



## Human Pegivirus (GB Virus Type C) and Its Relationship with HIV

### İnsan Pegivirus (GB Virüs Tip C) ve HIV ile Olan İlişkisi

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#### Dear Editor;

Human pegivirus (HPgV) is a RNA virus which is classified in *Flaviviridae* family. The virus is named as hepatitis G virus (HGV) or GB virus type C (GBV-C) (1).

HPgV was initially considered as hepatotropic depending on its definition in humans with non-A, non-B, and non-C hepatitis. Then, no association was found between acute or chronic hepatitis and HPgV. Therefore, virus (HGV or GBV-D) together with primate and bat viruses (GBV-A, GBV-C, GBV-D) were included in a new genus (Pegivirus). It was again named as HPgV. HPgV infection can occur via exposure to infected blood, sexual contact, and mother-to-child transmission (1).

HPgV can cause infections either alone or in combination with other factors such as hepatitis C virus (HCV) and HIV. The effects of this virus on chronic infections with hepatitis B virus or HCV have not yet been clarified (2). Furthermore, it was shown that the prevalence of HPgV increased in individuals infected with HCV and the HCV RNA levels were permanently high in livers of individuals infected by the combination of HCV/HPgV (1). It was specified that HPgV infection can have effects on chronic infection development or drug resistance (2). Viral hepatitis is commonly observed in chronic dialysis patients. The risk of HPgV infection increases in hemodialysis patients. However, this risk is lower in continuous ambulatory peritoneal dialysis patients (3).

Besides HPgV is associated with beneficial effects in HIV infection. Various studies and a meta-analysis found that survival in HIV-infected individuals was longer in patients with HPgV viremia

compared to those without viremia (4). It was concluded that GBV-C viremia was associated with lower mortality in HIV-infected patients. As a result of T cell activation which happens due to HIV infection, immune functions deteriorate and AIDS progresses after the loss of CD4 (+) T cells. In contrast, compared to chronic HIV-infected patients without HPgV infection, HPgV infection in patients with acute HIV infection is associated with significant decrease in the expression of T cell activation markers. This is independent from treatment (1). In comparison to inactive cells, HIV replication decreases in active peripheral blood mononuclear cells and this finding supports the relationship between HPgV and T cells proliferation (5).

Consequently, when its clinical outcomes and co-infections are considered, even though HPgV is a non-cytopathic factor, there should be further studies investigating the relationship between HIV-1 subtypes and HPgV infection as well as the possible molecular and cellular mechanisms behinds its effects.

#### Ethics

Peer-review: External and Internal peer-reviewed.

#### Authorship Contributions

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