PATHOLOGY NEWS

JDJ DIAGNOSTICS | Pathology Laboratories

Excellence • Integrity • Quality

Iron Deficiency

Dr. Adele Visser

Introduction

Iron deficiency is the most common cause of anaemia in the world, with the incidence as high as 10% amongst women of child-bearing age. The manifestation of iron deficiency may be true, due to a lack of iron stores, or functional, due to inadequate utilization of iron stores.

Pathophysiological Processes of Iron Deficiency

Iron deficiency is a progressive process and red cells undergo intensifying morphological changes. The range of laboratory parameters which undergo change will also change over time, with some more sensitive to early detection than others.

	Expected change with Iron deficiency	Expected change with functional iron deficiency	Causes of misleading results
BM hemosiderin	Reduced iron stores with iron stain.	Adequate or increased iron stores with iron stain.	Invasive sampling Inadequate sampling.
Ferritin	Decreased	Decreased	Inflammation or liver disease falsely increase levels.
sTfR	Increased		Not routinely available as test.
Iron studies	• Low serum iron. • Low transferrin saturation. • Increased transferrin concentration.	• Low serum iron. • Low transferrin saturation. • Normal to low transferrin concentration.	Altered with inflammation.
RCC Hb	Decreased	Decreased	Polycythaemia Vera may mislead interpretation.
MCV MCH	Decreased	Normal to Decreased	Seen in anaemia of chronic disease, thalassaemia etc.

Iron is not only used in the formation of haemoglobin, but also forms part of the iron-containing complex of cytochrome enzymes which are essential in the oxidative phosphorylation pathway.

Therefore, in patients with iron deficiency, although tiredness may be ascribed to the degree of anaemia, it is greatly exacerbated by the inadequate ATP production on cellular level as well.

In the event of a severe anaemia where a transfusion is indicated, the underlying cause of iron deficiency should be established before the transfusion is undertaken. Red cell concentrates contain ample amounts of iron, which serves as a bolus infusion, which will likely alter all subsequent biochemical testing of iron status.

Causes of True Iron Deficiency

Increased iron loss	Bleeding	Surgery or trauma Gastrointestinal bleeds Menorrhagia Blood donation or phlebotomy
	Chronic haemoglobinuria	Mechanical heart valve haemolysis Paroxysmal nocturnal haemoglobinuria
Decreased intake of iron	Dietary deficiency	Limited meat intake
	Malabsorption	Sprue Achlorhydria due to partial gastrectomy, atrophy, PPI use or inflammatory bowel disease
Increased utilization of iron		Pregnancy Rapid growth

Functional iron deficiency

Patients suffering from chronic infectious, inflammatory or neoplastic disease often present with hypoferremia despite adequate or even increased iron stores. The pro-inflammatory cytokines suppress effective haemopoiesis on various levels, including a blunted erythropoietin response, sequestration of iron in macrophages and reduction in transfer of iron to red cell pool. The typical haematological manifestation is a mild anaemia with normocytic to microcytic morphology.

Biochemistry shows a low serum iron and transferrin saturation as with true iron deficiency; however it also shows a normal to low transferrin concentration.

Monitoring treatment of iron deficiency

An array of oral and parenteral supplementations are available, and can be used depending on severity of the deficiency and side-effects experienced by the patient. Once oral therapy has been initiated, the haemoglobin level can be expected to increase by 2g/dL every 3 weeks, provided absorption is adequate and there is not continued loss.

An increased reticulocyte count is expected within 3 - 4 days following initiation of treatment.

If a patient does not response to treatment, one should consider continued haemorrhage, failure to comply with treatment, incorrect diagnosis (particularly thalassaemia trait and sideroblastic anaemia), mixed deficiencies, anaemia of chronic disease and malabsorption of iron supplements.

References

1. Rodgers G.P., Young N.S. 2005. Bethesda Handbook of Clinical Haematology.
2. Hoffbrand A.V., Moss, P.A.H, Pettit J.E. 2007. Essential Haematology.

JDJ Pathology Laboratories

Suite LG 2, Musgrave Park,
18 Musgrave Road
Durban, 4001

Tel +27 31 201 4647
Fax +27 31 201 4910

clientservices@jdjd.co.za
www.jdjd.co.za

www.facebook.com/JDJDiagnostics/