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Relationship between neurocognitive impairment and cardiovascular risk in an HIV-infected patient: a case report

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ABSTRACT: Neurocognitive and cardiovascular diseases are relevant problems in HIV-infected patients in the highly active antiretroviral therapy (HAART) era. Current knowledge suggests a possible link between cardiovascular disease and neurocognitive impairment in HIV-infected patients.
We report a clinical case showing a correlation between cardiovascular risk factors and lower baseline cognitive performance. We evaluated cardiovascular assessment with Framingham risk score (FRS), carotid ultrasound and anthropometric measures; cognitive status was analyzed using 8-test neurocognitive battery. Cardiovascular risk and neurocognitive impairment were closely related in our patient. Increased use of cardiovascular and neurocognitive evaluation tests can help in the clinical practice to quickly identify those needing therapeutic interventions, in order to delay disease progression.

Key words: HIV, Neurocognitive impairment, Cardiovascular risk.

INTRODUCTION

Although the introduction of highly active antiretroviral therapy (HAART) for treatment of HIV/AIDS has prolonged life and reduced mortality for opportunistic infections[1], a progressive increase in the prevalence of age-related comorbidities has been observed in the HIV-infected population[2-13].

HIV-infected patients have higher cardiovascular risk because of the association of traditional cardiovascular risk factors and use of HAART[14], as well as the presence of coinfections such as HCV. Similarly, concomitant risk factors increase are responsible for the increased prevalence of neurocognitive impairment[15]. HAART caused a reduction of HIV-associated dementia (HAD), but a remarkable increase of mild forms, such as asymptomatic neurocognitive impairment (ANI) and mild neurocognitive disorder (MND), has been reported[16].

Some published studies underline a possible relationship between cardiovascular risk factors and lower baseline cognitive performance[17].

CASE REPORT

Our patient was a 55-year old man, previous intrevenuous drug user, diagnosed with HIV infection in 1996. His CDC stage was B2, CD4 cell count nadir 130 cells/mm3 (6%) and he had HCV (genotype 4c) coinfection. He started antiretroviral therapy one year after diagnosis, with suboptimal adherence. He developed resistance to antiretroviral therapy (NNRTI and NRTI), lipodystrophy and HCV-related hepatopathy. His current HIV RNA was 200 copies/ml, CD4 cell count 880 cells/mm3 (28%), with CD4/CD8 ratio of 0.56. The patient was currently receiving dual therapy with darunavir/ritonavir 800/100 mg once a day and raltegravir 400 mg twice a day.

The patient had a low school level, was a smoker and practiced irregular physical activity. His recent history was negative for alcohol and psychoactive medications use.

He denied a family history of cardiovascular disease, neurodegenerative diseases, hypertension and diabetes. Furthermore, he had not previous cardiovascular events. His BMI was 21.9 Kg/m^2, and blood pressure was 120/70 mmHg.

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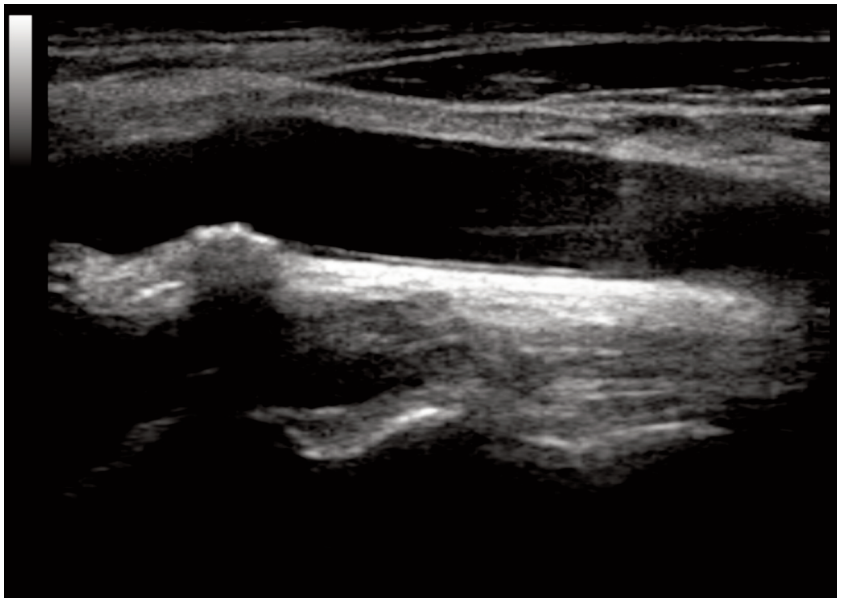

Figure 1. The ultrasound evaluation shows a fibrocalcific ateromatous plaque of the posterior wall of the right carotid bulb.

