as control group. Smoker is defined as the one who had smoked 20 cigarettes/bidi per day for 5 years (5-pack years) or equivalent. They will be divided into four sub-groups: Very light smoker (5-9 pack-years), light smoker (10-14 pack-years), moderate smokers (15-19 pack-years) and heavy smokers (>20 pack-years). Non-smokers neither smoked nor chewed tobacco in any form.

Exclusion Criteria

- Subjects with age <25 and > 70 years, Diabetics on insulin and oral antidiabetic medications, Antihypertensive medications, Lipid lowering drugs,
- Abnormal serum urea (>40 mg/dL) and creatinine (>1mg/dL) levels,
- Urinary tract infections,
- Alcohol consumption,
- Fever,
- Vigorous physical activity,
- Pregnancy, and
- those who are not willing to give consent.

Biochemical Analysis

Under aseptic precautions random venous blood sample of 2 mL was drawn from ante-cubital vein and collected in red top tube and serum was separated and analysed for high sensitivity C reactive protein (hSCRP). Early morning mid-stream urine sample was used for urine microalbumin and the same sample was used for urine microscopy to exclude patients with urinary tract infection.

Statistical Analysis

All statistical tests was performed using SPSS software. For comparisons of different variables student's t-test was be used. Pearson coefficient of correlation was used for assessment of relationship between variables. A p value <0.05 was considered statistically significant.

Results

A total of 100 male subjects were enrolled in the present study, out of which 50 subjects were smokers and 50 subjects were non-smokers. The mean age in smokers was 52.6 ± 10.2 and in non-smokers was 48.6 ± 11.13 years.

Table 1 Shows the division of smokers depending on duration of smoking	
Smokers	(n = 50)
Very light smokers	01
Light Smokers	20
Moderate Smokers	20
Heavy Smokers	09

It is observed from the table 1 that Smokers were further divided into 4 groups as Very light smoker (5-9 pack-years), light smoker (10-14 pack-years), moderate smokers (15-19 pack-years) and heavy smokers (>20 pack-years). Very light smokers were nil, light smokers were 30, moderate smokers were 20 and heavy smokers were

Table 2 Shows the comparison of hsCRP and microalbumin levels in smokers and non-smokers			
	Smokers	Non-smokers	P value
hSCRp (mg/L)	2.78	1.01	<0.001
Urine spot microalbumin in mg/dL	31.56	14.98	<0.001

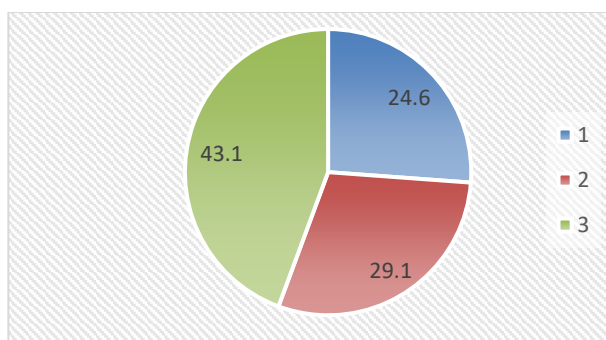


Figure 1. Shows comparison of mean levels urine microalbumin levels (mg/L) among smokers

1= light smokers (10-14 pack-years), 2 = moderate smokers (15-19 pack-years) and 3 = heavy smokers (>20 pack-years). It is evident from the figure 1 that mean levels of urine microalbumin is increased with increase in the duration of smoking between light smokers, moderate smokers and heavy smokers

Discussion

In our study we observed that mean levels of urine microalbumin, hsCRP were increased in non-diabetic, normotensive smokers, this increase was directly related to the amount and duration of smoking in smokers. Which is on accordance with the heart outcome prevention study, which documented that smoking was an independent determinant of microalbuminuria in all participants, non-diabetic and diabetic patients with high cardiovascular risk [7]. C-Reactive Protein (CRP) is synthesized by the liver in response to inflammation. CRP test in human blood is one of the most common hematology tests to measure non-specific inflammation. In the absence of an acute phase of inflammation, the level of CRP is relatively stable. An elevated baseline inflammatory status, as measured by CRP level, has been shown to increase the risk of several chronic conditions, including cardiovascular diseases lung cancer and colorectal cancer. Moreover, CRP levels are considered good long-term predictors of prognosis and relapse in patients with various chronic diseases, including colorectal cancer non-small-cell lung cancer (NSCLC) and respiratory gastrointestinal or cardiovascular diseases [14,15,16]

The focus of most of the research attempting to link CRP expression and incidence of chronic diseases that are confounded by smoking status has been in the area of coronary heart disease (CHD), and a recent review provides a comprehensive analysis of this field. Furthermore, the formation of atherosclerotic plaques in response to elevated CRP levels and hence the increased risk of atherothrombosis because of plaque rupture have been discussed in great detail by other authors and the numerous other pathways and mechanisms by which cigarette smoking can induce inflammation and therefore lead to plaque formation are also reviewed elsewhere. For example, work detailing postmortem analysis of coronary arteries in smokers vs. non-smokers who died between the ages of 15 and 34 as a result of external factors showed that advanced (grade 5) atherosclerotic lesions were far more prevalent in smokers compared with non-smokers [Odds ratio (OR) 9.61, 95% confidence interval (CI) 2.34–39.57]. Although assay of CRP in these cadavers showed no correlation between smoking status and increased CRP suggesting plaque formation cannot be simply explained by a single factor, at least in younger smokers [16,17,18, 19, 20,21]

Conclusion

Non-Diabetic, normotensive smokers have significantly higher levels of hsCRP and urine spot microalbumin levels in our smoker population. This increase is proportional to the duration of smoking in pack-years. This study has a large impact on the early detection of renal damage in subjects with smoking which could be prevented with early intervention. Early intervention can prevent further progression of the renal disease. There was limited data regarding effect of smoking on microalbuminuria and hsCRP levels in non-diabetic and

normotensive subjects, hence study will add to the new information to the existing limited data. Microalbuminuria test can be used as early routine screening test in subjects with smoking to detect the renal disease.

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