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Lifestyle and chronic kidney disease in elderly

Styl życia a przewlekła choroba nerek w wieku podeszłym

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Summary

Lifestyle has an important effect on many elements of health. Lifestyle-related risk factors for chronic kidney disease (CKD) include low fluid intake, alcohol abstinence, high alcohol consumption, smoking, low physical activity, high energy and macro- and micronutrient intake. Prolongation of human life span is associated with long-term exposure to environmental and behaviour factors; thus, the influence of lifestyle on health is increased in the elderly population. Although lifestyle-related risk factors can be modified, changing behaviors can be difficult. Furthermore, evidence demonstrating that lifestyle modification decreases the risk for CKD and improves kidney outcomes is limited. This is especially true in the elderly population because some effects of adverse behaviours, such as endothelial dysfunction and arteriosclerosis, are irreversible.

This review summarizes the current knowledge concerning the effects of lifestyle factors and their modification on the occurrence and progression of CKD, focusing on the elderly population.

Key words: CKD, lifestyle, elderly population

Streszczenie

Styl życia wywiera wpływ na wiele aspektów stanu zdrowia. Przyjmowanie zbyt małej ilości płynów, abstynencja, jak i nadmierne spożycie alkoholu, palenie papierosów, niska aktywność fizyczna, dieta wysokoenergetyczna o zaburzonym składzie makro- i mikroskładników są czynnikami ryzyka przewlekłej choroby nerek (PChN) związane ze stylem życia. Wydłużenie średniej długości życia człowieka wiąże się z dłuższym czasem ekspozycji na czynniki środowiskowe i związane ze stylem życia. Zatem niekorzystny wpływ tych zachowań na zdrowie jest szczególnie nasilony w wieku podeszłym.

Czynniki ryzyka związane ze stylem życia są modyfikowalne, jednak z wielu powodów, wprowadzenie zmian jest bardzo trudne. Dowody, na zmniejszenie ryzyka rozwoju PChN i poprawę czynności nerek w następstwie zmian stylu życia u ludzi są ograniczone, zwłaszcza wśród osób w wieku podeszłym. Wykazanie wpływu zmiany stylu życia na czynność nerek u osób starszych jest bardzo trudne ze względu na nieodwracalność zaburzeń, takich jak uszkodzenie śródbłonka i miażdżyca.

W niniejszym manuskrypcie dokonano podsumowania aktualnego stanu wiedzy dotyczącego wpływu stylu życia i jego modyfikacji na występowanie i progresję PChN, ze szczególnym uwzględnieniem populacji w wieku podeszłym.

Słowa kluczowe: PChN, styl życia, populacja w wieku podeszłym

INTRODUCTION

Lifestyle strongly influences many elements of health. Regular physical activity, smoking cessation, and appropriate intake of energy, fluids, and macro- and micronutrients can prevent many chronic conditions, improve function, and increase quality of life. However, over time, a positive energy balance due to sedentary lifestyle and excess energy intake leads to obesity, especially increased visceral fat deposits. Visceral obesity, which is prevalent in the elderly, is an important risk factor for hypertension, type 2 diabetes, and dyslipidaemia. In turn, obesity and its complications are risk factors for nephropathy and chronic kidney disease (CKD).

The human life span is also associated with long-term exposure to environmental and behavioural factors. Thus, the effects of adverse behaviors on health increase with aging. Although many lifestyle-related risk factors are modifiable, changing behaviors can be difficult. Moreover, little evidence exists to show that

lifestyle modification decreases the risk for CKD and improves kidney outcomes. It is particularly difficult to demonstrate the effect of modifying lifestyle-related risk factors on renal function, because some damage (e.g., endothelial dysfunction and arteriosclerosis) is irreversible.

This review summarizes the current knowledge concerning the effects of lifestyle and its modification on the occurrence and progression of CKD, focusing on the elderly population.

Fluid intake

The recently completed PolSenior study analyzed fluid intake in an elderly Polish population (1). The results showed that inadequate fluid intake (< 1000 ml/day) is more common with increasing age (13.3% at 65-69 years vs. 26.5% at > 90 years) and more common among women than men (25.3% vs. 19.4%). Inadequate fluid intake can reduce the glomerular filtration rate (GFR), which may partially explain the greater prevalence of low estimated GFR (eGFR) (< 60 ml/min/1.73 m^2) among women (2). The results of the PolSenior study revealed that 96.8% of elderly subjects who met the criteria for CKD were unaware they had the disease (2).

