emerging strategy in clinical life and appear to be effective in targeting IS.

**Methodology:** Study conducted on 92 ESRD patients on regular Hemodialysis from January/2017 to March/2017 and patients divided to two groups: intervention group (50 patients) receiving probiotics regimen containing 5 strains for 6 weeks while control group (42 patients) receiving placebo for the same period. Indoxyl Sulphate using ELISA measured before and after intervention.

**Results:** Reduction of IS 14 ± 22.71 μg/ml vs 3.6 ± 14.33 μg/ml in intervention group vs control group respectively P value 0.02. Reduction in serum phosphorus, CRP, lipid profile was recorded. **Conclusion:** Probiotics cause reduction in IS with reduction in phosphorus, CRP, lipid profile with no reported side effects.

**Role of activated charcoal in limiting the progression of chronic kidney disease in experimental albino rats**

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Chronic kidney disease (CKD) impairs the intestinal barrier function which is mediated by influx of urea and its conversion to ammonia by microbial urease. The degradation of epithelial tight junction (TJ) allows influx of noxious products such as indoxyl sulfate causing systemic inflammation. **Aim:** to explore the ability of oral activated charcoal to adsorb urea, urea-derived ammonia and indoxyl sulfate and its ability to ameliorate CKD-induced intestinal epithelial barrier disruption, systemic inflammation and progression of Chronic kidney disease.

**Methods:** Rats were randomized into four groups with regular chow diet. Group I: Sham control, Group II 5/6th nephrectomized rats for 6 weeks, group III 5/6th nephrectomized rats for 6 weeks supplemented orally by activated charcoal immediately after surgery (4 g/kg/day), group IV 5/6th nephrectomized rats for 6 weeks supplemented by activated charcoal 2 weeks after surgery (4 g/kg/day). Animals were scarified and the brain tissues were processed for histopathological examination.

**Results:** Compared with the sham controls, the untreated CKD rats showed significantly elevated serum urea, creatinine, indoxyl sulfate, C Reactive Protein as well as increased diastolic blood pressure, reduced body weight gain and histological manifestation of colonic erosions and renal fibrosis. Both early and late administration of activated charcoal resulted in significant reduction in serum urea, creatinine and C Reactive protein. Also relative improvement in body weight gain with partial restoration of the colonic mucosal integrity and reduction in renal fibrosis index were observed with charcoal treatment. Indoxyl sulfate was only significantly reduced with early treatment. Activated charcoal was unable to combat the change in diastolic blood pressure with CKD.

**Conclusion:** Administration of activated charcoal attenuated uremia-induced disruption of colonic mucosa and the associated endotoxemia, inflammation and progression of CKD specially with early supplementation.

**Influence of stem cells and hepatocyte growth factor on aluminum induced central neurotoxicity in rats**

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Recently the trials to use each of Mesenchymal stem cells (MSCs) and Hepatocyte growth factor (HGF) as an effective medication for treatment has been increased.

**Aim:** Exploring the potential therapeutic effect of a combination of both bone marrow Mesenchymal stem cells (BM-MSCs) and Hepatocyte growth factor (HGF) in Al induced neurotoxicity as a trial to limit the progression of Alzheimer’s disease (AD).

**Methodology:** the present study was carried on 60 male albino rats. 10 rats were used for isolation and propagation of MSCs, and the remaining 50 rats were divided into five equal groups. Group I served as control group, Group II, Aluminum Neurotoxic Rats: Rats were injected i.p. with solution containing 100 mg/kg AlCl3, 5 days /week for 15 weeks. Group III, Aluminum Neurotoxic Rats treated with Mesenchymal stem cells. Group IV, Aluminum Neurotoxic Rats treated with Hepatocyte growth factor. Group V was Aluminum Neurotoxic Rats treated with both drugs. As Behavioral test; radial arm maze was performed. It determined the impairment of both reference and working memories. After behavioral test; at the end of the 15th week, rats were scarified and the brain tissues were processed for estimation of APP and BACE-1 gene expression, detection of stem cells homing and detection of Bcl2 by immunohistochemistry.

**Result:** Administration of Al caused the following changes: In radial arm maze, in the final outcome rats showed memory impairment that characterized by increasing both time and working correct errors. In addition, it caused significant increase in APP and BACE-1 gene expression. This was accompanied by decrease of Bcl2 in brain sections. Treatment with BM-MSCs caused the following changes: In radial arm maze, rats were closer to Al group, while they showed finally increased time and working correct errors versus normal control group. Also, it caused significant decrease in APP and BACE1 gene expression and relative restoration of the brain Bcl2 content after its depletion with Al intoxication. Treatment with HGF caused the following changes: In radial arm maze, rats showed obvious improvement characterized by decreased time needed to end the task, decreased working correct errors and total working errors versus Al group. At the same time, the reference errors were decreased relative to normal control rats. Also, it caused significant decrease in APP and BACE1 gene expression and Relative restoration of the brain Bcl2 content. Simultaneous treatment with BM-MSCs and HGF caused the following changes: In radial arm maze, rats’ behavior was definitely midway between both Al and normal control groups, with the final outcome showed insignificant change versus them. Also, it caused significant decrease in APP and BACE1 gene expression and relative restoration of the brain Bcl2 content after its depletion with Al intoxication.

**Conclusion:** Treatment of Al intoxicated rats with MSCs and HGF ameliorated the Al neurotoxic features. Combination of both caused synergistic effect.