Clinical case review

Haemoglobin 24 g/L and still alive

Case scenario and GP commentary: **LAWRENCE WAPNAH** BCom(Hons), MAcc, CA, BM BS, FRACGP
Commentary: **CHRIS BARNES** MB BS, FRACP, FRACPA, and **ALISON STREET** AO, MB BS, FRACP, FRACPA

Parents are not always aware of the nutritional needs of their children, as illustrated in this case of severe iron deficiency anaemia in a young boy.

Case scenario

The presentation

A boy aged 1 year and 8 months was brought to see at me at my city practice. His mother was concerned because during the past four weeks he had lost his usual interest in running around and playing with toys, was more distressed than usual (crying when not clinging to her) and was eating less but drinking lots of milk.

On examination, the boy had a sallow appearance, but was otherwise morphologically normal, fully co-operative, smiling, orientated and helpful for his age. He was tachycardic and had a prominent pansystolic murmur. There was nothing otherwise of note. The boy had been born at term, his vaccinations were up to date and he had had an unremarkable history to then. Opportunistic physical examination of his 4-year-old sister, who was present at the appointment, was normal.

With differential diagnoses including severe anaemia and the worsening of an unknown congenital cardiac condition, I arranged for the boy to attend the city's main children's hospital. Although concerned and despite my urging, his mother was reluctant to take him to hospital that morning, so we compromised and I organised some urgent blood tests, with planned review later that day.

A couple of hours later, the local pathologist contacted me: the boy's haemoglobin level was 25 g/L (normal value, 120 g/L), but in clotted blood. The pathologist asked whether the result could be correct and if the test needed repeating. I said yes to both and phoned the boy's mother to find she had taken her son to hospital, as I had initially asked, straight after the blood test.

Hospital admission and follow up

At the hospital, repeat and further blood tests were conducted. The boy's haemoglobin was 24 g/L and mean corpuscular volume 49 fL (normal range, 77 to 98 fL). White cell and platelet counts were normal. Subsequent iron studies showed a serum iron concentration of 1 µmol/L (normal, 12 to 31 µmol/L), serum transferrin 47 µmol/L (normal, 20 to 45 µmol/L), transferrin saturation 1% (normal, 15 to 55%), and serum ferritin less than 5 µg/L (normal, 10 to 100 µg/L).

After being given two units of blood, the boy was completely back to normal. His diagnosis was 'dietary-caused, severe iron deficiency anaemia'. Basically, he just drank too much milk. This is the lowest haemoglobin I have seen in a person not actively bleeding from a major haemorrhage.

How common is such a low haemoglobin level in iron-deficient infants? How likely is it that a change in diet led to iron deficiency in only four weeks? How much can a clotted sample of blood affect the results of haematological tests?

Dr Wapnah is a General Practitioner at Seacrest Medical Centre with admitting rights to the Mount Hospital, Perth, WA, a Doctor in the Remote Area Health Corps, and is on the Board of Diabetes WA. Dr Barnes is a Consultant and Laboratory Haematologist at the Royal Children's and Royal Women's Hospitals, and Director of the Henry Ekert Haemophilia Treatment Centre, Royal Children's Hospital, Melbourne, Vic. Associate Professor Street is Head of the Haemostasis/ Thrombosis Unit and Haemophilia Centre, Alfred Hospital, Melbourne, Vic.

Commentary

Epidemiology and aetiology of iron deficiency

Although severe iron deficiency anaemia in Australian children is relatively uncommon, iron deficiency is common and continues to be a significant problem in selected risk groups, including children from immigrant families and children of Aboriginal decent.[1,2]

A number of factors may contribute to the development of iron deficiency in children. Although the presence of parasitic disease and enteropathies, including coeliac disease, may be associated with iron deficiency, a diet deficient in iron and with excessive cows' milk intake, as in the case above, continues to be the major cause for the development of iron deficiency in children.[3] Cows' milk is low in iron and its iron is poorly absorbed. Early introduction and excessive cows' milk consumption has been shown in several large international studies to correlate with low iron stores and anaemia. In a study of anaemia in 12-month-old infants in Europe, every additional month of exposure to cows' milk was associated with an average decrease of 20 g/L in haemoglobin.[4] Cows' milk is an important part of the diet of young children, providing a rich source of calcium, but the diet needs also to include iron-containing food.

In addition to the haematological abnormalities associated with iron deficiency, the link between this deficiency and lower developmental scores is well established.[5] The developmental effects of iron deficiency appear to persist despite iron replacement therapy.[6] Pica (a craving to eat strange foods or objects) is an unusual but well-recognised manifestation of the non-haematological impact of iron deficiency.

