

IRON - TRACE ELEMENT

Introduction

Atomic mass 55.9, iron is an essential component of haemoglobin with disorders of deficiency well documented. Iron toxicity is discussed further.

Exposure

Acute: excessive ingestion. This is usually accidental, particularly in children, but may also represent intentional overdose.

Chronic: may result from excessive absorption of dietary intake or parenteral iron administration. Excessive absorption may occur in the setting of ineffective erythropoiesis (eg sideroblastic anaemia, severe alpha- or beta-thalassaemia), porphyria cutanea tarda, chronic excessive alcohol ingestion or chronic liver disease. Parenteral iron administration is generally the result of regular red cell transfusions, such as may be required in the setting of severe beta-thalassaemia, sickle cell disease, myelodysplastic syndromes and refractory aplastic anaemia. The cause of iron overload, in the absence of transfusional iron overload, is almost always hereditary haemochromatosis (HH).

Pathology

Acute toxicity

Initial (1/2-6 hours) features are gastrointestinal: abdominal pain, vomiting, diarrhoea, haematemesis, melaena, lethargy. Shock and metabolic acidosis may then intervene after 6-72 hours, often after a period of apparent clinical improvement. It is the result of hypovolaemia and/or cardiotoxicity. Hepatotoxicity is common from 12-96 hours. After 2-8 weeks bowel obstruction may occur secondary to scarring of the gastrointestinal tract.

Monitoring

Acute

Serum iron: very useful in acute toxicity.
Serum total iron binding capacity – unreliable in acute toxicity.

Chronic

Serum iron, iron saturation and ferritin are all expected to increase.

A fasting transferrin saturation ≥ 60 percent in men or ≥ 50 percent in women will detect about 90 percent of patients with homozygous HH. However, many investigators have advocated using a "cutoff" value of 45 percent transferrin saturation for both men and women, which will lead to fewer patients being missed, at the expense of an increased false positive rate.

An elevated plasma ferritin is generally less sensitive than the transferrin saturation in screening for HH because a greater degree of iron overload is required to raise the ferritin concentration. Genetic testing for HH should be performed in all patients suspected of having this disease. Quantitation of iron in liver tissue biopsy is diagnostic of haemochromatosis.

Analysis

Trace Elements analyses iron using inductively coupled plasma mass spectrometry.

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