Hereditary hemochromatosis is characterized by the abnormal progressive absorption of dietary iron in the intestines, which is then stored in the liver, heart, and other organs. Inherited as an autosomal recessive disorder, the most common genotype that causes hereditary hemochromatosis is HFE C282Y/C282Y (Adams & Barton, 2010). Signs and symptoms often appear in adults aged 50–69. Complications in the liver include fibrosis, cirrhosis, complete liver failure, or hepatocellular carcinoma. Damage to other organs from iron overload may result in diabetes mellitus, cardiomyopathy, gonadal dysfunction, arthritis, and dementia. Excess iron storage is thought to be toxic to cells. Increased amounts of free radicals are generated that can damage cellular and subcellular membranes. In this disease, the goal of treatment with phlebotomy is to deplete enough iron to normalize the body’s iron stores by mobilizing approximately the same amount of iron out of the liver. The goal of treatment depends on the baseline level of iron overload should be reconsidered. Once the ferritin goal is met, levels will be drawn monthly until the level reaches a total less than 200 mg/dl; thereafter, it will be drawn after every one or two phlebotomies. If the patient becomes anemic or the ferritin goal is reached after only a few phlebotomies, the diagnosis of iron overload should be reconsidered.

In addition to phlebotomy, some practitioners recommend specific dietary restrictions to reduce dietary absorption of iron. The restrictions include limiting alcohol intake and avoiding vitamin C supplements, oral iron replacement,