Gastroenterología y Hepatología

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LETTER TO THE EDITOR

Hepatitis C therapy and anticonvulsants

Tratamiento de la hepatitis C en pacientes con anticonvulsivos

Dear Editor,

Treatment of chronic hepatitis C in patients who are undergoing anticonvulsant treatment may be challenging. Drugs that are potent P-glycoprotein (P-gp) and cytochrome P450 (CYP) inducers (e.g. carbamazepine, phenobarbital, phenytoin) are contraindicated with direct acting antivirals (DAA) because of the decrease in exposure, with a potential loss in efficacy.[1] Temporal interruption of anticonvulsant therapy, or even changes in the treatment regimen are usually very difficult, if not impossible.

We present the case of a 55 year old female with a diagnosis of epilepsy (secondary to neonatal hypoxic-ischemic encephalopathy) who was undergoing treatment with clonazepam (0.5 mg/d), carbamazepine (400 mg/12 h) and phenobarbital (50 mg/d). The patient was diagnosed with chronic hepatitis C more than 20 years ago; she had never received treatment. Over the last 5 years, AST and ALT values increased up to 100 IU/L and transient elastography reached 13.6 kPa in December 2019. An abdominal ultrasound examination showed a normal liver but a portal vein measuring 13 mm and spleen size of 13 cm (both suggesting the presence of portal hypertension). After consultation, her neurologist did not consider acceptable to modify/interrupt the current anticonvulsant therapy.

We discussed the pros and cons of antiviral treatment with the patient and finally decided to initiate sofosbuvir/velpatasvir for12 weeks. Since we were not able to measure DAA concentrations during treatment, we tested for HCV-RNA every 2–4 weeks. Baseline viral load was 3.662.000 IU/mL. At week 2 of therapy HCV-RNA had decreased to 42 IU/mL. At week 4 of treatment aminotransferases returned to normal values and HCV-RNA was already undetectable. At weeks 8 and 12 (end of therapy) HCV-RNA remained undetectable. Twelve and 24 weeks after treatment discontinuation HCV load remained below the quantification (SVR12 and SVR24). Transient elastography value decreased to 10 kPa at SVR12.

Despite the fact that current clinical guidelines contraindicate the use of DAA in patients who are undergoing treatment with some anticonvulsants,[1] we believe that in selected cases (were liver disease may compromise life expectancy) HCV therapy should be considered. Although a decrease of DAA levels during treatment with P-gp or CYP inducers is likely and can be predicted,[2,3] the levels at which antiviral efficacy is compromised (in vivo) are no well known. For this reason, frequent determination of viral load during treatment may be helpful to reassure that HCV replication is effectively inhibited during therapy.

Funding

No funding.

Conflicts of interest

XF acted as advisor for Gilead and Abbvie.

References

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https://doi.org/10.1016/j.gastrohep.2020.11.003