1. Line 120; Please add new figure(s) on the MYD88L265P and CD79B mutation, and fully explain what kind of mutation was specifically. For example, add the new figure on the result of direct sequencing, and so on.
Answer：The result I got was a written report, I could not provide figure of direct sequencing.

2. Discussion; Search for and review similar cases that have been reported to date, and add a new Table on the summary of these cases.
Answer： I Search similar cases, and add a new Table1.

3. Discussion; Please add a new schematic figure (s) on where and how it affected the transformation from WM to DLBCL, or the redundant long description of characters only explanation. The present descriptions is difficult to understand for WJCC readers, I think. Please add new schematic figures on NF-kB signaling pathway in the ABC subtype (line138), CD79B-dependent BCR activation pathway (line152), oncogene signaling pathway (line190) and explain it clearly and concisely.
Answer:I add new schematic figures and signaling pathway, and explain it.
Figure 5. NF-κB activation through the classical pathway is a hallmark of the ABC DLBCL subtype. MYD88 is an adapter protein that couples TIR-containing receptors, regulating downstream signaling circuits, including the NF- κB. MYD88 coordinates the IRAK family kinases into a helical signaling complex through the interaction with the IRAK kinase. Phosphorylation of IRAK1 by IRAK4 will allow for the recruitment of the ubiquitin ligase TRAF6 and the activation of the downstream pathways. The mutation in MYD88L265P forms a stable, phosphorylated form of IRAK1, which upregulates gene expression signatures of NF-κB. The BCR consists of IgL and IgH chains that are noncovalently coupled to the CD79B (Ig-β) and CD79A (Ig-α) subunits, which regulate BCR surface trafficking, internalization, and expression. Upon antigen encounter, the BCR, CD79A and CD79B transmit signals to multiple downstream signaling pathways. Once BTK is recruited to the BCR signaling complex, LYN or SYK can phosphorylate and activate BTK. In turn activate PKCβ, leading to phosphorylation of CARD11 and activation of NF-κB. CBM complex, a signaling hub consisting of CARD11, BCL10, MALT1, and other proteins, which is required for the activation of classical NF-κB pathway in lymphocytes.

4. Line183; I think figure 5. is unnecessary. Please reconsider it. If necessary, please check whether the source can be posted and indicate whether or not it has been partially modified
Answer: I removed the original figure 5.

5. Abstract What does the authors mean with MCD subtype of DLBCL?
Answer: MCD subtype of DLBCL : DLBCL with MYD88L265P and CD79B mutations were defined as the MCD genetic subtype

6. Case Presentation Does this patient had splenomegaly?
Answer: no evidence of splenomegaly

Dear Editor. Thank you very much for your comments. Your help really means a lot to me. Discussion: The manuscript has been almost properly revised accordingly, however, just one minor criticism should be addressed as below.Minor)1. See, for example, Table.1 in a recently published paper, Chun-Yan Weng, et al. World J Clin Cases. May 26, 2022; 10 (15): 4971-4984. Please create a new table for each item such as age, gender, histological type, staging, treatment method, clinical course, outcome, and so on. Instead, delete your Table in revised manuscript. Answer： I uploaded new Table1 to replace the old one. The table was summarized according to the comments. I uploaded a new manuscript, and the blue marks are the parts I changed. All files see attachment.