We would like to thank the reviewers and editors for careful reading of our manuscript and their comments. We would like to answer as follows:

Reviewer #1:

Specific Comments to Authors: This case report draws attention to the potential role of trientine in the triggering of colitis in a patient with Wilson disease (WD). Although the case is interesting because it underlines the importance of the appropriate and early diagnosis of WD, there is no original information. The close chronological link between administration of trientine and the onset of colitis and the resolution of colitis with the suspension of trientine and following steroid treatment, although they strongly support the triggering role of trientine, does not allow to exclude other possible causes of colitis in a cirrhotic patient.

We think that the diagnosis of hemorrhagic colitis as an adverse drug reaction is well supported by negative stool cultures and exclusion of CMV. Although cirrhotic patients can develop portal hypertensive colopathy, this usually does not become hemorrhagic.

Furthermore, even if we accept the causal role of trientine in the triggering of colitis, the case report does not add much to what has already been reported.

There are only 4 cases of colitis during trientine treatment described in the literature:

Dahlman T, et al. QJM. 1995 Sep;88(9):609-16.
Boga S, et al. BMC Pharmacol Toxicol. 2015 Nov 20;16:30.
Mayr T, et al. J Pediatr Gastroenterol Nutr. 2021 Jan 1;72(1):115-122

We therefore believe that publication of additional cases of this probably uncommon side-effect of pharmacotherapy of a rare disease is of scientific interest.

Reviewer #2:

Specific Comments to Authors: The authors reported a 51-year-old woman who was diagnosed with liver cirrhosis due to decompensation with ascites. Etiologic evaluation raised suspicion of hereditary hemochromatosis because of compound heterozygosity HFE p.C282Y/p.H63D and phlebotomy was started. Re-evaluation showed low ceruloplasmin, increased urinary copper excretion and the presence of Kayser-Fleischer rings. Wilson disease was confirmed by genetic analysis. The main concerns is the diagnosis. The patient was diagnosed as hemochromatosis based on the compound heterozygosity HFE p.C282Y/p.H63D. The author did not provide any informaiton of the HFE p.C282Y/p.H63D variants. The readers do not know the variants were in the same allele or in the different allele and the readers do not know the pathogenesis of the two variants. Furthermore, readers do not know whom the variants came from respectively. It is the same thing that the genetic variants of the ATP7B.

We do not provide more details of genetic evaluation of hereditary hemochromatosis as it is not the main focus of this case report. The genetic diagnosis of Wilson disease on the other hand is reported in detail with two known pathogenic variants in ATP7B.

And in the Results part, it was stated the presence of two heterozygous pathogenic variants, namely c.3207C>A, p.(His1069Gln) and c.2305A>G, p.(Met769Val). However in the Discussion part, it was stated that the patient was homozygous for the variant c.3207C>A (His1069Gln).

We recognize that the discussion part may be have been confusing as it additionally describes the genetic analysis in another patient that has similarities with our present case. As this is of minor interest, we shortened and clarified this part in the discussion section.

Please show the pedigree and the Sanger sequencing results.

We would choose not to add a simple pedigree as it would only contain information on the index case and her parents. This would not provide additional information beyond the manuscript text

There are many typos and grammar errors, and asking a English native speaker to revise th manuscript is necessary.

We did additional language revision.

Science Editor:

I have read this case report submitted to the World Journal of Hepatology with great interest. The expert reviewers have raised several issues. First, the question regarding the diagnosis of Wilson disease and further clarification on the definition for the patient´s variant c.3207C>A (His1069Gln) is necessary.

We are glad to have attracted the editor's interest. The variant c.3207C>A (His1069Gln) is described as pathogenic variant in several publications. We added the following reference as an example:

Pop TL, et al. World J Hepatol. 2021 Oct 27;13(10):1428-1438.

Reviewers were also concerned about the novelty of the study and the causal relationship between trientine and the colitis. Authors must address these points.

We addressed this point in answers to reviewer #1

Company Editor-in-Chief:

The scientific quality of this study does not reach the publication standard of the World Journal of Hepatology. Rejection, with the opportunity of re-submission after revision.

We are grateful for the opportunity of resubmission and hope that we satisfactorily addressed the reviewers and editors concerns.