Diagnosis of Iron Deficiency Anaemia

Serum iron is not useful in the diagnosis of iron deficiency. Serum iron fluctuates widely regardless of iron stores. Why? Bacteria cannot store iron, and depend on environmental iron for replication (Remember blood agar?). Thus reducing serum iron will reduce bacterial replication rates, and augments the host organism's ability to fight the infection - a rather elegant physiological defence mechanism. (Bacteria species differ in their 'sensitivity' to iron levels). Hence there is a teleological rationale that part of the inflammatory response is to reduce serum iron by restricting iron release from stores and reducing iron absorption. Hepcidin (discovered 2001) is a key regulator of this process. From a Darwinian perspective, inflammation has been overwhelmingly due to infection, but since the 'pax antibiotica' after penicillin, this is less relevant. In the absence of bacterial infection, some aspects of the inflammatory response (such as iron regulation) are non-advantageous. So..... patients with chronic inflammatory states may develop actual or functional iron deficiency. And in the context of general inflammation, ferritin may appear adequate but there may be functional iron deficiency.

- A Ferritin level **below 30 µg/L is diagnostic of iron deficiency.**
- Ferritin levels **between 30-100 µg/L are highly suggestive** of iron deficiency. CRP will help clarify if infection is present.
- Iron deficiency is **unlikely when ferritin is >100 µg/L.** If anaemia is present, consider functional iron deficiency (FID) also known as anaemia of chronic infection.

When the clinical features and haematology profile are suggestive of iron deficiency, but the ferritin is normal, consultation with a haematologist / pathologist is suggested. Additional tests, such as Total Iron Binding Capacity, Transferrin saturation and C-Reactive Protein (CRP) to identify inflammation, may be required to identify functional iron deficiency (FID), where iron stores may be adequate, but cannot be utilised.

It has been suggested that Iron therapy in the context of bacterial infection may actually “fuel" sepsis. This is an ongoing discussion ‘in the literature’. If it does, transfusion may have the same effect, although this too is controversial. The appropriate role of EPO therapy is not clear. EPO may be thrombogenic and may accelerate metastases. Pragmatically, and in clinical trials thus far, iron therapy alone is effective.