

Final Abstract Number: 40.069

Session: Virology and Viral Infections (Non-HIV)

Date: Thursday, June 14, 2012

Time: 12:45-14:15

Room: Poster & Exhibition Area

Plasmatic glycoconjugates level in patients with porfiria cutanea tarda

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Background: A pathogenic link between HCV infection and PCT is sustained by high frequency of antibodies against hepatitis C virus in patients with PCT.

This study was designed to evaluate if molecular and structural alterations of glycoconjugates could be produced by infection with HCV or could be the results of physiopathological disorders in PCT.

Methods: Seric assessment of TSA (total sialic acid), PASA (protein associated sialic acid), LASA (lipid associated sialic acid) and FSA (free sialic acid) were made for 88 patients with PCT (men aged between 35 and 86 years) divided in 3 groups:

- Group A included 42 patients with PCT and chronic alcoholism;
- Group B included 37 patients with PCT and chronic infection with hepatitis C virus;
- Group C included 9 patients with PCT without other risk factors.

The results in the three groups were compared with those in control group (Group D- that included 60 healthy adult men). Diagnose of PCT was based on clinical data, imagistic study and biological evaluation (high urinary coproporphirines, increased urinary uroporphirines, increased serum iron and changes in liver function).

Results: We obtained in patients with PCT and chronic alcoholism (group A): TSA=102,6±16,4mg/dl, PASA =69,4±12,3mg/dl, LASA=28,8±6,1mg/dl and FSA=3,9±3,7.mg/dl. In patients with PCT and HCV (group B), we obtained the following values: TSA=96,7,±18,5mg/dl, PASA =73,1±14,2mg/dl, LASA=23,4±3,6mg/dl and FSA=5,06±0,9mg/dl. In patients with PCT without other risk factors (group C) we found: TSA=81,6±12,4mg/dl, PASA =66,2±4,7mg/dl, LASA=21,9±4,5mg/dl and FSA=2,33±0,55mg/dl.

Glycoconjugates levels in group D were: TSA=59,8±10,2mg/dl, PASA =57,1±9,3mg/dl, LASA=17,3±2,1mg/dl and FSA=0,95±0,22mg/dl

In group B we observed a positive statistical significant correlation between TSA and FSA ($r=0.428$, $CI=0,286-0,612$, $p<0,05$), relation that was not found in the other groups.

Conclusion: Hepatitis C virus initiates or modifies sialic acid biodistribution in different compartments of the body in patients with PCT. HCV amplifies cutaneous manifestations of PCT, fact that could be explained by the metabolic deficit in uroporphyrinogen decarboxylase during the viral infection.

<http://dx.doi.org/10.1016/j.ijid.2012.05.231>

Final Abstract Number: 40.070

Session: Virology and Viral Infections (Non-HIV)

Date: Thursday, June 14, 2012

Time: 12:45-14:15

Room: Poster & Exhibition Area

Occurrence of active and latent forms of human herpesviruses 6 and 7 infection in patients with central nervous system demyelinating diseases

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Background: The problem of the demyelinating processes is actual. In recent years believe that a possible etiopathogenetic agent is herpes viruses. The leading role belongs for the human herpesviruses 6 and 7 types (HHV-6, HHV-7), which can be not only trigger a pathological process, but also cause progression of the disease. The HHV-6 and HHV-7 viruses after primary infection remained in the form latent/persistent infection for life, under effect of different factors they can be activated. The aim: investigation the frequency detection of active and latent/persistent forms of HHV-6, HHV-7 in patients with demyelinating diseases of central nervous system.

Methods: In this retrospective study were enrolled

- 23 patients (13 females, 10 males; mean age 31±6)
- 12 patients-donors (6 females, 6 males; mean age 30±6) underwent as control group. There were inspected 8 patients with multiple sclerosis; 9 – the acute encephalitis; 6 – the meningoencephalitis.

Blood took away from all patients and liquor took away from patients with demyelinating diseases. Nested PCR used for determining the nucleotide sequence in DNA. HHV-6, HHV-7 DNA isolated from lymphocytes and blood serum.

Results: The presence of sequences HHV-6 in blood cells as a marker of latent/persistent infection was revealed in 4 of 8 of patients with multiple sclerosis, in 3/9 with acute encephalitis, 2/6 with meningoencephalitis and in 3 of 12 donors. It was also revealed DNA HHV-6 in serum as a marker of active infection: in 4/8 of patients with multiple sclerosis and in 3/9 with the acute encephalitis. The presence of sequences HHV-7 DNA in blood cells revealed in 33% multiple sclerosis patients and 25% acute encephalitis. It was recorded HHV-7 viremia in 14% patients with multiple sclerosis and 10% patients with the meningoencephalitis.

Conclusion: It was revealed the occurrence of HHV-6 and HHV-7 in patients with demyelinating diseases. Both forms of infection, active and latent/persistent, were registered in all patients.

<http://dx.doi.org/10.1016/j.ijid.2012.05.232>