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Effects of HIV Infection on Pulmonary Artery Heaviness in Children

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Introduction

Since the advent of highly active anti-retroviral therapy, HIV-related mortality has decreased dramatically. As a consequence, patients are living longer, and HIV infection is becoming a chronic disease. Patients and caretakers have to deal with chronic complications of infection and treatment, such as cardiovascular diseases, which now represent an important health issue, even in the pediatric population. Prevalence of pulmonary arterial hypertension (PAH) in the adult HIV population is around 0.4-0.6%, which is around 1000- to 2500-fold more prevalent than in the general population. In recent adult PAH registries, HIV has been identified as the fourth cause of PAH, accounting for approximately 6-7% of cases. Therefore, regular screening is recommended in HIV-infected adults by many experts. If HIV-associated PAH is mainly reported in HIV-infected adults, pediatric cases have also been, albeit rarely, described. This scarcity may be due to a very low PAH prevalence, or due to the lack of systematic cardiovascular screening in pediatric patients. As PAH may manifest only years or decades after infection, a systematic screening should perhaps also be recommended to HIV-infected children. In this context, we retrospectively looked for PAH screening in children included in our national Swiss Mother and Child HIV cohort study.

Description

A questionnaire was sent to all pediatric infectious disease specialists taking care of HIV-infected children in the cohort. The questions tried to identify symptoms suggestive of cardiovascular risk factors and asked which screening test was performed. In the 71 HIV-infected children for which we obtained an answer, no child was known for PAH. However, only two had been screened for PAH, and the diagnosis was not confirmed. In conclusion, PAH in HIV-infected children is possibly underestimated due to lack of screening. Systematic echocardiographic evaluation should be performed in HIV-infected children. WHO estimated in 2010 that 34 millions of humans were infected with HIV, 10% being younger than 15 years old. In children, infection mainly happens through transmission between mother and child during the pre-, peri-, or postnatal period (through breastfeeding). The risk of infection differs according to the mode of transmission, and also depends on the viremia of the source patient. The infected person often presents an asymptomatic period lasting 5-10 years without treatment, depending on viremia and CD4 T-cell count [1].

Since the advent of highly active anti-retroviral therapy (HAART), HIV-related mortality has decreased dramatically both in adults and children. As a consequence, patients are living longer, and HIV infection is becoming a chronic disease. Patients and caretakers have to deal with chronic complications of the infection and the treatment, such as cardiovascular disease, which now represent an important health issue in this patient population. Because of the lack of randomized control studies, it is difficult to differentiate the etiological role of the virus itself from the impact of HAART in pulmonary arterial hypertension (PAH), as well as other cardiovascular manifestations. PAH is a serious disease of the pulmonary arteries characterized by vascular remodeling due to dysfunctional proliferation of smooth muscle and endothelial cells. It leads to an increase in pulmonary vascular resistance and pulmonary arterial pressure (PAP). PAH classification was updated during the World Symposium on Pulmonary Hypertension in 2013. This classification gives more importance to pediatric PAH compared to the previous Danapoint Classification [2-4].

Despite this, no systematic cardiovascular screening is usually suggested to HIV-infected children. To demonstrate this, we sent a questionnaire to all infectious diseases specialists taking care of HIV-infected children in Switzerland, part of the Swiss Mother and Child HIV (MoCHIV) cohort study to evaluate if they look for PAH symptoms and perform screening procedures. We will first review HIV-related PAH (HIV-PAH) and second describe the results of our questionnaire. Pulmonary arterial hypertension was described in HIV-infected patients for the first time in 1987 (13). Since then, HIV has been identified as one of the associated forms of PAH, accounting for approximately 6-8% of cases (14-17). However, because of the high HIV prevalence worldwide and the poor screening, HIV-PAH may be an important unrecognized cause of PAH. Even if confounding factors, such as drug use and co-infection with hepatitis C virus (HCV) have been reported , more than 80% of PAH cases in the HIV population are directly related to HIV and/or its treatment.Pulmonary arterial hypertension is defined as a resting PAP >25 mmHg, associated with a pulmonary arterial occlusion pressure (PAOP) ≤15 mmHg . HIV-PAH diagnosis can only be confirmed once all other possible etiologies have been excluded [5].

Diagnosis relies on clinical symptoms, such as fatigue and dyspnea, as well as clinical findings, such as an increase in the pulmonary component of the second heart sound, a tricuspid regurgitation murmur or a right fourth heart sound, and signs of right heart failure. Chest X-rays may show cardiomegaly and pulmonary arteries enlargement, and the electrocardiogram shows right ventricular hypertrophy with right axis deviation. When PAH is suspected, echocardiography is the most useful diagnostic tool. Confirmation with right heart catheterization (RHC) is the gold standard. Some authors recently proposed a diagnosis algorithm [6].

Prevalence of HIV-PAH is around 0.2–0.6%, which is 1000- to 2500-fold more prevalent than in the general population . However, as PAH may present with very scarce symptoms and no systematic screening is usually offered in the HIV-infected population, these results are probably underestimated. Most previously mentioned studies included only symptomatic patients. When all HIV-infected patients of a cohort are included and/or when using only echocardiography without RHC confirmation, HIV-PAH prevalence reaches 2.5–10%. Compared to patients with HIV-PAH diagnosed using RHC, mean PAP values were 20 mmHg higher when diagnosis was established only with echocardiography, suggesting that echocardiography overestimates PAP. Hsue described pathological PAP by echocardiography in 35% of their HIV-infected cohort. In a recent study, HIV-PAH prevalence was 57%, but only patients with tricuspid regurgitation were included and methods to calculate PAP were different [7].

HIV-related PAH physiopathology is not completely understood, is

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probably multifactorial, and includes genetic factors. It is hypothesized that HIV acts as a trigger, maintaining chronic inflammation and immune activation. Histological features, which closely resemble those seen in idiopathic PAH, show in most cases a pulmonary arteriopathy with so-called "plexiform lesions" associated with concentric laminar intimal fibrosis, medial hyperplasia, and white cells. They suggest chronic inflammation. Occasionally, plexiform lesions are lacking, possibly because it is an earlier stage of the disease. More rarely, thrombotic arteriopathy of the small vessels has been described.

Conclusion

As simian immunodeficiency virus (SIV) infection of macaques shares many characteristics with HIV infection, these animals have been used as non-human primate model of HIV: plexiform lesions were only recovered among macaques infected with SHIV, a viral construct containing the HIV Nef protein in an SIV backbone, but not among SIV alone-infected macaques. In contrast, in another animal study, all SIV and SHIV macaques had elevated PAP associated with histological changes consisting mostly of intimal and medial hyperplasia. PAP values were higher in SIV and SHIV macaques than in healthy macaques, and PAP increased as early as 3 months after SIV infection.

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