The prevalence and natural history of anaemia in an optimally treated heart failure population

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Abstract

The prevalence of anaemia in heart failure (HF) is becoming better recognised, yet little is known about its natural history in a HF population.

We examined the records of 200 consecutive patients who were admitted to our service with New York Heart Association (NYHA) class IV HF, survived and were followed for six months following discharge. Complete records were available on 120 patients. Anaemia was defined as a haemoglobin concentration of < 13 g/dL in males and < 12 g/dL in females. Forty-one patients (34%) were found to have anaemia of unknown cause on admission. At follow-up (mean time 6.1±0.3 months), 28 patients were persistently anaemic. The haemoglobin concentration in the remaining 13 had returned to normal. A further group of 11 patients had become anaemic during the six-month follow-up period. All patients had been treated with maximally tolerated medical therapy. Anaemia was found to be equally prevalent in patients with preserved systolic function HF. Factors found to be independently associated with lower haemoglobin at follow-up were female sex, a history of gastrointestinal disease, inflammatory disease and a low glomerular filtration rate (GFR). Haemoglobin concentration at follow-up was found, on univariate analysis, to be associated with an increased risk of a HF-related admission during the follow-up period and increased severity of HF symptoms. On multivariate analysis, haemoglobin concentration at follow-up was found to be an independent predictor of NYHA class III–IV symptoms. In conclusion, anaemia is prevalent in a population admitted with class IV failure. While the haemoglobin concentration had normalised in a significant number of patients during follow-up, the presence of anaemia six months after discharge was associated with having a HF-related readmission and independently associated with moderate-to-severe HF symptoms.

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Introduction

A number of recent reports have focused on the subject of anaemia in chronic heart failure (HF). Several studies from the 1990s showed that the mean haemoglobin in patients with congestive HF is about 12 g/dL.[1-3] The initial paper to focus on anaemia specifically in HF patients demonstrated a prevalence of anaemia (defined as Hb < 12 g/dL) of 55.6% in a population attending a HF clinic, though this was in a select group of patients attending a nephro-cardiology clinic.[4] Several subsequent studies have reported the prevalence of anaemia (with varying cut-off values for haemoglobin level) in HF patients as between 14% and 50%.[5-10] Factors which were associated with the presence of anaemia in these studies included symptomatic severity of HF,[4,6,7] impairment of renal function[4,6-10] and other co-morbid conditions.[6,8-10] Furthermore, anaemia has been identified as an independent predictor of mortality,[6,8,9] and a predictor of hospital readmission[8] in HF patients.

The cause of anaemia in most cases is not easily identified. Cardiac failure is associated with renal impairment,[11,12] which may be an important factor in the development of anaemia. A low cardiac output may also impair bone marrow function.[13] Right-sided HF can cause malabsorption, nutritional deficits and impaired metabolism.[14] The elderly make up the majority of patients with HF,[15] and the existence of co-morbidities in this population may be of relevance in the development of anaemia.

The effect of correction of anaemia on the heart has been most widely studied in patients with chronic renal disease. In the setting of haemodialysis patients, the treatment of anaemia with erythropoietin has been shown to increase exercise capacity and to reduce exercise-induced cardiac ischaemia.[16] Structural benefits have also been observed, including a reduction of left ventricular hypertrophy, left ventricular end-diastolic and end-systolic diameters and an increase in left ventricular ejection fraction (LVEF).[17,18] A more recent study showed that the beneficial cardiovascular effects seen with correction of anaemia in dialysis patients do not persist on discontinuation of erythropoietin therapy.[19]

An initial non-randomised study using erythropoietin and intravenous iron to correct anaemia in HF patients whose treatment already conformed with best practice guidelines, demonstrated a significant improvement in New York Heart Association (NYHA) class, an increase in LVEF and a significant reduction in

hospitalisations.[4] A follow-up open-label randomised study by the same group provided similar results.[20] Furthermore, correction of anaemia with erythropoietin and iron in HF patients has also been shown to be associated with significant improvements in peak VO_2 and exercise duration.[21]

Many questions remain unanswered in this evolving area. The prevalence of anaemia in an unselected community HF population, including those with preserved systolic function, requires further definition. To date, studies have looked at anaemia in HF patients based on haemoglobin concentrations assessed at one time point only. The natural history of anaemia in patients with HF over time and in response to maximally tolerated angiotensin-converting enzyme (ACE) inhibitor and beta blocker therapy is also unknown. The aim of our study was to investigate these issues in the population attending our HF service.

