Dementia Caused by Hepatitis C Virus—Case Report

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Abstract: HCV (hepatitis C virus) infection produces a chronic systemic disease that induces chronic hepatitis, cirrhosis and hepatocellular carcinoma, and also can induce dementia. Dementia can be defined as a syndrome of global and progressive impairment of acquired cognitive abilities. Importance of early detection of HCV infection in prevention of cognition decline in infected patients is emphasized along with the fast progressive form of dementia caused by HCV through possible pathogenetic mechanisms presented in this paper.

Key words: HCV infection, dementia, progressive form of dementia.

1. Introduction

Dementia can be defined as a syndrome of global and progressive impairment of acquired cognitive abilities, caused by organic disease of the central nervous system, with preserved consciousness, in which particularly damaged the ability of memory, learning, abstract thinking, orientation and understanding of visual-spatial relationships. Epidemiological data on the incidence of dementia are frightening and, therefore, are numerous studies about etiology, pathophysiology and treatment options conducted [1, 2].

The most common forms of dementia are Alzheimer’s type, frontotemporal dementia, vascular dementia, “Lewy body” dementia, Parkinson disease with dementia, progressive supranuclear palsy, dementia caused by hydrocephalus, tumor. All types of dementia have mostly slow progressive course and affect the elderly population with a higher incidence of fungal diseases above 65 years of age. Dementia syndrome can also be caused by metabolic causes, which include various toxic conditions (systemic infection, alcoholism, drug overdose, poisoning by heavy metals and organic solvents), Vitamine B12, B1, B2 deficiency, chronic hepatic, renal and cardio-respiratory encephalopathy, endocrine disorders (diabetic ketoacidosis, chronic hypoglycemia, hypothyroidism, hypo- and hyperparathyroidism, adrenal insufficiency and Cushing’s syndrome), diseases of fungal accumulation (metachromatic leukodystrophy, Kufs disease, Hallervorden-Spatz disease), and disorders of the electrolyte (hyper- and hyponatremia, hyper- and hypocalcemia). All mentioned types of dementia are reversible if recognized and treated on time [3-5].

Infections can also be cause of dementia syndrome. The authors would like to show a patient with hepatitis C and fast progressive type of dementia.

2. Clinical Case

E.R. male patient, born in 1967, admitted to hospital due to heteroanamnesis of recent cognitive decline, addiction to heroin from 1982 to 2003, with the methadone therapy in continuation until 2008 without recidive. Hepatitis C-type HCV (hepatitis C virus) 3a diagnosed in 2008, number of virus copies 100739 HCV/RNA (ribose nucleic acid) in 1 mL of serum, the degree of liver fibrosis 5/6, treated with pegylated interferon plus ribavirin for one year when the treatment ceased. On admission to hospital, patient was conscious, slow in speech, disoriented in time and
space, MMSE 17/30, neurological status without significant neurological differences. MSCT brain indicated the heterogeneous hipodense zone of white brain matter without sharp limits to the surrounding tissue. Described finding was designated by open etiology. MRI finding included diffuse large number of periventricular confluent lesions of high T2 signal, FLAIR and T1 sequences (Fig. 1). We have no earlier MRI record to compare.

Liver function tests lightly elevated above the normal range, ammonium level is normal and ultrasound of the liver is in order. HIV (human immunodeficiency virus) antibody is negative. CSF (cerebrospinal fluid) analysis detected oligoclonal bands and in the serum as well (type 3), proteins in the cerebrospinal fluid slightly increased to 0.53, present 73/3 leukocytes. Microbiological analysis of cerebrospinal fluid was negative, PCR (polymerase chain
reaction) for JC (John Cunningham) virus in the cerebrospinal fluid was also negative. The course of diseases was malignant, with extremely rapid progression of cognitive deterioration to complete disorientation, failure to execute simple tasks with the gradual disturbance of consciousness within 14 days, development of tetraplegia, aphasia and aphagia till lethal outcome 16 days after admission to hospital. Patient was treated with high doses of corticosteroids and ceftriaxone with symptomatic precaution special measures.

3. Discussion

HCV (hepatitis C virus) infection produces a chronic systemic disease that induces chronic hepatitis, cirrhosis and hepatocellular carcinoma.

HCV is known to cause central nervous system dysfunction like fatigue and depression regardless of the liver diseases severity [6, 7]. Also, HCV may be associated with impairment in attention/working memory, executive functioning, verbal learning, memory, processing speed. This symptom cannot be explained by hepatic encephalopathy or drug abuse. Respectively, these symptoms are often relieved after antiviral therapy [8-11].

Possible hypothetical mechanism suggest that HCV infect PBMCs (peripheral blood mononuclear cells), especially macrophages. Infected macrophages cross the blood brain barrier (a phenomenon of Trojan horse) with a gradual expansion in permissive cells of the brain. The primary target cells in the brain are microglial cells. Affected macrophages and microglial cells may cause the release of proinflammatory cytokines like TNF-alpha, IL-1, IL-6, and neurotoxins, such as NO (nitric oxide) and viral proteins what leads do neurocognitive dysfunction. Similar changes are described in HIV infection with AIDS (acquired immune deficiency syndrome) type of dementia. Until now it was considered that in HCV replication in macrophages is present at lower level and does not lead to such a degree of dementia [12-19].

Recently another was published dealing with the relationship between HCV-infected patients and an increased risk for dementia. The study included Taiwanese patients only; the results might not be generalizable to other populations. Taiwan has one of the highest HCV prevalence rates in Northeast Asia. In this study, HCV might increase the risk of dementia 36% [20] compared with non-HCV subjects and the attributable proportion of dementia in relation to HCV was 17.4% [20].

Worldwide, the prevalence and number of people with anti-HCV has increased from 2.3% to 2.8% between 1990 and 2005 [21].

4. Conclusions

HCV infection is relatively common health problem. WHO estimates that about 170 million people worldwide are infected with Hepatitis C (WHO, 2000). HCV causes both acute and chronic infection. Acute HCV infection is usually asymptomatic, and is only very rarely associated with life-threatening disease. About 15–45% of infected persons spontaneously clear the virus within six months of infection without any treatment. The remaining 55–85% of persons will develop chronic HCV infection. It is an important public health issue that HCV infection may increase the risk for dementia. It is very important to bear in mind that there is an increased possibility of developing dementia syndrome in patients with chronic hepatitis and therefore attention should be payed on to early onset of neurological signs and symptoms indicating the development of dementia. If such signs and symptoms are noticed it is necessary to intensify treatment for HCV infection in order to prevent neurological complications in form of dementia syndrome. In conclusion, emphasis should be placed on early detection of cognition decline in HCV-infected patients to prevent dementias.

References

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