

Effects of Smoking on Chronic Kidney Disease

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Abstract

Cigarette smoking is a poor factor for multiple organs and systems in our body, which can promote the occurrence of cancer and increase the risk of cardiovascular accidents. Meanwhile, smoking also plays an important role in the development of Chronic Kidney Disease (CKD). Smoking promotes the development of renal fibrosis through the following mechanisms: Endothelial cell dysfunction, oxidative stress, activation of growth factors, tubular atrophy, insulin resistance, Advanced Glycation End Products (AGEPs) formation and impaired lipoprotein metabolism. And, smoking is associated with increased risk of proteinuria than non-smoking. Thus, smoking is a poor independent risk factor for our body, which should be cessation and take more appreciate.

Keywords: Smoking; Chronic kidney disease; Renal fibrosis

Introduction

Nowadays, cigarette smoking has been recognized as a harmful and carcinogenic factor to respiratory and cardiovascular systems. Furthermore, the role of smoking as an intrinsic nephrotoxic factor in the occurrence and progression of Chronic Kidney Disease (CKD) is becoming more appreciate [1]. A meta-analysis involved 15 prospective studies including 65 064 CKD cases demonstrated that cigarette smoking was an independent risk factor for CKD [2]. In this study, the Relative Risks (RR) in former smoker for CKD and End-Stage Renal Disease (ERSD) were 1.27 and 1.51, respectively. However, the RR for CKD (1.34) and ERSD (1.91) were increasing for current smokers, which indicated that cigarette smoking has a dim effect on kidney disease. The aim of this study was to review the relationship between cigarette smoking and CKD.

Discussion

Progressive tubulointerstitial fibrosis is the ultimate pathway for the progression of various renal diseases to end-stage renal disease [3]. Cigarette smoking accelerates the process of kidney fibrosis, leading to a faster decline of kidney function. Kurus M et al. [4] found that male Wistar rats showed obvious renal tubular atrophy, renal interstitial fibrosis, and other changes after exposure to tobacco smoke for 6 weeks. The mechanisms of renal fibrosis caused by smoking maybe related to endothelial cell dysfunction, oxidative stress, activation of growth factors, tubular atrophy and impaired lipoprotein metabolism [5-7].

Nicotine is one of the key toxic chemicals in tobacco, which plays a critical role in renal fibrosis. Khanna AK et al. [8] confirmed that nicotine in tobacco affects the viability of renal tubular epithelial cells through oxidative stress. Furthermore, nicotine can reduce cell damage, Pro-inflammatory and Pro-oxidant properties, which can promote mesangial cell proliferation and increase production of the extracellular matrix [9-12]. Glycotoxins is another fatal toxic chemical of cigarette smoke, which can rapidly induce Advanced Glycation End Products (AGEPs) formation. Several studies have shown that AGEPs can increase vascular permeability and promote vascular transform [13,14], which can mediate a faster progression of CKD, especially in patients with diabetes [15]. Last but not least, insulin resistance is also a critical factor associated with smoking. Insulin resistance is an independent predictor of CKD and albuminuria [16,17].

In a study of 5 403 cases, it was found that smokers in males were more likely to develop proteinuria than non-smokers, and the risk was related to daily smoking. While, the relationship between smoking and proteinuria was not significant in females [18]. In addition, the creatinine clearance rate of smokers does not exhibit lower than that of non-smokers in the study of more than 28,000 cases [19]. This difference was more significant in males, but not in females. This effect seemed to be reversible after smoking cessation. Creatinine clearance is associated with the relative risk of proteinuria. Current smokers have a higher risk of developing proteinuria than non-smokers. The role of creatinine clearance in men and women is similar, and this effect is irreversible after smoking cessation. This is partly due to glomerulosclerosis damage caused by long-term hyperfiltration in smokers.

Smoking also affects the levels of inflammatory factors such as IL-6, IL-8, CRP, TNF-alpha, IL-10 and homocysteine in hemodialysis patients, and smoking also increases the risk of cardiovascular and cerebrovascular accidents, and the high incidence of insomnia and restless legs syndrome [20]. For long-term smokers, the incidence of peritonitis was significantly higher than that of non-smokers.

Conclusion

Smoking is a poor independent risk factor for CKD and ERSD, which should be take more attention. Although little is known about the role of long-term smoking in the progression of kidney disease, one thing is certain, that is, no matter what the etiology of kidney patients, smoking should be cessation.

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