Methods

We retrospectively examined the medical records of the first 200 consecutive patients attending our HF service. All patients had been admitted to hospital with NYHA class IV HF between November 1998 and July 2001. The diagnosis was confirmed by a senior member of the cardiology staff, based on a compatible history, physical signs consistent with the diagnosis, a chest radiograph demonstrating pulmonary venous congestion and response to treatment. Therapy for these patients was instituted in line with current consensus guidelines. All patients attained clinical stability (weight stable, on oral medications at a stable dose for 48 hours) prior to discharge. All were followed at the HF clinic at three and six months following discharge. None of the patients had a blood transfusion while in hospital or during the follow-up period. None underwent cardiac transplantation or major cardiac surgery during follow-up.

Haemoglobin levels had been obtained on admission, prior to discharge (in the main within one to three days of discharge) and at a follow-up out-patient visit (mean time 6.1±0.3 months). Anaemia was defined as a haemoglobin concentration below 13 g/dL in men and 12 g/dL in women.[22] This was calculated using a Sysmex XE-2100 automated haematology analyser, which has a variation coefficient of 1.0% or less. We retrospectively reviewed patients' charts for information on serum level of iron, vitamin B12, folate and history of concomitant illness, in particular gastrointestinal bleeding and chronic systemic illness. Complete records were available for 120 patients.

Renal impairment was defined as a creatinine concentration of greater than 150 µmol/L, as this level has been associated with an increase in neurohormone levels and a reduction in long-term survival in patients with HF.[23] We also calculated the glomerular filtration rate (GFR) using the Cockroft-Gault formula ((140-age (years)) x weight (kg) x K)/serum creatinine where K=1.23 for men and K=1.05 for women)[24] to allow a more precise estimate of renal function.

Statistical methods

SPSS (Statistical Package for the Social Sciences) Version 11 statistical software was used to analyse the data. Statistical models employed included t-test for continuous variables and chi-squared analysis for discrete variables (two sided, α=0.05). The primary end points chosen were: the presence of a lower haemoglobin at six-month follow-up visit, HF-related readmission within six-months of the index discharge and severity of HF symptoms at six-month follow-up were the primary end points.

As regards univariate and multivariate risk factor analysis, a multiple linear (for continuous variables) and logistic (for discrete variables) regression model for all 120 individuals included variables with univariate p-values of $\leq$ 0.25. The inclusion of irrelevant variables can result in a poor model fit and bias parameter estimates, thus the likelihood ratio test was used to identify independent variables with low explanatory power. In addition, goodness-of-fit indices (model chi square, Hosmer-Lemeshow) were compared to assess the fit of each specification of the multiple regression model.

Table 1. Comparison of baseline characteristics between the study population and those not included in the analysis

	Study population	Not included in analysis	p value
Patients (n)	120	80	N/A
Male	75 (62.5%)	54 (67.5%)	0.469
Mean EF (±SD)	36±11.5%	39±15.4%	0.244
LVSD	92 (76%)	61 (76%)	0.946
Age (±SD)	69.78±12.3	71.3±9.9	0.34
Aetiology			
Ischaemic	71 (59%)	54 (67.5%)	0.233
Dilated	15 (12.5%)	16 (20%)	0.216
Hypertensive	14 (11.6%)	6 (7.5%)	0.174
Valvular	20 (16.6%)	6 (7.5%)	0.059
Length of stay (days)	13.07±6.8	16.1±6.76	0.278
Medications			
ACEI≥CTD	92 (77%)	63 (78%)	0.73
ARB	11 (9%)	9 (11%)	0.63
Beta blocker	72 (60%)	36 (45%)	0.037
Frusemide > 40 mg daily	59 (49%)	35 (43%)	0.452
Digoxin	71 (59%)	51 (64%)	0.515
Nitrate	59 (49%)	37 (46%)	0.686
Aspirin	66 (55%)	51 (64%)	0.219
Warfarin	30 (25%)	21 (26%)	0.843

