Response:

We would like to thank you for your careful reading, helpful comments, and constructive suggestions, which has significantly improved the presentation of our manuscript.

We have carefully considered all comments from the reviewers and revised our manuscript accordingly. In the following section, we summarize our responses to each comment from the reviewers. We believe that our responses have well addressed all concerns from the reviewers. We hope our revised manuscript can be accepted for publication.

Our modifications are as follows:

1.We regret that we did not clearly write the position of the spleen after torsion. The spleen is located in the left lower abdomen after torsion. I have modified and annotated it in lines 1-2 of the imaging examination.

2.Thank you very much for reminding us about the time of taking aspirin. Aspirin continued to take for 6 months and has been added to the line 7 of the treatment section.

3.We are very sorry that the laboratory data are not comprehensive. We have added the platelet, prothrombin time, activated partial thromboplastin time，Fibrin degradation products，D-dimer and liver function data to the laboratory examinations section in lines 4-15. platelet count was 185×10^9/L (normal range: 125×10^9–350×10^9/L); prothrombin time was 14.4s (normal range: 9.4s-12.5s); activated partial thromboplastin time was 30.8s (normal range: 25.4s-38.4s); fibrin degradation products was 12.31μg/ML(normal range: ＜5μg/mL); D-dimer was 2017ng/mL (normal range: ＜250ng/mL) ; albumin was 36.4g/L (normal range: 65g/L -85g/L); alanine aminotransferase was 12U/L (normal range: 7U/L-40U/L); glutamic oxaloacetic transaminase was 38U/L (normal range: 13 U/L -35 U/L); alkaline phosphatase was 79U/L (normal range: 35 U/L -100U/L); glutamyltransferase was 9U/L(normal range: 7 U/L -45 U/L); total bilirubin was 15.1 μmol/L (normal range: 3.4 μmol/L-23.3 μmol/L); and direct

bilirubin was 5.7 μmol/L (normal range: 0 μmol/L-6.8 μmol/L). These laboratory data show that the patient's liver function is basically normal, which is non cirrhotic portal vein thrombosis, and the patient is in a hypercoagulable state. Unfortunately, patients with protein S, protein C and antithrombin III have not been examined accordingly.

4.Thank you for raising the question of portal vein thrombosis and the corresponding image. The main and right PV branches were filled with defects. We have marked it in lines 5-7 of the imaging examinations section and in Figure 2, figure B.

5.We are sorry that we did not describe the detailed process of portal vein thrombectomy. The procedure of portal vein thrombectomy was as follows: the tube wall was cut longitudinally near the main portal vein and the right portal vein branch, with a length of about 1cm. At the same time, in cooperation with the Department of Vascular Intervention, thrombectomy catheter and balloon were placed through the portal vein incision. We have added lines 1-6 in the treatment section.

6.We regret that the description of the pathogenesis and treatment of portal vein thrombosis is not sufficient. We have consulted the literature and added the following contents:

PVT can be divided into acute and chronic manifestations, as well as causes of cirrhosis and non cirrhosis. The patient's liver function in this case is basically normal, with no history of liver cirrhosis. Portal vein thrombosis is formed in the short term, considering acute non cirrhotic causes. The pathophysiology of PVT is related to the disorder of Virchow's triad, where venous stasis, endothelial damage, and increased hypercoagulability make the patient prone to thrombosis. We believe that splenic venous congestion and hypercoagulability in patients after splenic torsion may be the mechanisms underlying portal vein thrombosis.

Thrombolysis, thrombectomy, or transjugular intrahepatic portosystemic shunt are PVT treatment methods. Yet, there is no consensus on

intervention strategy. Practical guidelines support anticoagulation as the first-line treatment for PVT. The treatment of PVT requires multidisciplinary management for different patients. A detailed introduction to the management and prognosis of wandering spleen torsion with PVT has not been reported in the literature. For patients underwent splenectomy for complete splenic infarction and developed portal vein thrombosis, Anticoagulant therapy is necessary, but the need for combined surgery remains to be further discussed. We have added the content to paragraphs 2 and 4 of the DISCUSSION section.