Anaemia in chronic heart failure: what is its frequency in the UK and its underlying causes?

N Cromie, C Lee, A D Struthers

RESULTS
Thirty nine of the 269 patients (14.4%) selected had a mean haemoglobin concentration of < 11 g/dl. Of these 39 patients, three were microcytic and eight were macrocytic. It is difficult to compare this figure with Silverberg's work since his cutoff was unrealistically high (haemoglobin concentration < 12 g/dl) which may be why 79% of his NYHA class IV patients were quoted as being anaemic.

Of the group classified as having a microcytic anaemia (MCV < 76 fl) ferritin was low in only one of three patients (table 2). However, this group is too small for any detailed analysis of the precise cause of the anaemia.

In the patients with a normocytic anaemia (MCV 76–96 fl), 43% of them had evidence of renal impairment (creatinine ≥ 160 µmol/l) and 40% had evidence of possible hypothyroidism (TSH > 4 mu/l), but no patient had evidence of any deficiency of any haematinic (iron, B12, folate) (table 2).

Eight patients (21%) were found to have a macrocytic anaemia, half of whom had evidence of renal impairment with

Abbreviations: CHF, chronic heart failure; MCV, mean cell volume; NYHA, New York Heart Association; SOLVD, studies of left ventricular dysfunction; TSH, thyroid stimulating hormone
one isolated case of possible hypothyroidism. Interestingly, as in the normocytic group, no patient had evidence of any haematinic deficiency (iron, B12, folate).

Looking at the group as a whole, renal impairment was present in 44% and possible hypothyroidism was present in one third of those who were assessed for hypothyroidism. However, despite 40% of them having either a microcytic or macrocytic anaemia, a specific haematinic deficiency (iron, B12, folate) was virtually absent.

**DISCUSSION**

We found that anaemia in CHF is common, occurring in 14.4% of our hospitalised population. The studies by Silverberg and colleagues suggest that anaemia can aggravate the symptoms of heart failure, and that either active or prophylactic treatment of anaemia will improve symptoms and signs and decrease the need for hospitalisation. Therefore, a case can now be made for treating anaemia in CHF whenever it is found. This begs the question: what is the correct way of treating anaemia in each case of CHF?

Before this study, we had anticipated that the cause of the anaemia in CHF would be heterogeneous and that erythropoietin would only be appropriate for a minority who turned out to have a true anaemia of chronic disorder, whether it be caused by renal impairment or not. However, what is striking from our results is that a haematinic deficiency of any kind was exceedingly rare. Possible hypothyroidism was found in a small group, but it is difficult to know whether this was the cause of the anaemia or not in any case. Most of the anaemia seen in CHF does appear to be an anaemia of chronic disorder, some of which could be caused by renal dysfunction and some by an unknown cause. In either case, the erythropoietin regimen as proposed by Silverberg and colleagues might be the only therapeutic option available in the majority of anemic CHF patients. Further work is therefore required to confirm or refute the benefits of erythropoietin treatment reported by Silverberg and colleagues.

---

**REFERENCES**

