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Short Communication

Intestinal Changes in People with Primary Immune Thrombocytopenia

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DESCRIPTION

About one third of clinical hemorrhagic disorders are caused by Primary Immune Thrombocytopenia (ITP), a kind of hemorrhagic sickness mediated by autoimmune abnormalities. Although immunological dysregulation, infection, and genetics are known to have a role in ITP pathogenesis, these factors are complex and remain unknown [1]. The pathogenesis of ITP, which is characterised by increased expression of the pro-inflammatory cytokine IL-2 and decreased expression of the anti-inflammatory cytokine IL-4, is generally agreed to be influenced by T Helper 1 (Th1) polarisation and Th1/Th2 imbalance. Also connected to the gut microbiome is ITP. For instance, after Helicobacter Pylori (Hp) was eradicated, the platelet count and cytokine level of ITP patients rebounded. ITP and intestinal flora, a sizable population of microorganisms populating the human digestive tract, have recently been the subject of research. Detected ten phyla in normal individuals and eight in ITP patients, respectively [2]. The phylum of Bacteroidetes was higher in the ITP patients, whereas the phylum of Proteobacteria was decreased. Also, they discovered a connection between gut flora and platelet quantity and activation. Actinobacteria, Fusobacteria, and Verrucomicrobia phyla all showed increases in ITP patients they also noticed functional changes in the gut flora of ITP patients in their investigation and showed that in ITP patients, Blautia, Streptococcus, and Lactobacillus were enriched whereas the Bacteroides phylum decreased [3]. In a similar vein, they discovered altered intestinal flora metabolites in ITP patients. They explained the alteration as the result of altered inflammatory pathways. Several autoimmune illnesses have also been linked to changes in gut flora, and possible causes have been proposed. Researchers reported a decline in the Firmicutes and Actinobacteria phyla and an increase in the Proteobacteria phylum in the study of Crohn's disease [4]. They speculate that intestinal inflammation and metabolites may be responsible for the changes. Systemic lupus erythematosus was also linked to Altered Gut Flora (SLE). Streptococcus and S.anginosus were discovered to have a positive correlation with SLE activity, presumably because of their pro-inflammatory actions, which may have led to an increase in the release of inflammatory factors and systemic immunity. Alterations in intestinal flora, notably Faecalibacterium, Megamonas, which were thought to be linked to immunological responses in the disease, have also been discovered in psoriasis patients. One issue with the majority of the aforementioned studies is that, in order to examine changes in intestinal flora in disease, they only used one sequencing technique (16S rRNA or metagenomics). Only a few research combined the two approaches. While metagenomic sequencing and 16S rRNA are now the two most popular technologies used for gut flora studies, each has drawbacks [5].

CONCLUSIONS

As 16S rRNA sequencing only has accuracy at the genus level, it is challenging to discriminate between species with high levels of resemblance. Metagenomics, in contrast, enables accuracy down to the species level and permits functional research. Yet, because of its expensive cost and the intricate statistical method, its use is restricted. Several sequencing techniques have been shown to produce conflicting results. Hence, combining two procedures will prevent bias brought on by the methods. No work has used both 16S rRNA and metagenomics to examine the alterations in the gut flora in ITP. Hence, research on the connection between intestinal flora and ITP must be done using both 16S rRNA and metagenomics. In this work, both of the techniques used to examine how the gut flora changed at various levels and to pinpoint any functional changes that were brought on by those changes. Randomly choosing control groups has reportedly been shown to provide falsely good results that were actually brought on by disparities between the healthy controls and the healthy patients in terms of things like lifestyle and food habits. By choosing healthy controls from the patients' family members, who are more comparable to one another in those variables, one might reduce this issue.

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