Hepatitis A virus infection in guillain-barre syndrome

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Abstrak

Background

Prodromal factors of Guillain-Barre syndrome (GBS) are often associated with previous viral infection (60%). The ailment supported by the acquired immunomediated disorder concept. Viral hepatitis is very rarely found in GBS, preceded by cytomegalovirus (15-18%), Campylobacter jejuni (28%), and Epstein-Barr virus (5%). There is no specific etiology of GBS because those viruses usually appear sporadically (subclinically). All hepatitis virus infection can cause neurological complications, including GBS.

Case Report

We report two cases of hepatitis A virus infection (HAV) in GBS patients in Dr. Sardjito General Hospital during 5 years of observation (1996-2000) from 92 GBS patients. The diagnosis of HAV was based on more than 2 times increment of transaminase enzyme, positive IgM anti HAV, negative HbsAg, and negative IgM ami HCV. The diagnosis of GBS was based on clinical symptoms of acute generalized paralysis, cerebrospinal fluid examination, and electromyelography. In both cases, sub-clinical and sporadic symptoms appeared several days before paralysis, which makes it more likely that the prodromal period of GBS occurred at the same time of HAV incubation period.

Discussion

The incidence of HAV in GBS patients during 5 years of observation was 2%. This corresponds with the case reported by Verona et al, 1996 and Pelletier et al, 1985, i.e. the presence of peripheral neuropathy (n. facialis and n. occulomotorius). Possible alternative pathways for hepatitis virus complicating as GBS are perivascular and endometrial peripheral nerve infiltration by mononuclear cells, T cell sensitization, stimulation of IL-2 growth factor surface receptor, and B cell stimulation. All of the conditions mentioned above causes necrotizing arteritis, vascular occlusion, and at the end, segmental demyelinization. Hepatitis virus may replicate in the central nervous system or peripheral nervous system, subsequently developing into multiple neuropathy disorder and poly arteritis.

Conclusion

The diagnoses of HAV and GBS in both cases were established. HAV is one of several viruses that may trigger GBS. In both cases, HAV infection was sub-clinical and sporadic. Symptoms of hepatitis infection subsided along with improvements in the patient's neurological status. Acute viral hepatitis has a wide clinical spectrum and laboratory manifestation that is in accordance with the severity, varying from unclear symptom (anicteric) to jaundice. Acute hepatitis A, B, C infections have the same symptoms in general. However, hepatitis B and C tend to be more severe. The mildest symptoms are transaminase enzyme level increment, no jaundice, gastrointestinal symptoms, flu-like symptoms, and sometimes it can not be diagnosed. The more severe symptoms are jaundice with obvious generalized symptoms.' The incidence of

hepatitis A is difficult to be determined accurately because of its characters, i.e. sporadic, endemic, and has a high rate of asymptomatic infection.23-4