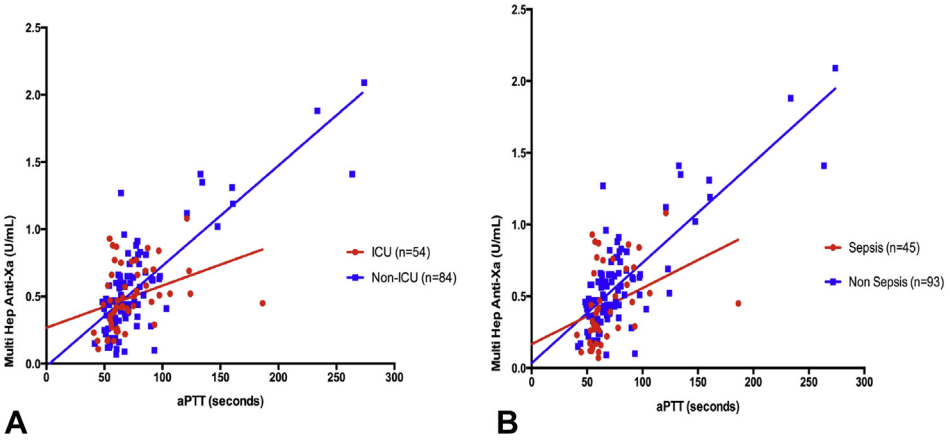

Fig. 2 Correlation for Multi Hep Anti-Xa assay and aPTT for (A) ICU versus non-ICU patients and for (B) septic versus non-septic patients.

complementary testing to aPTT, to dose UFH infusion more accurately for a selected group of patients in whom aPTT measurement is unreliable, such as ICU or septic patients. Further prospective studies are required to look at the clinical outcomes of using an anti-Xa assay to monitor UFH activity.

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Congenital dyserythropoietic anaemia: an unexpected diagnosis in an adult referred with elevated serum ferritin

Sir,

A common reason for referral to a haematology clinic is an elevated serum ferritin. Although some patients will have hereditary haemochromatosis (usually secondary to an *HFE* gene mutation), there are a number of other disorders that are uncovered on further investigation, including chronic liver disease and inflammatory conditions.[1] We present a case where being alert to a subtle abnormality in the complete blood count of an adult male led to a diagnosis of congenital dyserythropoietic anaemia (CDA),[2] with tissue iron overload. This case is also of particular interest because the patient appears to have a novel form of CDA.

At presentation, this Chinese male was 46 years of age. He was referred to our haematology clinic because on a routine health check he was found to have an elevated serum ferritin of 1245 μg/L, with increased transferrin saturation of 0.82. We noted that his haemoglobin level was borderline and initial investigations showed haemoglobin 125 g/L, reticulocytes 117 × 10^9/L, bilirubin 35 μmol/L and haptoglobin <0.2 g/L. Examination of the blood film showed markedly abnormal red cell morphology with tear drop poikilocytes, membrane projections and occasional blister cells, but it was not spherocytic (Fig. 1). The features overall suggested a non-spherocytic haemolytic anaemia.

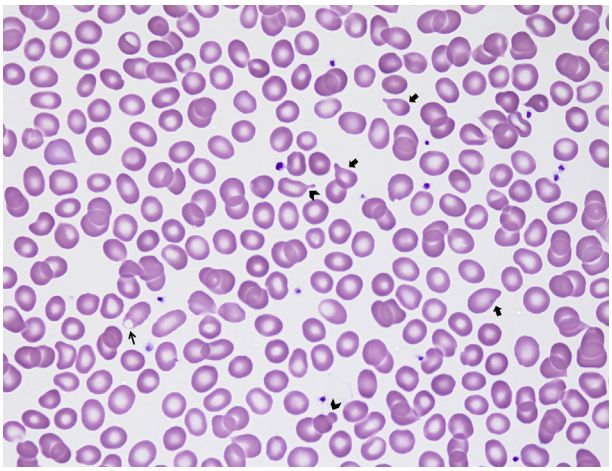

Fig. 1 Peripheral blood film with markedly abnormal red cell morphology including tear drop poikilocytes (block arrows), membrane projections (arrowheads) and blister cell (arrow).

His father, age 74 years, lives in China and was diagnosed with 'myelodysplasia' 4 years previously. A blood test result made available to us showed haemoglobin 101 g/L (with normal neutrophil and platelet counts) and ferritin 840 µg/L. His brother, age 44 years, who also lives in China had a blood test that showed haemoglobin 151 g/L and ferritin 503 µg/L. There was no comment on red cell morphology for either, and we have not had an opportunity to review their blood films. Our patient has a daughter, age 18 years, who had neonatal jaundice, and son, age 14 years. Both are in good health and their parents have not wanted them tested to date.