In daily clinical practice, physicians recommend generous fluid intake (≥ 4 l/day) for patients with CKD, with higher fluid intake for patients with higher serum creatinine levels. However, direct evidence for the association between adequate fluid intake and better kidney performance is lacking (3). Indirect evidence for this recommendation includes the results of two cross-sectional studies performed in different populations. These studies showed that increasing fluid intake was associated with a lower risk of CKD (defined as eGFR < 50 ml/min). Participants with the highest quintile of fluid intake (3.2 l/day) had the lowest risk (odds ratio [OR] = 0.50, 95% confidence interval [CI] 0.32-0.77) (4).

Better documented is the effect of dehydration on kidney function. Dehydration is an important risk factor for acute kidney injury. Adequate fluid intake may be even more important for individuals with undiagnosed CDK.

Alcohol consumption

Although there is no evidence that alcohol consumption impairs renal function, heavy drinkers may experience repeated episodes of acute kidney injury caused by dehydration (5). These episodes may induce or exacerbate interstitial kidney fibrosis. Studies analysing the relationship between moderate or heavy drinking and CKD have reported conflicting results. Several studies found that moderate alcohol consumption was not associated with renal dysfunction (6, 7). For example, a 14-year prospective study of 11 023 initially healthy men showed that those who consumed two to six drinks per week had a risk for serum creatinine elevation (> 133 μmol/L) similar to that of men who consumed no more than one drink per week (6). Similarly, results of the Australian Diabetes, Obesity, and Lifestyle (AusDiab) study (8) showed that moderate drinking decreased the risk of *de novo* development of renal function impairment (eGFR < 60 ml/min/1.73 m^2) in both men (OR = 0.34, 95% CI 0.22-0.59) and women (OR = 0.68, 95% CI 0.36-1.27). Funakoshi et al. recently reported that the prevalence of CKD is 40% lower in daily drinkers than in non-drinkers among professionally active men (9). In addition, Schaeffner et al. (6) found that consuming at least seven drinks per week had a beneficial effect on kidney function (OR = 0.71, 95% CI 0.55-0.92). In contrast, Shankar et al. reported that frequent alcohol consumption (> four drinks per week) increased the risk for CKD (OR = 1.99, 95% CI 0.99-4.01) in 5-year longitudinal analysis of 3.392 Americans without CKD (10). Additionally, Perneger et al. reported a higher prevalence of drinkers among patients with end-stage kidney disease than in age-matched controls (11).

The above-mentioned studies did not all focus on the elderly; however, results obtained in younger populations should be generalizable to the elderly population. Results of the PolSenior study show that 17.1% of the elderly Polish participants (men 27.0%, women 6.9%) reported frequent or very frequent alcohol consumption (12). Frequent alcohol consumption is associated with a lower risk for impaired kidney function (eGFR < 60 ml/min/1.73 m^2) in the elderly Polish population (22.4% vs. 36.5% of those who abstained from drinking) (13).

Smoking

Smoking is another known risk factor for CKD. A number of pathophysiological links between smoking and kidney damage have been identified, including the promotion of renal atherosclerosis (14), alterations in systemic and renal hemodynamics (15), and endothelial dysfunction (16). The relationship between smoking and end-stage kidney disease was initially shown in a longitudinal study of diabetic patients (17). A study by Biesenbach et al. reported that the rate of GFR loss was twice as high in smokers as in non-smokers (18).

The National Health and Nutrition Examination Survey II (NHANES II) reported that individuals who smoked > 20 cigarettes/day had a 2.3-times higher risk for CKD (7). In the AusDiab study, smoking was significantly associated with renal impairment in men (OR = 3.59) but not in women (19). In this study, lifetime exposure, but not current level of smoking, was related to lower eGFR (19).

Smokers may also have an increased risk of albuminuria. In the general Korean population, smoking was associated with a 38% higher risk for albuminuria (as assessed by urine dipstick) (20). Pinto-Sietsma et al. observed a 65% higher risk of low-grade albuminuria in smokers (21). Some studies have suggested that men are more vulnerable to develop albuminuria than women (22, 23). Haroun et al. estimated the risk of

CKD attributable to cigarette smoking in a population-based study as 31% (24). However, CKD risk related to smoking in the elderly population should be greater, because of long-term exposure to smoke along with other factors that lead to kidney injury.

