Effects of Kibow Probiotic Supplementation Renadyl™ on Uremic Toxins in Hemodialysis Patients

Subodh J. Saggi1, Mary C. Mallappalli1, Usha N. Vyas2, Griet Lrl Glorieux3, Peter Liang1, Pari Ranganathan2, Bohdan Pechenyak1, Gary R. Briefel1, Lorraine L.a Thomas1, Raymond C. Vanholder1, Natarajan Ranganathan1 and Eli A. Friedman1.

1SUNY Downstate Medical Center, Brooklyn, NY; 2Kibow Biotech, Inc, Newton Square, PA and 3Ghent Univ Hospital, Ghent, Belgium.

Abstract

Our prior studies in patients with CKD 3-4 (n=31) given Renadyl™, a safe proprietary dietary supplement that metabolizes nitrogenous wastes in the bowel, at a dose of 90-270 B CFU per day, over a 4 month period showed that BUN, creatinine and K+ levels declined. We now conducted a prospective double blind cross over trial with placebo and Renadyl™ in 26 stable CKD patients on hemodialysis. Dosage administered was 180 B CFU per day, given in 3 divided doses. Our primary aim was a 20% reduction in BUN levels over an 8 week period. Patients’ dialysis prescriptions administered was 180 B CFU per day, given in 3 divided doses. Our primary aim was to identify uremic toxins or markers of inflammation that are decreased in ESRD patients is safe and showed a protective effect by the trend to reduce markers of inflammation. Further investigation in a larger population or at a higher dose might yield mechanistic insights into the probiotic effects on the inflammatory cascade of uremia.

Methods

Patients were assigned to take either the placebo or Renadyl™ first for 8 weeks, followed by a washout period of 8 weeks and finishing with 8 weeks of the placebo or Renadyl™ (depending on which was taken first). Each patient’s blood samples were taken at the first visit, after finishing 8 weeks of placebo, and after finishing 8 weeks of Renadyl™. The sera were used to measure C-reactive protein (CRP), total and/or free serum concentrations of indoxyl sulfate, indole acetic acid, p-cresyl sulfate, hippuric acid, serum pentosidine, 3-carboxy-4-methyl-5-propyl-2-furan-propanoic acid (CMPF), uric acid and beta-2 microglobulin. Solutes were measured by HPLC and ELISA. Qol changes were assessed by a modified SF-36 questionnaire. Patient adherence was assessed by pill count and stool culture to verify probiotic growth during study and absence during placebo period. Data were analyzed using ANOVA for a crossover study with a mixed model methodology in SAS to account for treatment, period and sequence effects.

Results

Our data show that Renadyl™ reduces the levels of CRP, the levels of total indoxyl glucuronide, and WBC counts. This suggests that Renadyl™ exerts some protective effect by reducing the levels of inflammation in the form of WBC counts and CRP. Future investigations using a larger population may yield mechanistic insights on Probiocti's effects on uremia and the inflammatory cascade.

Conclusions


References

Objectives

Previous studies had shown that Renadyl™ was able to improve quality of life and decrease BUN in patients with ESRD. However, the mechanism of action and what toxins are also reduced are unknown. Our goal is to identify uremic toxins or markers of inflammation that are decreased in response to the Probiotic Renadyl™.