Laboratory results showed total cholesterol level = 163 mg/dl, LDL = 88 mg/dl, Tg = 224 mg/dl, HDL = 30 mg/dl. Cardiovascular risk estimated with the FRS was 22.9% at 10 years and 10-year ASCVD Risk Estimator was 10.9%.

Carotid ultrasound showed a normal intima media tickness (IMT) with left and right value respectively of 0.56 and 0.57 mm. However, he had an atheromatous fibrocalcific plaque at the right bulb (Figure 1).

We performed a complete neurocognitive assessment with a battery of 8 tests to evaluate the patient neurocognitive status, i.e. Trail Making Test-A (TMT-A) which explores the subject abilities in focus and attention, Trail Making Test-B (TMT-B) which explores executive functions and cognitive flexibility, Digit Span (DSp) which evaluates short-term memory and cognitive flexibility, immediate (Rey 15) which evaluates short-term memory and delayed (D-Rey 15) recall of Rey's 15 which explores long-term memory, Digit Symbol (DSy) which evaluates information processing speed and visual-motor skills, Letter fluency test (Flu) which allows to explore verbal fluidity, Rey complex figure (R-Fig) to evaluate praxic and visual construction skills. We also considered 2 global z-score NPZ-4 (TMA-Z+TMB-Z+DSp-Z+DSy-Z) and NPZ-8 (TMA-Z+TMB-Z+DSp-Z+DSy-Z+15-Z+15dif-Z+Flu-Z+FigR-Z) and we also evaluate the presence of depression by using questionnaires (CDI and DS14) that scored negative, since depression could negatively influence neurocognitive skills. We observed that this patient presented impaired neurocognitive skills with: TMA-Z (2.1), TMB-Z (-1.9), DSp-Z (-3.1), 15-Z (-2.2), 15 dif-Z (-2.3), DSy-Z (-1.7), Flu-Z (-1.8), FigR-Z (-6.7), NPZ-4 (-2.2), NPZ-8 (-2.7).

DISCUSSION

The use of HAART has dramatically changed the natural history of HIV infection prolonging the survival and reducing morbidity connected to opportunistic infections and mortality[18].

Nevertheless, this success implies a progressive ageing in HIV-infected populations, with an increase in age-related non-infectious diseases.

Although HAART caused a reduction of AIDS dementia complex, paucisymptomatic forms of neurocognitive HIV-associated diseases have increased over time[19-25].

HIV-infected patients frequently present many cardiovascular risk factors including alterations of lipid profile, arterial hypertension, diabetes, cigarette smoking. These factors are associated with neurocognitive decline in HIV-negative population too[26].

In our study, we explored the association between neurocognitive skills and cardiovascular risk in a HIV-infected patient. Our results show that the patient had a high global cardiovascular risk at 10 years according to FRS and ASCVD Risk Estimator.

We observed that this high cardiovascular risk could be connected to low school level, longer duration of HIV and antiretroviral therapy, and coinfection with HCV. The relationship between cardiovascular risk and school level was already observed in earlier studies conducted on HIV-negative subjects and represents a possible surrogate of a less healthy lifestyle[27].

The global cardiovascular risk of this particular patient, which was asymptomatic for cardiovascular disorders, could be crucially connected to impaired neurocognitive skills. Furthermore, it suggests that the study of neurocognitive performance represents a crucial point not only to diagnose early neurocognitive impairment, but also to predict subclinical cardiovascular disease in these patients.

CONCLUSIONS

Our case report suggests that neurocognitive disorders and subclinical cardiovascular disease are closely connected in HIV-infected patients.

The use of simple and fast tests for neurocognitive evaluation, as well as the evaluation of cardiovascular

risk should be implemented in the management of all HIV-infected patients, to quickly identify those at high risk, who can benefit from interventions able to delay clinical progression to advanced disease.

Conflict of Interests:
The Authors declare that they have no conflict of interests.

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