Comments to the Authors,

This manuscript reported a non-sense heterozygous nonsense mutation in the exon region of *CYLD* gene in a Spanish MFT1 pedigree (2 affected and 4 un-affected). Nonsense mutation in CYLD gene has been reported to be occurred frequently in several heritable disease, such as familial cylindromatosis (FC) and Brooke-Spiegler syndrome (BSS) in numerous populations. The study was performed rigorously and the findings sound very interesting. What’s more, it would be an exciting example to emphasize the identification of the similar genetic variates contribution to similar phenotypes. However, I have several concerns to make the manuscript more solid,

**Major Compulsory Revisions**

1, What’s the logic to make the conclusion of “different mutational events are responsible for the development of the Austrian case and the Spanish and Dutch cases”? The different mutational event can be inferred from different haplotype origin? Or How to define such haplotype (length, SNP number and so on)?

2, How to exclude other genetic contribution which might also occurred in this Spanish MFT1 pedigree. How many MFT1 caused mutations has been reported in the previous studies?

3, The comprehensive exhibition to the normal haplotypes in this region should be provided with hapmap or 1000 genome data so that the genetic background would be more clear to the readers. Is there any recombination hotspot in this region?

**Minor Revisions**

1, Please give more details to the reference in the background section, such as ‘MFT1, familial cylindromatosis (FC; MIM 132700) and Brooke-Spiegler syndrome (BSS,MIM 605041) have been independently mapped to chromosome 16q12-q13 by several groups[1, 2, 3, 4]’. Which sort of method and the sample size, population or model would be help to the readers.