Key: EF = ejection fraction; SD = standard deviation; LVSD = left ventricular dysfunction; ACEI = angiotensin-converting enzyme inhibitor; CTD = clinical trial dose; ARB = angiotensin II receptor blocker

Results

Of the first 200 patients attending our HF unit, 80 patients had either died (38%), been lost to follow-up (21%) before six-month follow-up clinic visit or did not have their haemoglobin concentration measured (41%). The only statistically significant

difference in baseline characteristics and medications between the patients who were and were not included in this study was beta blocker use (60% in the study population and 45% in those not included in the study analysis, p=0.037) (table 1).

One hundred and twenty patients are included in this retrospective review. Forty-one (34%) had anaemia of undetermined cause on admission to hospital. At a mean follow-up of 6.1±0.3 months, 28 patients were persistently anaemic (64% male). The haemoglobin level of 13 patients (77% male) who were initially anaemic on admission had returned to normal (54% had a change in haemoglobin concentration of > 1 g/dL). A further group of 11 patients (73% male) had become anaemic since initial hospital discharge (82% had a change in haemoglobin concentration of > 1 g/dL). A group of 68 patients had normal haemoglobin levels throughout the observation period. The frequency of anaemia in the total population was 43%. The prevalence of anaemia on admission and at follow-up was 34% and 32% respectively.

Baseline characteristics (table 2)

On admission, 41 patients had anaemia of unknown cause (group A) and 79 patients had normal haemoglobin concentrations (group B). Patients in group A had a significantly lower mean ejection fraction (although there was no significant difference in numbers of patients with LV systolic dysfunction, defined as a left ventricular ejection fraction < 45% between the groups), and were significantly more likely to have renal impairment and a past history of gastrointestinal (GI) disease than those whose haemoglobin concentration was normal.

A greater percentage of patients in group A had a history of an inflammatory disease, hypothyroidism and chronic obstructive airways disease (COAD), though the differences between the groups for these conditions did not reach statistical significance. Patients in group A were significantly less likely to tolerate beta blocker therapy by the time of follow-up. Patients in group A had a higher mean age than those in group B, a difference which approached statistical significance (p=0.08).

Biochemical and haematological data

There was a statistically significant difference in mean haemoglobin concentration between the groups at all time points. The GFR was lower in group A compared with group B both on admission and at follow-up, the difference showing a trend towards statistical significance at both time points. There was no other significant difference between the groups. The mean serum iron concentration was low in both groups, although (non-significantly) lower in group A patients. Serum ferritin was well within normal limits in all patients, a pattern consistent, in group A, with anaemia of chronic disease (table 3).[25,26]

There was a statistically significant increase in haemoglobin concentration in male group A patients over time following discharge. No significant change in haemoglobin concentration occurred among their female counterparts. No significant change in GFR over time occurred in this group (table 4).

There was no statistically significant change in haemoglobin concentration over time in patients in group B, although there was a decrease in mean haemoglobin concentration among female patients which showed a trend towards significance (13.50±0.89 to 13.10±1.02, p=0.06). There was a trend towards a significant increase in mean GFR over time in group B patients (table 5).

Table 2. Baseline characteristics of 120 heart failure (HF) patients according to anaemia status on admission