Other investigations, that showed no abnormality, included direct antiglobulin test, Heinz bodies, haemoglobinopathy screen, HAM's acidified serum lysis test, G6PD screen and HFE mutation analysis. Erythrocyte osmotic fragility testing showed no evidence of spherocytosis (fresh or incubated); there was a mild left shift (resistance) on the incubated test. EMA binding was normal and abdominal ultrasound showed

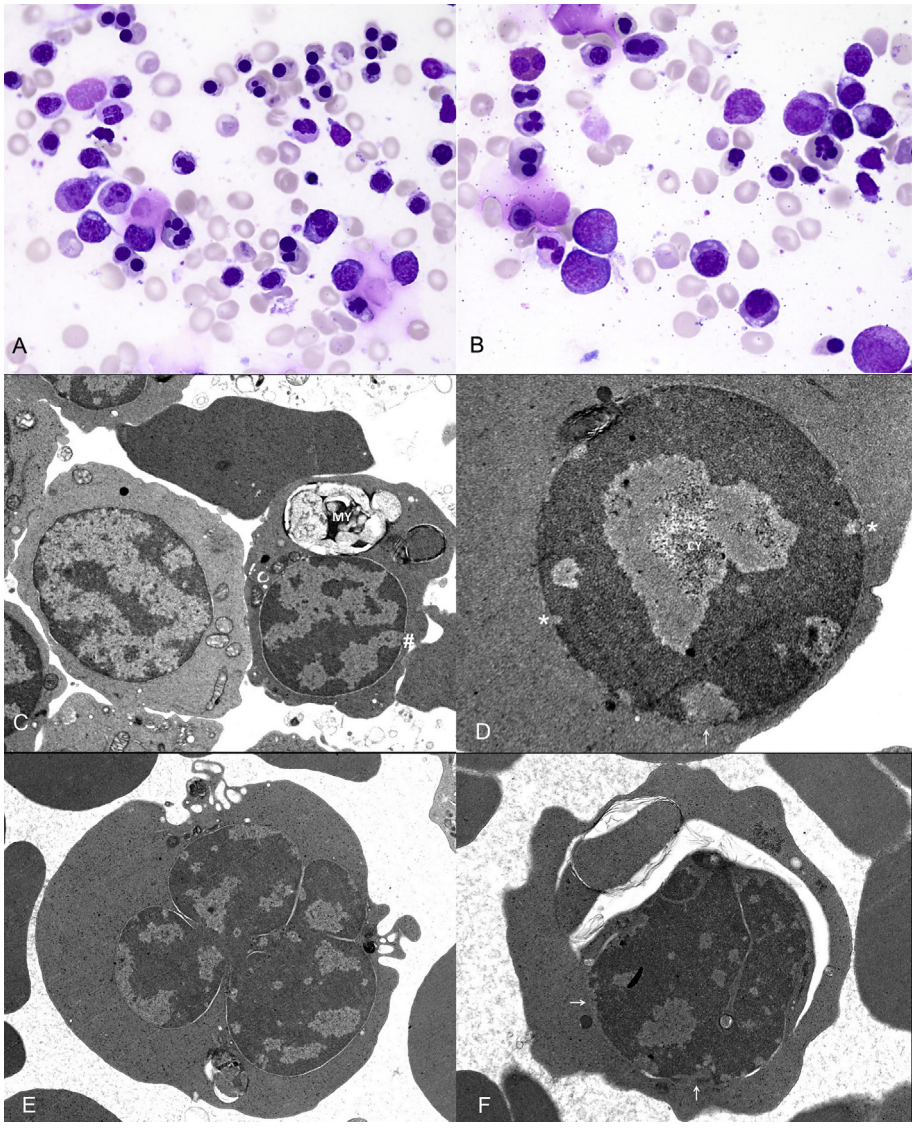

Fig. 2 (A,B) Bone marrow aspirate showing dyserythropoiesis with binucleate and multinucleate forms. (C–F) Bone marrow electron microscopy. (C) Normal early erythroid cell and later cell with widened nuclear pore (hash) and prominent myelin figure (MY). (D) Cytoplasm invaginating nucleus (asterisk), partial loss of nuclear membrane (arrow), cytoplasm between chromatin (CY). (E) Incomplete nuclear division and disorganised nuclear membrane. (F) Fissure surrounding nucleus and partial loss of nuclear membrane (arrow).