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Persistent immune activation in HIV-associated pulmonary arterial hypertension

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Background: Pulmonary arterial hypertension (PAH) is a potentially deadly complication in HIV infection but few is known on its pathogenesis.

Material/methods: We compared immune activation markers in eight HIV-PAH patients, eight HIV-infected patients without PAH (HIV group) and eight healthy donors (HD).

Results: PAH appeared 12.2 years after HIV infection, while 7/8 patients were receiving antiretroviral treatment (ART) with a mean LT-CD4+ count at 389 cells/mL and HIV plasma viral load below detection in half of them. At inclusion, mean HIV infection duration was similar in HIV-PAH group (19.1 years) and HIV group (19.2 years), p =0.98. Mean exposure time to ART was similar between HIV-PAH group, (13.7 years) and HIV group (14.1 years) (p =0.91). Mean LT-CD4+ count was 446 cells/mL in HIV-PAH compared to 631 cells/mL in HIV group (p= 0.12).

Mean soluble (s)CD14 plasma level was increased in the HIV-PAH (1.88μg/mL) compared to HIV (1.27μg/mL), p= 0,01 and HD (1.02μg/mL), p=0,02. Mean TNF plasma level was higher in HIV-PAH (5,65 pg/mL) compared to HIV (2,26 pg/mL, p=0,04) and HD (0,98 pg/mL, p=0,03). Mean IL-6 plasma level was strikingly elevated in HIV-PAH group (122 pg/mL) over HIV (2,41, p=0,04), but not between HIV and HD (1,83 pg/mL, p=0,11). We found a significant decrease of non-classical monocytes (CD14+CD16low) in HIV-PAH with an overrepresentation of CD163+ monocytes. Activated lymphocytes CD8+CD38+HLADR+ were higher in the HIV-PAH (3.30%) compared to HIV (1.45%, p=0,008) and HD group (1.65%, p=0,008). Mean endothelin plasma level, a key protein in PAH pathogenesis, was strikingly elevated in the PAH group (8,6 pg/mL) compared to HIV group (1,9 pg/mL, p= 0,0005). In 4 patients we demonstrated an intrapulmonary overproduction of endothelin during right heart catheterization.

Conclusions: Altogether our results demonstrate that, compared with asymptomatic HIV-infected patients, HIV-PAH is associated with a higher level of persistent immune activation.