Reviewer's report

Title: Anticoagulant therapy for nodular regenerative hyperplasia in a HIV-infected patient

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Reviewer: Vincent Mallet

Reviewer's report:

The case of a Human Immunodeficiency Virus (HIV)-infected patient with nodular regenerative hyperplasia (NRH) and severe portal hypertension that improved under anticoagulants is presented. The patient had been previously exposed to didanosine and harbored protein S deficiency. This case adds further evidence that HIV-associated NRH is linked to thrombophilia, but still, there is not enough data to recommend the use of anticoagulants in HIV-associated NRH. The manuscript should be modified to focus on the putative mechanism(s) of HIV-associated NRH. With their observation, the authors should highlight the relation between thrombophilia and HIV-associated NRH which is, to date, not so clear.

1. Major revisions

This patient had several episodes of gastrointestinal hemorrhage, one of them under vitamin K antagonist (VKA). Please explain and discuss how you managed these complications before and under anticoagulants.

The following mechanisms: HIV-1 vasculotropism and toxicity, didanosine and thrombophilia (especially Protein S deficiency) should be better discussed. Please reference the recent studies on the subject.

The conclusion of this paper is that anticoagulants should be evaluated in controlled studies.

2. Minor revisions

Please reference the recent papers on the subject [1-5].

In the introduction, first paragraph, it is inaccurate to state that the central veins are squeezed by the enlarged hepatocytes in NRH, especially in HIV-associated NRH where obliterative venopathy seems to be the main mechanism [3, 6].


Level of interest: An article whose findings are important to those with closely related research interests

Quality of written English: Acceptable

Statistical review: No, the manuscript does not need to be seen by a statistician.

Declaration of competing interests:

Honoraria received from Schering-Plough, Bristol-Myers Squibb, Gilead Science.