

Successful administration of iron dextran in a patient who experienced a life threatening reaction to intravenous iron dextran

Severe life threatening reactions to iron dextran are rare with an estimated incidence of 0.6%.¹ Anaphylaxis is the clinical manifestation of immediate hypersensitivity that occurs after the interaction between a specific antigen and cell-bound antibody. Anaphylactoid or anaphylaxis-like reactions are clinically similar to anaphylaxis but are not antibody mediated. They result from agents acting directly on mast cells and/or basophils causing mediator release or activating the complement cascade. We report a patient with a severe life-threatening reaction to iron dextran who was able to tolerate iron dextran after protective pretreatment with methylprednisolone, diphenhydramine, ephedrine, and dextran 1 (Promit).

A 58-year-old white female developed a short bowel syndrome after subtotal gastrectomy and multiple small bowel resections due to adhesions. Severe iron deficiency anemia ensued because of intolerance to impaired absorption of oral iron. Subsequently, she experienced recurrent severe hemolytic transfusion reactions and a life-threatening reaction after infusion of intravenous iron. Because there were no alternative methods of iron administration for this patient, she was treated with iron dextran utilizing a pretreatment protocol described previously by Monaghan et al.² A similar graded challenge protocol with rapid dose escalation had also been successfully utilized by Altman and Petersen in a patient with an anaphylactoid reaction to iron dextran.³ The patient was given methylprednisolone 50 mg intravenously 13, 7, and 1 hour prior to iron

dextran. Diphenhydramine 50 mg intramuscularly and ephedrine 25 mg intramuscularly were given 1 hour prior to iron dextran. Dextran 1 (150 mg/mL), 20 mL intravenously was given one minute prior to iron dextran. Iron dextran (50 mg in 100 mL normal saline) was administered intravenously at 20 mL/hour just after dextran 1. Subsequent doses of iron dextran were administered at: 22 hours (100 mg in 250 mL NS at 40 mL/h); 28 hours (200 mg in 250 mL NS at 50 mL/h); 28 hours (200 mg in 250 mL NS at 50 mL/h); 47 hours (400 mg in 250 mL NS at 60 mL/h); 52 hours (500 mg in 250 mL NS at 60 mL/h); 71 hours (500 mg in 250 mL NS at 80 mL/h); and 75 hours (250 mg in 250 mL NS at 80 mL/h). No further doses of iron dextran were administered.

The exact mechanism by which iron dextran causes anaphylaxis or an anaphylactoid reaction is unknown. One previously suggested cause has been release of mast cell/basophil mediators by anaphylatoxin derived from dextran specific immune complexes.⁴ Pretreatment with dextran 1 is thought to act by binding to and neutralizing excess dextran-specific antibodies in the circulation. A postulated non-immunologic mechanism may be that preadministration of dextran 1 blocks corresponding receptors on mast cells/basophils, thereby preventing direct release of mediators by the iron-dextran complex. The package insert suggests that pretreatment with Promit alone should suffice to prevent a reaction. Since pretreatment with methylprednisolone, diphenhydramine, and ephedrine would nonspecifically attenuate either ana-

phylactic or anaphylactoid reactions to the iron-dextran complex or Promit itself, we elected to include pharmacologic prophylaxis. Specific IgE directed toward iron-dextran is rare, and has apparently been demonstrated in only a single patient.⁵ Thus rapid desensitization was not felt to be necessary.

Our patient received two grams of iron dextran without adverse effects. This is the third report of a life-threatening reaction to iron dextran in a patient who was able to tolerate parenteral iron therapy after undergoing a pretreatment regimen with methylprednisolone, diphenhydramine, ephedrine, and dextran 1.^{2,3} The ability to prevent a reaction in a patient who needs iron dextran is invaluable and potentially life saving. This case report confirms the efficacy of this treatment protocol in such patients.

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