RESPONSE TO ERYTHROPOIETIN THERAPY AND ITS RELATION TO THE PRESENCE OF ANTI-ERYTHROPOIETIN ANTIBODIES IN PEDIATRIC PATIENTS ON REGULAR HEMODIALYSIS

Thesis
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INTRODUCTION

Anemia develops early in the course of renal failure, becomes prominent as the disease progress, and contributes substantially to disability. Patients with renal failure have lower plasma levels of erythropoietin and less erythropoiesis than other patients with similar degree of anemia, suggesting inadequate production of erythropoietin by the diseased kidney as the primary mechanism of renal anemia (Klahr, 1996).

Over the last decade, the availability of rHuEpo had led to the almost complete disappearance of the severe anemia of ESRD. However, despite a widespread in the use and increased doses of rHuEpo, a substantial percentage of patients didn't respond to therapy and didn't achieve HCT level of more than 30% (Locatelli et al., 2001).

Resistance to rHuEpo therapy in patients with CRF may be due to iron or foliate deficiency, aluminum toxicity or hyperparathyroidism with bone marrow fibrosis. Inadequate HD can also contribute to resistance to erythropoietin therapy (MacDougall I., 2004).

A previous study had suggested the presence of anti-erythropoietin antibodies as an additional cause of erythropoietin resistance (Casatelli et al., 2000), and a significant increase in the number of patients developing severe anemia had been noted during the course of erythropoietin therapy due to neutralizing antibodies (Echardt and Casadevall, 2003).

The presence of strong neutralizing anti-erythropoietin antibodies is considered the most common cause of occurrence of pure red cell aplasia (PRCA) which occurs in children (Casadevall, 2002).