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Editorial

Microbiota Diversity Shifts in Aplastic Anemia: Emerging Therapeutic Opportunities

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Aplastic anemia (AA) is a rare but serious hematologic disorder characterized by bone marrow failure and peripheral pancytopenia. Its reported incidence in India ranges from 2 to 4 cases per million annually, marginally higher than in Western populations, and it disproportionately affects adolescents and young adults due to a range of genetic, infectious, and environmental triggers [1]. Standard treatment protocols include immunosuppressive therapy (IST) using antithymocyte globulin (ATG) and cyclosporine, hematopoietic stem cell transplantation (HSCT) for eligible patients with matched sibling donors, and supportive care such as transfusions and infection prophylaxis. However, disparities in donor registries, access to transplant infrastructure, and affordability of ATG limit uniform care delivery across the country [2,3].

Emerging research into the gut–bone marrow axis suggests a potentially significant role for gut microbiota in AA pathogenesis. Microbial profiling studies in AA patients reveal reduced abundance of beneficial taxa including *Faecalibacterium* and *Ruminococcus*, which are known for their anti-inflammatory properties and contribution to gut integrity [4]. In contrast, elevated levels of potentially pro-inflammatory species such as *Escherichia coli* and *Clostridium citroniae* have been observed, raising the possibility that microbial imbalance may contribute to immune dysregulation [4,5].

A recent Mendelian randomization analysis provides suggestive evidence of a causal link between specific microbial taxa and AA susceptibility. Ten taxa were identified as genetically associated with AA risk, including a protective role for *Parasutterella*, which exhibited a negative association and may contribute to immunologic homeostasis [6]. Conversely, increased genetic correlations were observed with higher-level taxa such as *Bacteroidia*, *Oxalobacteraceae*, *Anaerotruncus*, *Eubacterium hallii group*, *Intestinibacter*, *Bacteroidales*, *Mollicutes RF9*, and members of *Family XIII UCG001*, predominantly within the *Bacteroidetes* phylum, suggesting a role in promoting immune dysregulation. It is important to note that while these associations indicate potential causal relationships, they do not establish definitive causality. Moreover, the analysis found no strong evidence that AA directly alters microbiota composition, strengthening the hypothesis that dysbiosis may precede disease onset rather than result from it.

These insights lay the groundwork for microbiota-based adjunct therapies in AA management. Probiotics and synbiotics aimed at restoring beneficial species, dietary interventions targeting gut homeostasis, and emerging modalities such as fecal microbiota transplantation (FMT) — though still investigational in hematologic settings — may offer future therapeutic options [7,8]. As these strategies mature, regional microbiome diversity and population-specific patterns must be considered to optimize translational relevance for Indian patients.

In conclusion, microbiota diversity shifts in AA represent a promising frontier for research and intervention. We advocate for

the inclusion of microbiome profiling in AA registries, expansion of longitudinal studies through ICMR-backed consortia, and adaptation of clinical guidelines that incorporate gut health. Recognizing microbial communities not as collateral victims of disease, but as potential contributors to pathogenesis, reframes our approach to AA and opens doors for precision therapeutics in India's hematologic landscape.

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