There is no doubt that smoking cessation improves overall health. Smoking cessation may also specifically benefit the kidneys. For example, the rate of GFR loss among former smokers is lower than that of current smokers (25). Moreover, eGFR decline is more rapid among current smokers (-1.79 ± 0.35 ml/min/y) compared with former smokers (-1.54 ± 0.37 ml/min/y) (26). However, it has not yet been demonstrated that smoking cessation significantly improves kidney performance in elderly populations.

Physical activity

NHANES III reported that, compared with elderly individuals without CKD, those with CKD were more likely to be physical inactive (28.0% vs. 13.5%) and disabled (23.9% vs. 17.6%), defined as having difficulty with performing the activities of daily living (27, 28). However, the cross-sectional study design precluded the establishment of causality. Similar findings were obtained in a study carried out in the United Kingdom, where low eGFR (< 45 ml/min/1.73 m^2) was associated with impaired functional status (i.e., partial dependence on activities of daily living and lack of physical activity) in the elderly population (29). The effect of physical inactivity on mortality was similar between groups (CKD and non-CKD) (27).

The longitudinal AusDiab study found no association between leisure-time physical activity and eGFR in the general population (30). However, obese, physically inactive individuals were significantly more likely to have albuminuria at baseline compared with active, non-obese individuals. Baseline physical activity was not associated with the following outcomes: 5-year doubling of albumin/creatinine ratio, with a final albumin/creatinine ratio ≥ 2.5 mg/mmol in males and ≥ 3.5 mg/mmol in females in the absence of albuminuria at baseline and *de novo* development of low eGFR, and 5-year decline in eGFR > 10%, with eGFR ≥ 60 ml/min/1.73 m^2 at baseline and final eGFR < 60 ml/min/1.73 m^2 (30). Interventional studies addressing the effect of increased physical activity on CKD development or progression have not yet been performed. However, increased physical activity is known to have beneficial effect on weight reduction, muscle function (preservation of muscle mass), exercise tolerance, functional status, and quality of life.

A recent meta-analysis performed by Heive and Jacobson from the Cochran Renal Group (31) evaluated cardiovascular training, mixed cardiovascular and resistance training, resistance-only training, and yoga in adults with CKD. This study reported the benefits of regular exercise on fitness, walking capacity, cardiovascular dimensions, and health-related quality of life. Analysis of the cardiovascular dimensions showed a small decrease in resting blood pressure (systolic: -6.1 mmHg, 95% CI -2.1 to -10.1; diastolic: -2.3 mmHg, 95% CI -0.6 to -4.0) and heart rate (-6 beats per minute, 95% CI -2 to -10). Results of the PolSenior study also revealed that low physical activity is a risk factor for both low eGFR and CKD in the elderly Polish population (13).

Energy intake

The prevalence of obesity, according to World Health Organization diagnosis criteria, is estimated at 31.9% in the elderly Polish population (39.0% of women and 25.6% of men), and visceral obesity, as determined by International Diabetes Federation criteria, is estimated at 80.8% (89.5% of women and 72.8% of men) (32). The main cause of obesity is excess energy intake; thus positive energy balance may indirectly increase the risk of CKD. Cross-sectional and longitudinal clinical studies have demonstrated that obesity leads to changes in renal hemodynamics, such as glomerular hyperfiltration and albuminuria (33). Afferent arteriole dilation is a primary cause for glomerular hyperfiltration in obese individuals. Additional mechanisms include increased activity of the renin-angiotensin system, efferent arteriole vasoconstriction, and increased renal sympathetic tone (34, 35). Obesity is directly associated with albuminuria (36), and obesity-related complications, such as hypertension and type 2 diabetes, exacerbate kidney hemodynamic disturbances and increase the prevalence of albuminuria (37, 38). The direct effect of obesity on kidney function was confirmed by an interventional study showing that moderate weight loss related to energy restriction decreases proteinuria by approximately 30% in overweight diabetic subjects (39). Renal functional disturbances are more common in obese subjects than glomerulosclerosis development (40). The results of a 15-year follow-up study revealed a 10-fold increase in obesity-related glomerulopathy related to the increased prevalence of obesity and suggested that obesity-related glomerulosclerosis is associated with genetic predisposition, age, severity and duration of obesity, and concomitant diseases (41).

