

DIABETIC CONTROL IN ELDERLY AND COGNITIVE DECLINE

Nur Riviati¹, Ridzqie Dibyantari^{1*}, Rini Nindela², Bintang A Prananjaya³, Afifah Z Salsabila⁴, Aidi A Putri⁴ ¹Geriatric Division of Internal Medicine Department, RSUP Dr Moh Hoesin/ Universitas Sriwijaya ²Neurology Department, RSUP Dr Moh Hoesin/Medical Faculty of Universitas Sriwijaya ³Psychiatry Department, Medical Faculty of Universitas Sriwijaya ⁴Medical Faculty of Universitas Sriwijaya Email: ridzqie.dibyantari@ui.ac.id

ABSTRACT

As the population aging increasing rapidly worldwide, including in Indonesia, the health problems come as consequences as well as delivering health and social care. At this moment 14% of Indonesian consist of aging population, defined as those above 60-year-old of age. Aging ameliorates normal hormonal and immunology system, consequently elderly are prone to infection as well as metabolic disorder, namely diabetes. Good glycemic control in elderly can prevent cognitive decline such as mild cognitive impairment and dementia. The aim of this paper is to discuss the importance of diabetic control in elderly, focusing on cognitive decline related to diabetes

Keywords: diabetes, dementia, elderly



INTRODUCTION

Over the past century there have been very remarkable changes in numbers and characteristics of elderly throughout the world.¹ The growth of older population has resulted from a general increase in the overall population size but has been particularly affected by major declines in several cause of mortality.¹ The increased survival of elderly also accompanied by the decline of death rates, and this will cause the population doubled dramatically.^{1,2}

Aging is a process that converts young adults, most of them are healthy and in no need of assistant from doctors, into older adults whose deteriorating physical condition leads to progressively increasing risk of illness and health.² Diseases are the most silent consequences of aging, but aging produces many changes not classified as diseases.²

Diabetes is one of the leading chronic medical conditions among elderly population putting elderly at risk for cognitive decline, vascular complication, as well as dependency and several degenerative diseases.³ Type 2 diabetes (T2DM) is the most prevalent form of diabetes in elderly and is considered as age-related disorder.³ Elderly are at risk of developing T2DM due to several interacting factors such as genetic susceptibility, lifestyle, and aging influences. Interaction of these factors then reduce insulin secretory capacity by pancreas and insulin resistance by peripheral tissues.³

Cognitive decline is recognized as an important comorbidity of T2DM and there is strong epidemiological evidence that links diabetes and cognitive decline.⁴ Deficit in cognitive functioning can roughly divided into three stages, i.e., diabetes-associated cognitive decrements, mild cognitive impairment (MCI) and dementia.⁴

This paper will discuss diabetic control in elderly and cognitive decline as a basis of our pro bono work in educating public, pre-elderly, and elderly population particularly.

METHODS

This community service activity was conducted as part of Medical Faculty of Universitas Sriwijaya Dies Natalis. We held online educational seminar via the Zoom meeting on Saturday, September 19th, 2021. This activity was held in collaboration between Geriatric Division of Internal Medicine Department RSUP Dr Moh Hoesin, Neurology Department and Psychiatry Department Medical Faculty of Universitas Sriwijaya.

DISCUSSION

Type 2 Diabetes (T2DM) is the most prevalent type of diabetes in elderly and is an age-related disorder. The diagnostic criteria used for this population is the same as other age group. Older adults are at risk developing T2DM because of the interaction of genetic susceptibility,



lifestyle and aging process causing reduced insulin secretion and insulin resistance in peripheral tissue.3

Hyperglycemia develops when there is imbalance of glucose production and glucose intake as opposed to insulin-stimulated glucose uptake in target tissue. Although insulin resistance contributes to altered glucose homeostasis, current evidence has found out that the direct effect of aging on diabetes pathophysiology is through impairment of b-cells function causing a decline in insulin secretion.³



Figure 1—Model for age-related hyperglycemia (12). Aging has direct effects on β -cell proliferation and function and contributes indirectly to impaired insulin sensitivity through lifestyle-related and comorbidity-related risk factors. The insulin resistance in turn may contribute to further impairment of β -cell function.

Evidence showed that higher HbA1c levels are associated with diabetes-associated cognitive decline, but the strength is weak. Glycated hemoglobin levels have also been linked to dementia risk in people without diabetes. Whether HbA1c levels are also related to dementia risk and depression is less clear. Observational studies have reported benefit of cognition of some glucose lowering compound over others, which suggests the need to assess outcomes other than glucose level and HbA1c to understand the effect of anti-hyperglycemia drugs on cognitive function.⁴

CONCLUSION

Glycemic control in elderly is important to preserve cognitive function and reduce risk of dementia. Routine follow up to physicians, active lifestyle, active socializing may be beneficial for successful aging.



REFERENCES

- 1. Brivio P, Paladini MS, Racagni G, Riva MA, Calabrese F, Molteni R. From healthy aging to frailty: in search of the underlying mechanisms. Curr Med Chem. 2019, 26(20): 3685-3701.
- Lombard DB, Miller RA, Pletcher SD. Bology of aging and longevity. In Halter JB, Ouslader JG, Studenski S, High KP, Asthana S, Supiano MA, et al, editors. Hazzard's Geriatric Medicine and Gerontology, 7th ed. McGrawHill, New York. 2017. p33-48.
- 3. Lee PG, Halter JB. The pathophysiology of hyperglycemia in older adults: clinical consideration. Diabetes Care. 2017, 40: 444-453.
- 4. Biessels GJ, Despa F, Cognitive decline and dementia in diabetes: mechanisms and clinical implications. Nat Rev Endocrinol. 2018, 14(10): 591-604.