Editors’ Note: In the American Academy of Neurology and the American Epilepsy Society practice guideline on sudden unexpected death in epilepsy (SUDEP) incidence rates and risk factors, Harden et al. made recommendations to clinicians caring for people with epilepsy including the need to inform their patients about SUDEP, the most common cause of epilepsy-related death. Commenting on the guideline, Stanton et al. note that certain pediatric populations (children with Dravet syndrome, Dup15q syndrome) face a significantly higher risk of SUDEP. They also suggest accurate surveillance data and expanded research to understand the mechanisms of SUDEP. The authors of the guideline agree. In “Dementia risk in renal dysfunction: A systematic review and meta-analysis of prospective studies,” Deckers et al. conducted random effects meta-analyses on the prospective association between potential markers of renal dysfunction and development of cognitive impairment or dementia. The authors concluded that albuminuria was a useful marker of renal dysfunction for screening the development of cognitive impairment or dementia. Prof. Kawada raises 2 concerns about the study. He cites another systematic review that evaluated the relationship between chronic kidney disease and physical frailty and cognitive impairment. In that study, the mechanism of chronic kidney disease–induced physical frailty and cognitive impairment was also unclear. He suggests multidimensional therapeutic interventions to study the effect of chronic kidney disease on subsequent physical frailty and cognitive impairment. Second, he suggests also studying the effect of cognitive impairment on subsequent kidney dysfunction. Deckers et al. agree that there is a need for additional studies on shared biological pathways between albuminuria and cognitive impairment or investigating the effect of multidimensional therapeutic interventions. They also agree that more mechanistic studies are needed to evaluate the bidirectional relationship between renal dysfunction and cognitive impairment or dementia.

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The new guideline by the American Academy of Neurology (AAN) and the American Epilepsy Society (AES) on sudden unexpected death in epilepsy (SUDEP) is a landmark.1 The communication between medical professionals and patients about SUDEP risk remains unacceptably low. Tragically, family members often first learn about SUDEP after their loved one’s death. Every patient and parent deserves to know the risks of epilepsy. For the first time, the AAN and AES recommend that neurologists inform them about SUDEP, the most common cause of epilepsy-related death.1

Generalized tonic-clonic seizures increase SUDEP risk; the greater their frequency, the greater the risk.2,3 Minimizing seizures through specialized medical care and strategies to reduce breakthrough seizures can reduce risk.2,3 Since SUDEP happens more often in sleep,2 nighttime supervision or monitoring may help and should be part of the patient/provider conversation. While the report assigns a blanket SUDEP risk ratio to children,1 certain pediatric populations (children with Dravet syndrome, Dup15q syndrome) face a significantly higher risk.4,5

All medical professionals should use these recommendations to initiate an honest and ongoing conversation tailored to their patient’s risk. We urgently need accurate surveillance data and expanded research to understand the mechanisms and save lives.

The authors concluded that albuminuria is speculated by several suggested potential mechanisms. The mechanism of chronic kidney disease is not fully understood. I have 2 concerns about their study. First, Shen et al. conducted a systematic review to evaluate the relationship between chronic kidney disease and physical frailty and cognitive impairment. They concluded that chronic kidney disease was a potential cause of frailty and cognitive impairment. The mechanism of chronic kidney disease–induced physical frailty and cognitive impairment has not been confirmed, and multidimensional therapeutic interventions would be useful to know the effect of chronic kidney disease on subsequent physical frailty and cognitive impairment.

Second, the effect of cognitive impairment on subsequent kidney dysfunction should also be evaluated by a meta-analysis on prospective studies. Worsening of albuminuria should also be evaluated by a meta-analysis on prospective studies. The development of cognitive impairment or dementia is speculated by the progression of cognitive impairment or dementia, and the bidirectional association between renal dysfunction and cognitive impairment or dementia should be comprehensively evaluated.

marking microvascular changes is not settled. Therefore, we embrace Prof. Kawada’s suggestion for future studies on shared biological pathways or investigating the effect of multidimensional therapeutic interventions.

Prof. Kawada’s second concern is about the effect of cognitive impairment on subsequent renal dysfunction. We agree that more mechanistic studies are needed that focus on the bidirectional relationship between renal dysfunction and cognitive impairment or dementia. These studies should include repeated assessment of both cognitive and kidney function, apply consistent methodology (e.g., neuropsychological test battery or formula/equation for estimating kidney function), and incorporate information on important confounders and mediators.


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