

Background

Co-infection with Human Pegivirus (HPgV, formerly referred to as GB Virus type C and Hepatitis G Virus) contributes to slower HIV disease progression by:

- inhibiting HIV replication,
 - lowering immune activation;
 - decreasing the expression of HIV co-receptors.
- However, the effect of HPgV on antiretroviral treatment (ART) outcome is unknown.

Objective

To investigate the possible effects of HPgV co-infection on ART outcome.

Hypothesis: HPgV co-infection might have a beneficial effect on HIV treatment success.

Materials and Methods

We included HIV+ patients participating in the Estonian HIV cohort study (E-HIV) who had:

- initiated ART between 2000 and 2014;
- a plasma sample in E-HIV biobank before the initiation of ART;
- received ART for at least 12 months;
- at least two HIV viral load (VL) measurements after ART initiation.

N = 4899

Patients included in the E-HIV database as of 31st of July 2016.

N = 242

Patients who met the inclusion criteria.

Virologic suppression (VS) – two consecutive on treatment VL-s were below 400 copies/ml.

Methods: nested PCR, gel electrophoresis.

Statistics: Fisher's exact test, logistic regression analysis, Cox proportional hazard models.

Results

Majority of the study subjects were young males with low CD4 count and high HIV VL (Table 1).

Table 1. Characteristics of the Study Population

Gender, n (%)	
Male	145 (60%)
Female	97 (40%)
Transmission route, n (%)	
Intravenous drug use (IDU)	73 (30.2%)
Non-IDU	134 (55.4%)
Unknown	35 (14.4%)
HCV serostatus, n (%)	
HCV+	121 (50%)
HCV-	99 (40.9%)
Unknown	22 (9.1%)
HPgV viremia, n (%)	
HPgV+	56 (23.1%)
HPgV-	186 (76.9%)
At the initiation of ART, median (IQR)	
Age, years	31 (27-37)
CD4 count, cells/ml	265 (168-341)
VL, log ₁₀ copies/ml	4.7 (4.2-5.3)

The prevalence of HPgV:

- 23% (95% CI 18.1%-29.1%) among all patients;
- similar distribution in terms of HIV transmission route and HCV seropositivity (Table 2).

Table 2. Univariate Logistic Regression Analysis of the Distribution of HPgV in terms of HIV Transmission Route and HCV seropositivity

TR	HPgV+	OR (95% CI)
IDU*	20 (27.4%)	1.0
Non-IDU	26 (19.4%)	0.64 (0.31-1.32)
HCV co-inf.		
HCV-*	20 (20.4%)	1.0
HCV+	32 (26.4%)	1.4 (0.71-2.81)

Note. TR – HIV transmission route; *reference

The achievement of VS:

- 82% of patients achieved VS with a median sensory time of 12 (IQR 4-24.75) weeks;
- No association between the presence of HPgV and the achievement of VS (p>0.05).

Conclusions

HPgV is common among Estonian HIV+ patients and the prevalence does not seem to be affected by HIV transmission route or HCV seropositivity.

Our results do not support our hypothesis that the presence of HPgV is beneficial in achieving VS in HIV positive patients.

- The presence of HPgV infection does not improve the suppression of HIV VL (HR 0.91 95%CI 0.65-1.27) (Figure 1).

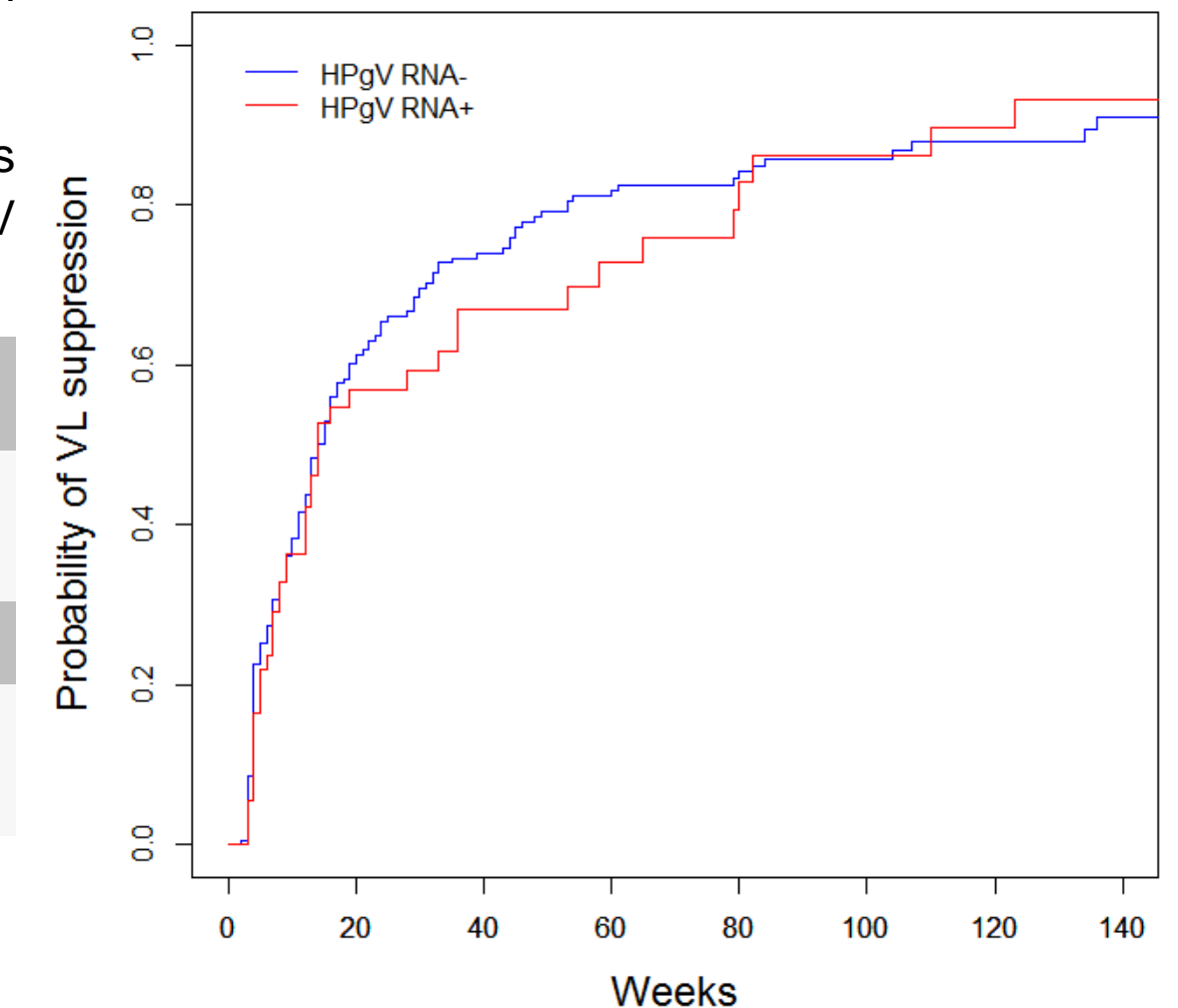


Figure 1. Kaplan–Meier curves of virologic suppression by HPgV RNA positivity. Note. The time is presented in weeks from the initiation of ART.