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GENOTYPIC RESISTANCE IN PATIENTS WITH PERSISTENTLY DETECTABLE LOW-LEVEL VIRAEMIA TREATED WITH TRIPLE NUCLEOSIDE ANTIRETROVIRAL THERAPY

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OBJECTIVE: A significant proportion of patients failing triple nucleoside analogue combinations appears to maintain long-term low plasma viral load (VL) (<104cp/ml) in spite of HIV resistance. The virological determinants of this particular evolution profile are not known.

PATIENTS AND METHODS: We retrospectively analysed HIV reverse transcriptase (RT) genotypic profile in 27 patients receiving triple nucleoside therapy for more than 6 months with stable VL<104cp/ml (LVL group). These genotypes were compared to those in eight patients with VL>104cp/ml (HVL group). LVL patients had a median gain of 214±247 CD4/mm³ relative to pretherapy levels and maintained a median VL of 2370 cp/ml (52–9500) for a median 32 months; at the beginning of treatment they had a median VL of 105 cp/ml and 238 CD4 cells/mm³. HVL patients, received only 10 months of triple nucleoside therapy.

RESULTS: Among LVL patients, all had at least one resistance mutation in RT, including mutations of codon 215 in 21. Interestingly, while 18 patients had a T215F mutation, only three had a T215Y mutation, which was always associated with L210W; in two of them T215Y was preceded by a T215F and appeared as viral load increased significantly. In 10/18, the T215F mutation was also associated with K219E and in 16 of 18 with a combination of D67N and K70R. In HVL patients, all developed at least one RT mutation. Remarkably, seven of eight patients had a T215Y mutation. Among them, six also displayed a M41L, but only two had a combination of D67N and K70R, while one had a K219E.

CONCLUSIONS: Among patients failing triple nucleoside therapy, the T215F/K219E resistance pathway, as opposed to the T215Y/L210W pathway, appears to be highly prevalent in patients retaining low VL levels. Whether this relates to the replicative capacity or the resistance properties of these viruses needs to be further investigated. That could influence the moment when a treatment must be modified in a failing patient.

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