VIROLOGICAL AND CLINICAL, EPIDEMIOLOGICAL CHARACTERISTICS OF NEW HIV-1 DIAGNOSIS ON COMPLEJO HOSPITALARIO DE NAVARRA, SPAIN

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Background

Molecular epidemiology studies of the human immunodeficiency virus (HIV) are important tools for tracking patterns of transmission and spread of the virus in a given country. The overall rate of resistance to antiretroviral drugs is high, both in patients with recent and chronic infection.

Objectives

To describe prevalence of primary resistance to antiretroviral drugs in patients naïve diagnosed with HIV-1 infection in our hospital during the period 1/1/2014-31/08/2016 as well as their virological and clinical-epidemiological characteristics.

Material/Methods

Retrospective descriptive study in which patients with newly HIV-1 diagnosed cases were included. Review chart (epidemiological, clinical and virological data) was performed. The microbiological diagnosis was performed with four generation screening technique (Abbott Architect HIV Ag-Ab Combo Assay). Positive results where confirmed by Geenius HIV-1/2 assay (Biorad) or INNO-LIA HIV-1/2 score line-immunoassay (Innogenetics). HIV-1 Viral load was made by Cobas AmpliPrep/HIV (TaqMan HIV-1 Roche™).

The resistance and molecular characterization was performed by sequencing at the National Microbiology Center. The genotypic prediction of tropism was performed according to the Geno2pheno program following the recommendations of the European Consensus Group for clinical management of HIV-1 tropism testing. Interpretation of antiretroviral resistance results was performed according to the algorithm of the Stanford University database.

Results

We included 108 patients with HIV-1 diagnosis, of whom 76.85% were men. The mean age was 37 years +/-13 years (min-max: 9-79 years).

✓ The most important acquisition route was sexual 97.1% (66/68), of whom 43.9% were men who have sex with men (MSM).

✓ 41.7% were late diagnosis and 18.5% were very late (<200 CD4/mL).

✓ 2.9% of patients had coinfection with HBV and 7.6% with HCV.

✓ Subtype was performed on 95 strains: 57.9% were B subtype (Figure 1).

✓ The tropism could be determined in 44 strains: 75% were R5 tropic variants, 46.6% were R5X4 tropic variants and 11.4% were tropic X4 variants.

✓ Antiretroviral resistance testing was performed on 93 strains. 100% were susceptible to nucleoside RT inhibitors (NRTIs). In the figure 2 are shown the resistances to different antiretroviral agents. Resistance mutations were detected in 15 samples (Table 1): 11.8% had a primary mutation against non nucleoside RTI (NNRTIs); 3.2% against PR inhibitors (PIs) one of them with primary mutation; 1.1% had secondary mutation against integrase inhibitors (INI). Mutations were detected in 5 strains to two antiretroviral families; (two with primary resistance mutation which conferred resistance to NNRTIs) and one to three families (which conferred resistance to PRIs). In table 1 are shown the detected mutations and its distribution by antiretroviral class.

Conclusions

- ✓ Sexual route continues to be the main form of acquisition; mainly in MSM.
- ✓ An important percentage had a non-B genetic forms which can be explained by the increase in migratory flows and expansion of non-Bs in transmission clusters.
- ✓ Our prevalence of primary HIV resistance was 11.8%, in Spain it ranges from 3%-15%. These are caused by virus transmission from ART patients without complete viral suppression, by the use of suboptimal treatment regimens or by lack of adhesion.

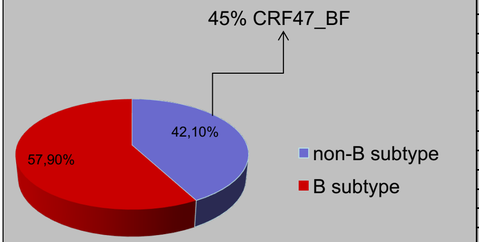

Figure 1. HIV subtype.

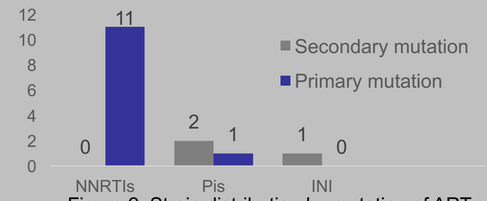

Figure 2. Strain distribution by mutation of ART.

Table 1. Mutations and resistance of ART.

Mutations			Resistance to NNRTIs
Primary	**Secondary**	**N**	**11.82%**
-	V106I	1	-
V106M, V179D	-	1	EFV, NVP
-	V90I	2	-
V106I, G190A	-	1	EFV, ETR, NVP
V106M, V179D	-	1	RVP
V179E, Y188L	-	1	EFV, NVP
V179DV	-	1	EFV, NVP, RPV
E138A	-	2	RPV
E138A	-	1	ETR, RPV
E138AE	-	1	RPV
V1061	-	1	EFV, ETR, NVP
T179D	-	1	RVP
Mutations			**Protease inhibitors**
Primary	**Secondary**	**N**	**3.22%**
K20I		1	NFV
L33I		1	-
-	L10I	4	-
-	L33I	6	-
-	L10I, L33I, A71T	1	-
-	L10V	7	-
-	A71T	3	-
-	A71V	2	-
-	E35Dg, A71T	1	-
-	Q58E	1	TPVr
-	L31I	2	-
-	L10V, K20I	1	-
-	L10I, K201	2	-
-	K20I	3	-
-	K20I, A71V	1	NFV
-	K201, T74S	1	-
Mutations			**Integrase inhibitors**
Primary	**Secondary**	**N**	**1.08%**
-	E157Q		EVG, RAL
-	L71I		-
-	L74I		-

NNRTIs= *Non nucleoside retrotranscripase inhibitors,* ***RTV=****Ritonavir,* ***EFV=****Efavirenz,* ***NVP=****Nevirapine,* ***RPV=****Rilpivirin,* ***ETR=****Etravirine,* ***NFV=****Nelfinavi,* ***TPR/r=****tipranavir/ritonavir,* ***EVG=****Elvitegravir,* ***RAL=****Raltegravir.*

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