The slow development of iron deficiency and insidious nature of the progression of anaemia in patients with nutritional deficiency can often lead to late presentation, with patients showing marked anaemia and cardiorespiratory compensation. Children are usually pale, often irritable and may be difficult to

examine. In the case above, the lack of cardiovascular compromise and symptoms associated with a haemoglobin of 25 g/L suggests evolution of anaemia over a prolonged time with corresponding haemodynamic adjustment.

Diagnosis of iron deficiency

The diagnosis of severe iron deficiency is usually straightforward. A marked microcytic anaemia with an associated increase in red cell variation in size and shape (often referred to as an increase in the red cell distribution width is typical of iron deficiency (Figure). A reactive thrombocytosis is often present. Iron studies demonstrate a low serum ferritin concentration and an increase in the total iron binding capacity. An isolated low serum iron level should not be used to diagnose iron deficiency because serum iron fluctuates widely in the setting of intercurrent infections and dietary intake.

In more mild cases, the diagnosis may not be as easily identified and the serum ferritin may be falsely elevated in the presence of comorbid conditions such as inflammatory conditions and infections; repeat testing may be helpful following resolution of the inflammatory process. Supplemental investigations, including assessment of soluble transferrin receptor, may be required, as may, rarely, a trial of iron therapy followed by a reassessment of haematological parameters.

The case presented was complicated by the full blood sample having a clot. The presence of a small clot should not affect haemoglobin estimation; haemoglobin estimations are usually performed on cell lysates. The presence of even a small clot may, however, affect the white cell count and platelet count.

Treatment of iron deficiency

The introduction of iron therapy in even severely iron-deficient children may be associated with a rapid response in the improvement in haematological parameters, with a brisk reticulocyte response and recovery of haemoglobin by up to 20 to 30 g/L/week. In the absence of symptoms of cardiorespiratory decompensation, such as cardiac failure, prompt initiation of iron therapy can avoid blood transfusion even in the most severely anaemic patients, thereby reducing costs and any potential side effects of transfusion.

Children often have problems with compliance with iron therapy because many iron preparations are unpalatable and may have gastrointestinal side effects. Ferrous sulfate is available in liquid form in Australia (Ferro-Liquid) and should be given in a dose of 0.5 mL/kg/day. Administration in divided doses or at night and an increase in dietary fibre are helpful in limiting gastrointestinal side effects. Parenteral iron preparations are also available, but intramuscular injections should be avoided because of the potential of the iron causing a 'tattoo' on the skin surface. Intravenous iron infusions can be given to patients with iron deficiency that is difficult to manage, but are associated with a risk of allergic reactions and are expensive.

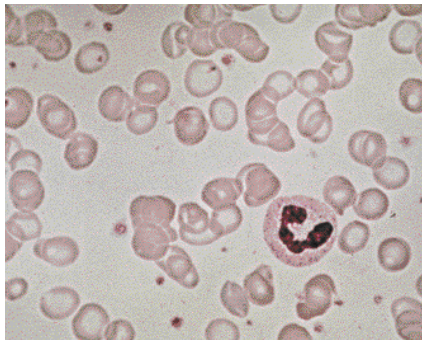
Iron repletion is a gradual process and therapy should be continued for three months. It is important to encourage parents and children to persist with treatment; focusing on the need to maximise neurodevelopmental outcome in children is often helpful for parents to understand the importance of iron therapy.

GP commentary

How does a case of life-threatening, nutritional, severe anaemia happen in a relatively affluent area of an Australian city? The answer in this case was simple. The daughter liked and had wanted other food, so the mother had given her it; however, the son wanted mainly milk, so the mother gave him more milk. Their mother had little insight into the nutritional needs of children. The children were otherwise well cared for and the mother seemed to be responsible.

Parents get advice on how to feed their children from a variety of sources: the extended family and friends, their own education, food advertising, the media, and health professionals. Some of this advice is conflicting; some is misleading.

Although iron deficiency is relatively straightforward to diagnose on the basis of blood tests, GPs rightly do not routinely perform blood tests on small children (outside of certain demographics or locations such as remote Aboriginal communities). They therefore have to rely on harder clinical judgement. MT



PHOTOLIBRARY

Figure. Light microscopy of blood smear from a patient with iron-deficiency anaemia showing irregularly shaped and smaller than normal red blood cells.

References

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COMPETING INTERESTS. None.