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Is it time to tip your glass to prevent CKD?

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Abstract

Chronic kidney disease (CKD) is a major global medical and public health challenge. Based primarily on a modest number of prospective epidemiological studies it appears that alcohol consumption reduces the risk of CKD in the general population. Our understanding of the potential benefits of alcohol consumption on CKD is likely to evolve in the future and will be informed primarily from observational epidemiological studies.

Chronic kidney disease (CKD) is a global medical and public health problem. Studies to determine risk factors at the population level, especially those that are modifiable, are needed to stem the burden of these diseases. Koning and colleagues add to the relatively modest (compared to the substantial numbers of studies for cardiovascular disease) literature on the effects of alcohol intake on risk of CKD (1). From the well known PREVEND Study, they observed an inverse association between self-reported alcohol intake and risk of CKD in both men and women. Participants of this population - based study were observed over a period of 10 years; CKD was characterized as a serum creatinine cystatin-C estimated glomerular filtration (eGFR) <60 ml/min/1.73 m^2 (CKD-Epi equation) or/and urinary albumin excretion (UAE) >30 mg/24h. Compared with nondrinkers, the hazard ratios (and 95% confidence intervals) for CKD risk were 0.95 (0.77–1.17) for occasional, 0.84 (0.71–1.00) for light, 0.77 (0.63–0.95) for moderate, and 0.69 (0.49–0.99) for heavier alcohol consumption (findings were similar when either the eGFR or UAE outcome was considered). This relationship was observed among sub –groups including those based on age, sex, smoking status, presence of hypertension and hypercholesterolemia. Sensitivity analyses, which excluded nondrinkers at baseline to remove the potential bias due to “sick quitters” among the nondrinkers, did not appreciably change these risk relationship. The authors conclude that it is premature “to draw any firm conclusions regarding alcohol consumption to reduce the risk of CKD”, nevertheless there is “no grounds to discourage light to moderate alcohol consumption at least in terms of its renal effects”. This is a useful addition to the literature on this topic and adds further evidence that alcohol intake may reduce the risk of developing CKD. The study includes men and women, a relatively large

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sample size, extended follow-up, use of proven measures to assess kidney function, validation of self-report of alcohol intake by measurement of high-density lipoprotein and appropriate (measured) recommendations. More studies of the caliber of PREVEND are needed to better understand the influence of lifestyle factors on the occurrence of CKD.

What are we to make of these findings? As is the case for most epidemiological studies, it is important to consider several issues of study design that may influence the results. First, one component of their definition of CKD required a participant to have an eGFR <60 ml/min/ $1.73m^2$. No mention is made in the report as to how many values were required for a participant to be a case of CKD. This is in contrast to urine albumin excretion where two measurements were obtained (these appear to be taken three weeks apart, however, this time interval does not coincide with KDIGO Guidelines on CKD Evaluation and Management which require markers of kidney damage to be present for at least three months). This raises the possibility of disease misclassification. Second, no information is provided on the distribution of the types of chronic kidney disease (or further clinical confirmation of CKD) experienced by this population. The study group at baseline had an average age of about 48 years; a prevalence of hypertension of 26.4% and of type II diabetes of 2.0%. Diabetes and hypertension are the two most important risk factors/"causes" for CKD in many countries but their incidence is not described in this report. A description of the likely causes of CKD might provide further insight into how alcohol intake affects different kidney diseases. Fourth, there was considerable attrition of the original sample. A subset of the PREVEND cohort was included in this report. Among the 5,476 men and women included in the sample only 3,004 (54.8%) were studied about a decade later. It is not clear how this attrition could impact the findings; the authors do not provide information on reasons for lack of follow-up data. It is also important to note that that a substantial proportion (about 70%) of the original cohort of PREVEND Study participants had a urinary albumin concentration ≥ 10 mg/L (an entry criterion; proportion with at least this albumin concentration at study entry in the cohort reported here was not described) and may represent an "enriched" (for CKD progression) sample and is thus a modified populated-based cohort as described by the authors. Fifth, the generalizability of the findings might be limited (the racial and ethnic composition of the study group was not provided) since the participants were recruited from a single city in the Netherlands. Sixth, it would be of substantial value to describe the rate (and patterns, if possible) of decline of kidney function decline; such granularity may also provide further insight the magnitude of the beneficial effect of alcohol. Finally, lifestyle-based factors other than alcohol use, such as exercise and diet, were not described. Whether greater alcohol intake may serve as a surrogate for a healthier lifestyle (greater amount of exercise, better diet) remains uncertain.

The findings of the PREVEND Study should be considered within the context of prior longer-term prospective studies of the effect of alcohol intake on CKD. The preponderance of the epidemiologic evidence from such studies (2–8), but not all (9), suggests that alcohol intake may be protective of CKD/loss of kidney function among the general population. Interestingly, these findings have been reported from studies around the world including Norway, Japan, Australia, China, the United States and now the Netherlands. Findings have been quite consistent despite varied outcome measures of kidney function (e.g., ≥ 25% estimated GFR decline, eGFR ≤55 ml/min, rapid decline in annual eGFR (>3 ml/min/

1.73m^2), annual change in eGFR , eGFR or dipstick proteinuria, eGFR decline ≥10%, eGFR <60 mL/min/1.73m^2 doubling of albumin to creatinine ratio, end-stage renal disease and eGFR <60 ml/min/1.73m^2). Generally, beneficial effects were observed among men and women. In some instances there was a dose response of increasing alcohol intake with lower risk of CKD. Thus, there is reasonably consistent evidence from prospective epidemiological studies of the protective effect of alcohol intake for CKD. Plausible mechanisms of the beneficial effect abound and this evidence is buttressed by a large number of studies in cardiovascular disease showing similar relationships.

Where do we go from here? It is extremely unlikely that a randomized clinical trial of alcohol intake in the general population will ever be conducted to conclusively describe its effects on risk of CKD. However, our understanding of the effects of alcohol will likely evolve through epidemiological observational (non-randomized) studies of the general population (e.g., risk of CKD) and among persons with established CKD (to assess effects on CKD progression). Such studies are likely to inform whether alcohol intake is to be included in risk scores for CKD in the general population and in persons with CKD and possibly on guidelines/recommendations on CKD for both groups. The benefits of alcohol could have a substantial impact on the public health burden of CKD, especially the incidence of these diseases in the general population.

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