similar rigidity but a different biology (chronic pancreatitis vs pancreatic cancer).⁴,⁷ 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, Europeans), of rates has 4. The breakthrough, = 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. Remarkably, 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.

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References
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