Assessing iron overload and toxicity

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Iron mediated toxicity is generally observed in tissues that accumulate the highest concentrations of excess iron. These usually follow increased iron intake, from gastrointestinal absorption or from repeated blood transfusions. Despite early differences of iron distribution with transfusional and non-transfusional overload, later stages of iron overload are similar, with extra-hepatic spread to endocrine tissues and myocardium. In some conditions, e.g. Sickle Cell Disease, this is less likely than others, e.g. Thalassemia Major. Very rarely, iron toxicity results from mal-distribution of iron without generalized iron overload. Assessment of iron overload should therefore consider not only the quantity of body iron but also its distribution. While serum ferritin is useful to follow trends in iron overload, it has limitations; overload is underestimated in Thalassemia Intermedia, while hyperferritinemia is often observed without iron overload if liver disease or inflammation is present. The utility of plasma iron measures such as NTBI or LPI to predict risk remains unclear in routine practice. Validated MRI techniques are now available to quantitate liver iron non-invasively, to predict risks of cardiomyopathy and to delineate iron distribution. Their wider application should reduce morbidity from endocrine dysfunction and mortality from cardiomyopathy and liver disease, provided appropriate chelation options are also available.