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Editorial

Diagnostic and Complications occur Obey in HIV-1-Infected Young People and Adolescents with Triple-class Antiviral Drugs Drug-Resistant Viruses in Seville

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Abstract

Drug resistance mutations jeopardise the success of antiretroviral therapy in children infected with the human immunodeficiency virus type 1 (HIV-1). We present the virologic and clinical outcomes of the Madrid cohort of perinatally HIV-infected children and adolescents after triple-class drug-resistant mutations were chosen (TC-DRM). According to IAS-USA-2013, we assigned patients with HIV-1 variants with TC-DRM to nucleoside reverse transcriptase inhibitors, nonnucleoside reverse transcriptase inhibitors, and protease inhibitors. From 2000 to 2011, we recovered pol sequences or resistance profiles, as well as clinical-immunologic-virologic data from the time TC-DRM was detected until December 2013. From 2000 to 2011, viruses carrying TC-DRM were found in 48 (9%) of the 534 children and adolescents studied, rising to 24.4% among the 197 with resistance data. 95.8% of them were diagnosed before 2003. 91.7% were Spaniards, 89.6% had HIV-1 subtype B, and 75% had mono/dual therapy as their first regimen. D67NME, T215FVY, M41L, and K103N (retrotranscriptase) were the most common TC-DRMs found in 50% of them (protease). Darunavir, tipranavir, etravirine, and rilpivirine susceptibility rates were 67.7%, 43.7%, 33.3%, and 33.3%, respectively, and all reported high resistance to didanosine, abacavir, and nelfinavir. Despite the presence of HIV-1 resistance mutations to the three main antiretroviral families in our paediatric cohort, some drugs, primarily the new protease inhibitors and nonnucleoside reverse transcriptase inhibitors, retained their susceptibility. These findings will contribute to better clinical management of HIV-infected children with triple resistance in Seville (Dagum C et al.,1997).

Keywords: Adolescents, Antiretrovirall treatment Children, Drug-resistant viruses, HIV-1, Madrid cohort, Paediatric population, Triple class failure, Virologic failur

INTRODUCTION

(Dubey A et al., 2009) By the end of 2013, there were 35 million people infected with the human immunodeficiency virus, including 2.5 million children under the age of 15. (Färe R et al., 2007) Access to antiretroviral therapy (ART) has altered the course of infection, lowering morbidity, mortality, and HIV viremia while (Anderson SN et al., 2019) also extending the life expectancy of HIV-infected adults and children. New effective combination therapies have improved clinical outcomes and raised the thresholds for drug resistance mutation development (DRMs). New antiretroviral (ARV) therapy has expanded treatment options for children. (Austin RS et al., 2011) However, over 100 mutations in the HIV-1 genome have been identified that confer varying degrees of resistance to one or more ARVs. (Avni R et al., 2017) According to international guidelines, first-line antiretroviral therapy (ART) for HIV-1-infected children and adults consists of two (Baumann K et al., 2020) nucleoside reverse transcriptase inhibitors (NRTIs) and a third agent from a different class, either a nonnucleoside reverse transcriptase inhibitor (NNRTI) or a

ritonavir-boosted protease inhibitor (RBP) (PI). ART failure with a NRTI/NNRTI combination occurs in 10% to 30% of patients each year, and is primarily caused by NRTI and/or NNRTI resistance (Baležentis T et al., 2021).

(Balsalobre-Lorente D, et al., 2019) Despite the fact that the number of children receiving ART is increasing globally, improving their survival, children are more likely to develop ARV resistance jeopardising the success of current and future treatment options. In HIV-infected patients, widespread resistance mutations to the three main ARV families (TC-DRM) frequently result in phenotypic or genotypic resistance to at least one drug from all three families (Barbera AJ et al., 1990).

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