Cognitive Decline in Chronic Disease

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Abstract

Background: Cognitive decline in adult individuals on treatment for diabetes or hypertension or HIV can have significant impact on the functional status and quality of life of an individual in chronic care for non-communicable diseases like diabetes, systemic hypertension and even for communicable diseases like chronic HIV and the HBA1c. Blood pressure measurement and HIV-viral load assays are used as quality indicators in the management and prevention of complications from these diseases respectively and the cause of cognitive decline can be difficult to ascertain when these conditions coexist.

Case 1: A 71 year old woman developed unexpected cognitive decline on treatment for hypertensive heart disease controlled on telmisartan 80mg OD, bisoprolol 5mg OD, rosuvastatin 10mg OD, aspirin 75mg OD, Lasix 20mg once daily and on Antiretroviral Therapy (ART) with viral suppression <50 copies/mL. Her ART history had AZT/3TC/NVP, changed to ABC/3TC/NVP due to severe anaemia in 2004. She suffered from osteopenia and arm fractures from minor trauma. And after ART switch to ABC/3TC/DTG in 2020, she developed diabetes and ART was switched to RLT/ATVr. The brain CT imaging was non-diagnostic but MRI revealed focal gray mater lesions considered lacunar infarcts. The routine CSF analysis was normal, however had a CSF viral load of .415,092 copies/mL.

Case 2: A 55 year old man, on treatment for systemic hypertension, Type-2DM and chronic HIV,

with excellent control for all the three conditions. developed unexpected cognitive loss with focal seizures and visual hallucinations. His treatment included gliclazide 80mg OD, empagliflozin 25mg OD, pioglitazone 30mg OD for DM, telmisartan/H (80/12.5) mg OD and S-Amlodipine 5mg OD for hypertension. His ART history included TDF/3TC/ EFV as initial therapy, that was changed to ABC/3TC/ DTG in September of 2021 due to raised creatinine but due to onset of diabetes in September 2023, the ART was switched to atazanavir/ritonavir plus raltegravir and he maintained viral suppression <50 copies/mL but in May 2024, he developed focal seizures, visual hallucinations and memory loss. The CSF examination was normal, except for a CSF Viral load of 27,849 copies/mL.

Discussion: Both of these patients had white mater MRI lesions, consistent with lacunar infarcts, with normal CSF chemistry and microscopy, however despite the excellent HIV-VL suppression in the peripheral blood, they had high CSF viral loads, with HIV drug resistance. They both attained complete cognitive recovery after a drug-resistance guided ART switch.

Conclusion: CNS-sequestration of HIV infection is a potentially reversible cause of cognitive loss among patients on ART with HIV viral suppression and it should be included in the clinical evaluation.

Key words: Cognitive decline, Diabetes, Hypertension