	Anaemic on admission (group A)	Normal Hb on admission (group B)	p value
Number (n)	41	79	
Age (years) (mean±SD)	72.51±13.36	68.37±11.54	0.08
Male gender (%)	26 (63%)	49 (62%)	0.881
EF (mean±SD)	28.22±19.15	35.34±11.99	0.034
LVSD (%)	30 (73%)	62 (78%)	0.514
LOS (mean±SD)	14.22±7.61	12.31±6.41	0.183
No. of blood draws	9.62±4.90	8.39±4.47	0.186
Aetiology			
Ischaemic (%)	23 (56%)	48 (61%)	0.622
Dilated (%)	5 (12%)	10 (12%)	0.942
Hypertensive (%)	7 (17%)	7 (9%)	0.184
Valvular (%)	6 (15%)	14 (18%)	0.667
Meds at follow-up			
ACEI≥CTD (%)**	32 (78%)	60 (75.9%)	0.493
ARB (%)	3 (7.3%)	9 (11.4%)	0.749
Beta blocker (%)***	19 (46.3%)	53 (67.1%)	0.028
Frusemide > 40 mg daily (%)	24 (58.5%)	35 (44.3%)	0.139
Digoxin (%)	26 (63.4%)	45 (57%)	0.495
Nitrate (%)	19 (46.3%)	40 (50.6%)	0.656
Aspirin (%)	21 (51.2%)	45 (57%)	0.549
Warfarin (%)	11 (26.8%)	19 (24.1%)	0.739
Co-morbidity			
Renal impairment	9 (22%)	7 (9%)	0.045
Inflammatory disorder*	5 (12%)	4 (5%)	0.271
Hx of GI disease*	5 (12%)	1 (1%)	0.017
Hx of hypothyroidism*	6 (15%)	0	0.448
COAD	9 (22%)	12 (15%)	0.355

* Fisher's exact test used due to low cell count (n≤4)
** 80% of patients with LVSD in group A and 81% of patients with LVSD in group B were on a trial dose of ACEI
*** 60% of patients with LVSD in group A and 74% of patients with LVSD in group B were on beta blocker therapy.
Key: Hb = haemoglobin; SD = standard deviation; EF = ejection fraction; LVSD = left ventricular systolic dysfunction; LOS = length of stay; Meds = medications; ARB = angiotensin II receptor blockers; Hx = history; GI = gastro-intestinal; COAD = chronic obstructive airways disease

Linear regression analyses

Factors which, on univariate linear regression analysis, were

Table 3. Biochemical and haematological data of 120 heart failure patients according to anaemia status

	Anaemic on admission (group A)	Normal Hb admission (group B)	p value
On admission (n)			
Hb (male) (g/dL) (75)	11.72±0.93	14.74±0.92	<0.000001
Hb (female) (g/dL) (45)	11.23±0.69	13.50±0.89	<0.000001
GFR (ml/min) (120)	51.05±21.72	58.11±22	0.102
Pre-discharge (n)			
Hb (male) (g/dL) (74)	12.07±1.23	14.25±3.6	0.004
Hb (female) (g/dL) (45)	11.53±0.99	13.49±1.10	0.000001
At six-month follow-up (n)			
Hb (male) (g/dL) (75)	12.14±1.57	14.42±1.53	0.004
Hb (female) (g/dL) (45)	11.21±1.31	13.10±1.02	0.000003
GFR (ml/min) (120)	52.39±25.99	60.49±26.28	0.111
TSH (0.3–3.8 mU/L) (45)	1.87±1.41	2.44±1.23	0.169
B12 (>150 ng/L) (32)	344.7±150	422.73±205	0.228
Serum folate (4–18 µmol/L) (32)	8.48±3.31	10.49±5.44	0.263
Serum iron (13–32 µmol/L) (23)	10.29±6.82	12.43±7.56	0.621
Ferritin (15–300 µg/L) (29)	165.55±153	146.96±91.5	0.797

Key: Hb = haemoglobin; GFR = glomerular filtration rate; TSH = thyroid stimulating hormone; B12 = vitamin B12

found to be significantly associated with a lower haemoglobin concentration at six-month follow-up were female gender, age, length of stay during index HF admission, NYHA class III–IV at follow-up, GFR at admission and follow-up, a history of hypothyroidism, a history of GI disease, and haemoglobin concentration on admission and pre-discharge. Factors which were identified in multivariate regression analysis (table 6) as independent predictors of lower haemoglobin at follow-up were female sex, a history of GI disease, an inflammatory condition and GFR at follow-up.

Logistic regression analyses

Univariate analysis identified several factors associated with an increased risk of having a HF-related admission following discharge from hospital, including haemoglobin concentration at follow-up. However, factors which were independently associated with an increased risk of readmission after multivariate analysis were NYHA class III–IV at follow-up, a higher dose of loop diuretic, and GFR on index admission to hospital (table 7).

