Response to comments:

1.While the authors reported the history and laboratory exams, they should include the information regarding inflammation and immune deficient conditions (like, WBC value, CRP, and HIV antibodies, etc).

We had added the information regarding inflammation and immune deficient conditions: Routine blood test results were normal (white blood cell count in serum of 5.1×109/L, absolute neutrophil count of 3.4×109/L, C-reactive protein of 1 mg/L). Serum cryptococcal antigen, antineutrophil cytoplasmic antibodies (ANCAs), antinuclear antibodies (ANAs), human immunodeficiency virus （HIV）antibodies, and syphilis antibody tests were negative.

2.The authors may want to include the images of subcutaneous lesions for showing the clinical courses.

The images of subcutaneous lesions for showing the clinical courses is in our Figure 1.

3.The information regarding the detecting the mutation is poorly described. They should add the description regarding the methods to detect the mutation.

We had added the information regarding the detecting the mutation: Two mutations in CARD9 were detected by ChIP-seq using high-throughput sequencing (detection region: exon region of approximately 20,000 genes in the human genome; detection strategy: the explicit disease-causing genes included in OMIM database “2018.11” were analysed) in the present case: ①chromosomal location: chr9:139266425; nucleotide change: c.106C>T; and ②chromosomal location: chr9:139262240; nucleotide change: c.1118G>C. 4. Abbreviations should be avoided in the title. The abbreviations have been removed from the title. 5.The authors should supplement the relevant immune deficiency and inflammatory indexes and the previous drug use. We had added the the relevant immune deficiency and inflammatory indexes: Routine blood test results were normal (white blood cell count in serum of 5.1×109/L, absolute neutrophil count of 3.4×109/L, C-reactive protein of 1 mg/L). Serum cryptococcal antigen, antineutrophil cytoplasmic antibodies (ANCAs), antinuclear antibodies (ANAs), human immunodeficiency virus （HIV）antibodies, and syphilis antibody tests were negative. We had added the information regarding the previous drug use: Piperacillin-tazobactam 3.375 g intravenous drip was administered every 8 h for 7 days. 6. The conclusions are not particularly innovative. We had modefied the conclusions: A good prognosis for fungal infection is associated with prompt identification and proper treatment. Given the findings of our case and the results of our literature review, multiple fungal infections in patients with CARD9 mutations are worthy of clinicians’ attention. Further study into the clinical characteristics and pathogenesis of CARD9 deficiency will yield new insight into therapeutic measures for protecting humans from these devastating fungal diseases. 7. In the discussion part, the literature review of

opportunistic infection bacteria is not sufficient. It should be supplemented that CARD9 lacks relevant reports on susceptibility to fungal infection, clinical characteristics, diagnostic methods, and prognosis. We had modefied the literature review of opportunistic infection bacteria: As a member of the CARD protein family, CARD9 plays an important role in the activation of antifungal mechanisms. It is a key adaptor that can mediate Dectin-1-, Dectin-2-, and Mincle-induced activation of transcription factors through formation of the CARD9–B cell lymphoma/leukaemia-10 (BCL10)–mucosa-associated lymphoid tissue lymphoma translocation protein 1 (MALT1) complex in response to fungal infection. These activated transcription factors mediate translation of key cytokines such as nuclear factor κB (NF-κB), which promotes T-helper cell (Th)1/Th17 differentiation, stimulating antifungal mechanisms in innate cells. CARD9 mutation is a rare inborn error of immunity and probably leads to impaired protection against fungal infections. However, detailed and comprehensive reports on CARD9 deficiency susceptibility to fungal infection, clinical characteristics, diagnostic methods, and prognosis are still lacking. Human CARD9 deficiency is reported to be responsible for the spontaneous development of persistent and severe fungal infections (such as infections caused by Candida albicans, Candida dubliniensis, Phialophora verrucosa, Trichophyton violaceum, Candida sp., Trichophyton mentagrophytes, Exophiala sp., Trichophyton rubrum, and Corynespora cassiicola). Conversely, Cladosporium infection has not been reported.