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<table>
<thead>
<tr>
<th>Scientific quality</th>
<th>[ ] Grade A: Excellent</th>
<th>[Y] Grade B: Very good</th>
<th>[ ] Grade C: Good</th>
<th>[ ] Grade D: Fair</th>
<th>[ ] Grade E: Do not publish</th>
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<td>Language quality</td>
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<td>[ ] Grade B: Minor language polishing</td>
<td>[ ] Grade C: A great deal of language polishing</td>
<td>[ ] Grade D: Rejection</td>
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<td>Conclusion</td>
<td>[ ] Accept (High priority)</td>
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<td>[Y] Minor revision</td>
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<td>Conflicts-of-Interest: [ ] Yes</td>
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SPECIFIC COMMENTS TO AUTHORS
This study investigates the possible link between prior appendectomy and the severity of Clostridium difficile infection (CDI). While the topic reported in this study is very interesting, several other factors may also be taken into consideration. As pointed out by the authors, Clostridium difficile infection occurs due to a dysbiosis of the gut (which may be manifested in two forms: reduced microbiota diversity and over-growth of certain strains like Clostridium difficile), and excessive use of antibiotics are the main risk factor of development gut dysbiosis and CDI. Thus, stronger antibiotics recommended in the conclusion of the manuscript should be further investigated. When we talk about microorganism infection, we need to take the human host immunity response into account, and not always thinking that antibiotic treatment being the sole mechanism responsible for the clearance of infections [1]. Thanks to the immune responses like phagocytosis and xenophagy, most of bacterial infections are self-limiting [1-3]. More importantly, our immune system will use these bacteria as source of essential nutrients [4,5] to support its proper functioning and to provide the essential nutrition needed by the whole body. The use of antibiotics actually deprives the patients of an indispensable source of essential nutrients. Nevertheless, as our immune system also contributes to nutrition acquisition by degrading human microbiota, pathogens and damaged body tissue cells, over-nutrition may occur, which may cause lipotoxicity and further tissue damage [6,7], promoting chronic inflammation and fuelling microbial dysbiosis (over-growth of specific microorganisms like Clostridium difficile). As the authors also collect socio-demographic data like weight and height of the patients and EMR information on patients’ comorbidities in this study, it will be more captivating if the authors can investigate the contribution of BMI or metabolic comorbidity to severity of CDI, and test the link between nutrition disorder and CDI. The following references