The effect of obesity on renal function has also been demonstrated by epidemiological studies that found a link between obesity and new-onset CKD. For example, in a cohort of 2585 patients followed for 20 years, each one-point increase in body mass index was associated with increased risk of new-onset CKD (OR = 1.23 per 1 standard deviation unite) (42). In addition, a higher rate of eGFR decline and more rapid progression to end-stage CKD has been shown in obese subjects (43). A large population study evaluating the risk for CKD progression over a span of 20 years confirmed that obesity is independent risk factor for CKD progression to end-stage disease (44). The effect of overweight duration on CKD development was demonstrated by a population-based case-controlled study, which reported a 3-fold higher risk for new-onset CKD in subjects

with a body mass index exceeding 25 kg/m^2 at 20 years of age, even after adjusting for the coexistence of hypertension and diabetes. Moreover, the coexistence of obesity and diabetes doubled the risk for new-onset CKD (45). However, the results of the PolSenior study suggest that among the elderly, the direct effect of obesity on renal function is abolished by co-morbidities such as hypertension and diabetes (13). Although overweight duration and the severity of obesity-related diseases are important risk factors for CKD, studies assessing the effect of weight loss on renal function in the elderly are lacking.

Macro- and micronutrient intake

Results of experimental studies suggest that diet composition also influences renal function. For example, high protein intake increases disturbances in kidney hemodynamics (e.g., glomerular perfusion pressure) in diabetic rats (46). In contrast, a low-protein diet slows the progression of kidney damage (47). Although data showing the long-term effects of high protein intake in humans are limited, acute protein load in diabetic subjects is associated with a 2-fold increase in filtration compared with healthy subjects (48). Dietary protein also appears to directly affect kidney function, because amino acid load promotes the *in situ* formation of advanced glycation end products (AGEs). Meat is the primary dietary source of AGEs (49). The AGE content of food is influenced by the distribution of protein and fat and by cooking (temperature, time, and moisture); high cooking temperature is the most important factor increasing AGE content in food (50). A low-AGE diet was shown to decrease systemic inflammation in diabetic subjects with and without CKD (51). Another study showed that AGEs are involved in the development of glomerulosclerosis in diabetic and non-diabetic humans (52).

Large epidemiological studies have confirmed that high protein intake (> 20% of energy intake) is associated with impaired kidney function. The results of NHANES III showed that low-grade albuminuria is related to protein intake in subjects with diabetes and hypertension (53). Kidney damage related to dietary protein intake has also been reported by a Dutch study of subjects aged 50 to 75 years old (54). Similarly, in a 11-year follow-up of women with eGFR of 55 to 80 ml/min/1.73 m^2, high protein intake was associated with 3.5-fold higher risk for worsening kidney function (> 15%) (55). Although meat protein has the most deleterious effect on renal function, excess intake of proteins from milk and plants can also cause CKD progression (56). The negative effect of high protein intake was demonstrated in subjects with impaired kidney function; data from studies in healthy subjects are lacking.

Results of meta-analyses assessing the effects of low protein intake on CKD have been inconsistent. For example, one meta-analysis reported that the protective effect of low protein intake was more pronounced in diabetic patients than in non-diabetic patients (57). These health benefits appear to be due to decreased AGE formation, which is toxic to renal tubules. However, one meta-analysis did not show protective effects of low protein intake in non-diabetic CKD patients (58), and another meta-analysis did not find a beneficial effect of low protein intake on eGFR (59). These discrepancies suggest that the effect of low-protein diets on kidney function may be related to CKD stage, with decreased benefits for individuals with more severe kidney damage.

A large randomized clinical study assessing the long-term effects of low protein intake found that dietary protein restriction reduced the risk of end-stage renal disease or death (60). However, studies assessing the effect of diet on CKD progression among the elderly have been limited to patients with impaired renal function. The prospective, randomized, multicenter Diet or Dialysis in Elderly study evaluated the safety and efficacy of a very-low-protein diet among Italian subjects older than 70 years and found that a very-low-protein diet postponed dialysis (median 12 months) and end-stage CKD (61). It should be stressed that long-term consumption of a very-low-protein diet is a risk factor for malnutrition, especially in elderly individuals. A protein intake of 0.8 g/kg/day may not be sufficient to meet the metabolic and physiological needs of all older people; a more reasonable target for protein intake in healthy elderly individuals is 1.5 g/kg/day or approximately 15 to 20% of total energy intake (62). Although American adults tend to consume more protein than is recommended (63). Protein intake in the elderly Polish population has not been assested. Taken together, these studies suggest that protein intake in elderly individuals should not be reduced below 0.8 g/kg/day, and this level of dietary protein restriction should be limited to diabetic patients with CKD stage IV and V. Protein intake above 1 g/kg/day should not be recommended for patients with early-stage CKD. It is important for 50 to 70% of dietary protein to come from foods that enhance its absorption, such as poultry, fish, and vegetables. In addition, culinary methods that do not promote AGE formation should be used, such as poaching, steaming, stewing, and boiling. There is a need for long-term prospective studies evaluating the benefits of low protein intake on CKD progression in elderly patients.

