EFFECT OF GLYCEMIC CONTROL ON ERYTHROPOIESIS STIMULATING AGENT REQUIREMENT IN DIABETIC MAINTENANCE HEMODIALYSIS PATIENTS

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Anemia management is an important aspect of maintenance hemodialysis (MHD) therapy. It has been reported that diabetics have lower serum erythropoietin concentration than non-diabetics while maintaining similar hemoglobin levels. This may be to an effect of angiotensin II on erythropoiesis. Diabetic associated hyperglycemia reflected as high hemoglobin A1c (HbA1c), has been found to increase angiotensin II production. A large study has reported lower recombinant human erythropoietin (rHuEpo) requirement in diabetic MHD patients. To our knowledge there is no data on the influence of glycemic control on rHu-Epo requirements. This study was performed to determine if glycemic control affected rHu-Epo requirement in diabetic MHD patients. A retrospective analysis of MHD patients from two large urban hemodialysis units was performed. Patients were included if they carried the diagnosis of diabetes and on MHD for over 3 months. Exclusion criteria included active infection, evidence of active blood loss, and recent hospitalization within the month prior to data collection. Patients were divided into two groups, low (<6.5%) and high (≥ 6.5%) HbA1c. Data from a total of 149 patients were analyzed, and of them 92 diabetic patients included. We found patients with HgA1c of < 6.5% (n=49) required a significantly higher dose of rHu-Epo when compared to those in the elevated HbA1c group (11,553 ± 10,306 units/HD vs. 6,147 ± 7585 units/ HD, p= 0.006) to maintain the same level of hemoglobin. There was no statistical difference in markers of iron adequacy and inflammation. There was a significant negative correlation of HbA1c with rHu-Epo dose (-0.289, p=0.006). These data suggest that glycemic control may play a role in rHu-Epo responsiveness. There was a decreased rHu-Epo requirement in diabetic patients with high HbA1c levels. This counterintuitive response may reflect an effect of hyperglycemia through angiotensin II mediated erythropoiesis. The clinical significance and mechanism of these findings need to be further elucidated.