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Use of High Dose Defroxamine and Nocturnal Home Hemodialysis to Reduce Secondary Hemochromatosis: A Case Study

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Topic: Approximately 50% of the patients who have End Stage Renal Disease (ESRD) on maintenance hemodialysis have serum ferritin levels > 500 ng/ml. Patients who receive frequent blood transfusions are at risk for secondary hemochromatosis since each unit of transfused packed red blood cells contain 200 to 250 mg of iron. Treatment for secondary hemochromatosis includes phlebotomy and chelation therapy. Defroxamine was introduced into clinical practice as an iron chelator in the 1970's. Hemodialysis allows the low molecular weighted chelated ferric iron to pass through dialysis membranes. Combining hemodialysis and chelation therapy is a viable intervention to reduce serum ferritin levels in ESRD patients with secondary hemochromatosis resulting from frequent blood transfusions.

Approach: A 40-year-old male with end stage renal disease (ESRD), myelodysplastic syndrome (MDS), type 1 diabetes, inflammatory bowel disease, and osteoarthritis developed secondary hemochromatosis related to frequent blood transfusions. He initiated dialysis in 2004, received a renal transplant in 2006 which failed in 2011 and was subsequently restarted on in-center Hemodialysis. Starting in March 2011 until May 2016 he received 201 transfusions of packed red blood cells. His serum ferritin peeked in July 2016 (10338 ng/ml). In order to combat the hemochromatosis the patient received 500 mg IM Defroxamine three times per week starting February 2013 until February 2016. The patient started short frequent home hemodialysis in March 2016. He switched to nocturnal home hemodialysis in July 2016. Upon initiation of home hemodialysis, the Defroxamine was administered intravenously. He received 1000 mg IV during the first hour of treatment five times per week starting April 2016. Upon initiation of nocturnal hemodialysis in June 2016, the dose was increase to 1500 mg IV (infused over six to seven hours five times per week). The amount of iron in his effluent was measured which lead to subsequent dose increases in July 2016 (2000 mg), August 2016 (2500mg) and in October 2016 to his maximum dose of 3000 mg.

Conclusion: There has been a steady decrease in the serum ferritin levels from a peak of 10338 ng/ml (July 2016) to 3216 ng/ml in September 2017. His Transferrin Saturation decreased from a peaked at 157% (October 2014) to 105% in July 2017. Initial effluent iron levels were 0.342 mg/L (6.84 mg per treatment, approximately 34.2 mg per week, 136.8 mg per month) on 1000 mg of Defroxamin. Effluent iron levels on 2500 mg of Defroxamin were 0.702 mg/L (14.04 mg per treatment, approximately 70.2 mg per week, 280.8 mg per month).

It was previously thought that, due to his MDS, he would be a non-responder to erythropoietin therapy and thus received his last dose of Darbepoetin Alfa in July 2011. In March of 2016, Darbepoetin Alfa was initiated with excellent results and a steady increase in his hemoglobin from 9.2 to 12.0 in July 2016. His last transfusion was August 2016.

Relevance/Implications: This patient, undergoing high dose Defroxamin combined with nocturnal hemodialysis, had a reduction in his serum ferritin level by 68.9% within 16 months. Further studies using this therapeutic regime are warranted.

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