Dear editor,

The comments of you and the reviewers were highly insightful and enabled us to greatly improve the quality of our manuscript. In the following pages are our point-by-point responses to each of the comments of the reviewers as well as your own comments.

To the editor:

We have prepared the figures using PowerPoint.

To the first Reviewer: 05658003

Thank you for your high appraisal.

To the second Reviewer: 05644467

Question 1: 1. The correlation between GSD and IBD is suggested to be further elaborated.

Answer: Thanks for your suggestion. Studies have shown that the production of endogenous glucose by the G6PT/G6Pase-β complex is the only energy source of mature neutrophils, which is essential for neutrophil function. Among patients with GSD-Ib, however, neutrophils are unable to produce endogenous glucose due to lack of G6PT, ultimately leading to increased neutrophil dysfunction and apoptosis. In addition, the only neutrophils remaining are unable to infiltrate the intestinal mucosa normally in patients with GSD and IBD, thus resulting in the persistent presence of intestinal bacteria. At the same time, continuous infiltration of macrophages leads to the development of granulomas, which in turn leads to chronic inflammation of the intestine, and eventually leads to intestinal fibrosis and fistula formation.

Question 2: Why the neutrophils of GSD patients decreased. What's the mechanism is?

Answer: Thanks for your suggestion. Studies have shown that the production of endogenous glucose by the G6PT/G6Pase-β complex is the only energy source of mature neutrophils, which is essential for the function of neutrophils. But in patients with GSD-IB, neutrophils are unable to produce endogenous glucose due to lack of G6PT, ultimately leading to increased neutrophilic apoptosis.

Question 3: Please provide this patient's diagnostic data of GSD and IBD in detail, such as gene mutation and important laboratory index.

Answer: Thanks for your suggestion. **Diagnostic data of GSD**: the genetic examination revealed that the patient had GSD Ib due to the gene mutation in the SLC37A4 gene. **Diagnostic**

data of GSD: 1.The patient was hospitalized for abdominal pain and diarrhea of 10 years duration. 2. Computed tomography indicated changes in the left transverse colon wall and significant narrowing of the intestinal lumen, leading to proximal colonic obstruction and fecal accumulation. Magnetic resonance imaging showed multiple segmental intestinal lesions in the jejunum, ileum, and colon, with obvious intestinal strictures in the colon. Colonoscopy revealed hyperemia and edema in the colonic mucosa, with multiple ulcers and intestinal strictures. The pathologic evaluation of a biopsy indicated acute and chronic colonic mucosal inflammation and granulation tissue formation. 3. The postoperative pathologic evaluation revealed chronic ulcer and intestinal abscess formation in the colon with diffuse mesenteritis, which was consistent with IBD.

We hope that the revisions in the manuscript and our accompanying responses will be sufficient to make our manuscript suitable for publication in World Journal of Clinical Cases.

We shall look forward to hearing from you at your earliest convenience.

Yours sincerely,

Jian Wan