similar rigidity but a different biology (chronic pancreatitis vs pancreatic cancer). At present it can be useful in cases with negative FNAB or as a guide for directing FNAB into zones that have a greater probability of malignancy (hard) and avoid necrotic tissue (soft). It cannot yet replace EUS-guided FNAB. Further studies are required that evaluate and determine the specific role of EUS elastography in the evaluation of solid pancreatic lesions.

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Conflict of interest

The authors declare that there is no conflict of interest.

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References


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Successful eradication of genotype 4 HCV with telaprevir-based triple antiviral therapy

Erradicación exitosa del virus de la hepatitis C, genotipo 4, con terapia triple antiviral estándar más telaprevir

A 48-year-old Caucasian woman with chronic hepatitis C virus (HCV) infection, genotype 4, presented to our clinic in the summer of 2011 to be evaluated for antiviral treatment. She had evidence of portal hypertension (splenomegaly, low platelets, small esophageal varices), cirrhosis, stigmata, and a liver biopsy from 2006 had shown bridging fibrosis. IL28B genotypes were subsequently found to be TT at both rs12979860 and rs8099917 loci. The patient had been treated in 2006 with peginterferon (peg-IFN) alpha-2a with 180 mcg SQ weekly and ribavirin (RBV) 1,200 mg/day for 48 weeks. Treatment was unsuccessful with a complete early virological response (EVR) followed by detectable HCV RNA at end-of-treatment visit, and she was then referred to our center for further evaluation. In 2008, she was treated with identical doses of peg-IFN/RBV, achieving a complete EVR (fig. 1). Due to dose-reductions in RBV resulting from

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Remarkably, currently non-HCV RNA levels were detectable in all patients treated with peg-IFN/RBV. However, in patients treated with boceprevir (BOC) in the first randomized trial, none of the patients treated with BOC (7) had detectable HCV RNA levels at the end of the treatment period (12 weeks). Furthermore, in patients treated with telaprevir (TPV) in the first randomized trial, none of the patients treated with TPV (8) had detectable HCV RNA levels at the end of the treatment period (12 weeks). These results suggest that TPV and BOC are effective inhibitors of NS3/4A isolated from genotype 4. We report a case of successful treatment using a TPV-containing regimen in a patient with genotype 4 infection, who had previously failed treatment with peg-IFN/RBV. A phase II randomized clinical trial showed effectiveness of 2 weeks of TPV monotherapy in reducing HCV RNA in treatment-naive patients with genotype 4. This effect was much greater when TPV was combined with peg-IFN/RBV, and the effect of triple therapy was greater than the effect of peg-IFN/RBV dual therapy (HCV RNA reduction of -0.77, -4.32, and -1.58 log_{10} IU/mL, for TPV, TPV+peg-IFN/RBV, and peg-IFN/RBV, respectively). The number of patients included in this study was, however, too small (n=8 per group) to draw any conclusions on triple therapy antiviral efficacy. Interestingly, there is no published experience on treatment with TPV for longer than two weeks in patients with genotype 4. To the best of our knowledge this is the first reported case of a treatment-experienced patient with genotype 4 infection to achieve SVR after a full-length TPV-containing regimen (12 weeks). We believe this isolated clinical experience with a difficult-to-treat patient contributes to the limited knowledge on the effectiveness of a TPV-based antiviral regimen on genotype 4. Without a doubt, new DAA (such as the new NS5B polymerase inhibitor) will open the door for new antiviral regimens with a specific activity against genotype 4. In the meantime, defining the role of the already approved DAA (particularly TPV) for the treatment of HCV genotype 4, based on well-designed studies, should be encouraged.

**Conflict of interest**

The authors declare that there is no conflict of interest.

**References**


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