Dietary phosphate intake is related to diet protein consumption. High-phosphate diet appear to increase the risk for renal damage in patients with CKD through increased tubular lumen phosphorus burden and concomitant transepithelial phosphate transport, and increased calcium filtration and reabsorption leading to its precipitation in endothelial cells and peritubular space, resulting in renal inflammation and fibrosis (64). Thus, low-phosphate diets may slow renal damage in CKD. Results of the PREPARE study showed that phosphate level was inversely associated with renal function decline and mortality in patients with CKD stage IV and V (65). However, recently published results of NHANES III did not find an association between high dietary phosphorus intake and increased mortality.

An important source of dietary phosphate is food additives such as disodium phosphate, monosodium phosphate, potassium triphosphate, sodium acid phosphate, sodium hexamethaphosphate, sodium tripolyphosphate, tetrasodium pyrophosphate, and trisodium phosphate. Consumption of food additives, in which almost 100% of the phosphorus is absorbed, is estimated to contribute up to 25 to 50% of dietary phosphate (66). It is unclear whether consumption of phosphate additives is a major problem in the elderly population. Optimal phosphorus intake in moderate CKD, typical for the elderly population, may need to be determined by intervention studies (67).

Carbohydrate intake may indirectly influence kidney function by affecting body mass, insulin sensitivity, and lipid levels. The American Diabetes Association recommends that up to 60% of daily energy intake should come from complex carbohydrates, such as whole grains, fruits and vegetables, and non-fat or low-fat dairy products, and daily dietary fiber intake should be 20 to 40 g (68). High fructose consumption has been associated with higher postprandial triglyceride levels and increased risk for gout and kidney stones (69). Although studies evaluating the effect of carbohydrate intake on kidney function in the elderly are lacking, the association between CKD and diabetes in the elderly Polish population (13) suggests that the consumption of simple carbohydrates should be restricted. The potential benefits of these changes in carbohydrate intake on kidney function in the elderly require confirmation.

The American Health Association recommends that individuals at high cardiovascular risk, including those with CKD, limit fat intake to below 30% of energy content, saturated fat to below 7% of energy content, and cholesterol to below 200 mg/day (70). Limited data suggest that increased omega-3 and monosaturated fatty acids may slow CKD progression (71). However, studies evaluating fat intake on CKD progressionin the elderly population are lacking. A high intake of saturated fat may contribute to kidney damage by inducing dyslipidaemia. Results of large prospective trials suggest that statins slow CKD progression (72). Although more studies specifically evaluating the effects of statins on CKD and end-stage renal disease in the elderly are needed, the benefits of lipid-lowering therapy may outweigh the risk of side effects and drug interactions in this population (73). In addition, non-pharmacological treatment of dislipidaemia in elderly patients with CKD should be strongly encouraged.

Although sodium intake in the elderly Polish population has not been reported, results of the MINISAL study reported higher sodium intake and lower potassium intake than recommended in Italian adults aged 35 to 79 years (74). In an animal study, high salt intake increased angiotensin II receptor 1 expression, aldosterone synthesis, and angiotensin-converting enzyme (ACE) activity (75). Moreover, high salt intake impaired renal functionin renal damage models, and salt restriction had a protective effect on kidney function in a renal ablation model (76). Cross-sectional studies have also shown that salt restriction slows renal disease progression in humans (77). Conversely, a recent study reported that salt intake exceeding 14 g/day may blunt the antiproteinuric effect of ACE inhibitor therapy and increase the risk of CKD progression to end-stage, independent of blood pressure control (78). Thus, restricting sodium intake in patients with CKD should be recommended to improve outcomes.

CONCLUSION

Data demonstrating the effect of lifestyle interventions on CKD progression in the elderly population are limited. Results of previously published studies suggest that lifestyle-related risk factors play both direct and indirect roles in CKD development. Although lifestyle modifications may not be able to reverse endothelial damage and arteriosclerosis in elderly patients, dietary changes and adequate fluid intake may slow or prevent CKD progression. Lifestyle changes are needed in younger populations to prevent obesity, obesity-related complications, and overall cardiovascular risk, thereby reducing the incidence of CKD among the elderly.

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