Haemoglobin concentration at follow-up was found to be associated with an increased risk of NYHA class III–IV HF symptoms at follow-up on univariate analysis. It was also found to be independently associated with NYHA class III–IV HF symptoms at follow-up using multivariate analysis. Ejection fraction was the only other factor found to be independently associated with NYHA class III–IV HF symptoms at follow-up (table 8).

Table 4. Change in haemoglobin concentration and GFR over time in patients who were anaemic at time of admission (group A) (n=41)

	On admission	At six-month follow-up	p value
Hb (male) (g/dL)	11.71±0.93	12.14±1.57	0.036
Hb (female) (g/dL)	11.23±0.69	11.21±1.31	0.257
GFR (ml/min)	51.05±21.72	52.39±25.99	0.454

Key: Hb = haemoglobin; GFR = glomerular filtration rate

Table 5. Change in haemoglobin concentration and GFR over time in patients with normal haemoglobin at time of admission (group B) (n=79)

	On admission	At six-month follow-up	p value
Hb (male) (g/dL)	14.74±0.92	14.42±1.53	0.15
Hb (female) (g/dL)	13.50±0.89	13.10±1.02	0.06
GFR (ml/min)	58.11±22.6	60.49±26.28	0.064

Key: Hb = haemoglobin; GFR = glomerular filtration rate

Discussion

Our study provides further evidence of the high prevalence of anaemia in HF. This prevalence was less than that reported by some groups[4,5,8] and greater than that reported by other groups,[7,9,10] but the reported figure of 34% at admission and 32% at follow-up are consistent with those observed by Horwich *et al*.[6] who used the same haemoglobin cut-off to define anaemia.

Our study provides original observations on the natural history of anaemia in this population, with a significant number of patients who were initially anaemic (32% of group A) having normal haemoglobin concentrations at follow-up. Moreover, a similar number of patients, who have normal haemoglobin concentrations at discharge and whose treatment conformed with current guidelines, had developed anaemia at the time of follow-up (14% of group B).

Although the presence of anaemia changes over time in patients, its prevalence in our population remained stable at between 32–34%. Some of this change may be explained by laboratory variation in haemoglobin levels around the cut-off values used to define anaemia in this study. However, in group A, 49% of patients had a change in haemoglobin concentration of greater than 1 g/dL from admission to time of follow-up and 85% of patients had a change of greater than 0.5 g/dL. In group B, 42% of patients had a change in haemoglobin concentration from admission to time of follow-up of greater than 1 g/dL and 59% had a change of greater than 0.5 g/dL. These changes are of a magnitude which cannot be explained by laboratory variation. The proportion of patients in the two groups with preserved

Table 6. Multivariate linear regression analysis of risk of anaemia at six-month follow-up

Variable	β	t	p value	CI lower (95%)	CI upper (95%)
Gender (male=1)	0.229	2.912	0.004	0.273	1.435
History of GI disease	-0.247	-3.130	0.002	-3.337	-0.750
Inflammatory disease	-0.290	-3.715	<0.0001	-3.048	-0.928
GFR at follow-up	0.191	2.366	0.02	0.002	0.024

Key: GI = gastrointestinal; GFR = glomerular filtration rate; CI = confidence interval

Table 7. Multivariate logistic regression analysis of a HF-related readmission during follow-up

Variable	β	Wald	OR	p value	CI lower (95%)	CI higher (95%)
NYHA III–IV at follow-up	1.568	4.771	4.799	0.029	1.175	19.600
Frusemide > 40 mg daily	1.629	3.897	5.097	0.048	1.012	25.684
GFR at admission	-0.035	3.377	0.966	0.066*	0.930	1.002

*The likelihood ratio statistic on the introduction of the GFR on admission variable is significant (χ^2=4.187, df=1, p=0.041). This indicates that the introduction of this variable significantly improves the predictive power of the model, even though the Wald statistic is insignificant.

Key: NYHA = New York Heart Association; GFR = glomerular filtration rate; CI = confidence interval

Table 8. Multivariate logistic regression analysis of the risk of NYHA III–IV at six-month follow-up

Variable	β	Wald	OR	p value	CI lower (95%)	CI higher (95%)
Hb conc at follow-up	-0.243	4.549	0.784	0.033	0.627	0.980
EF	0.033	5.467	1.033	0.019	1.005	1.062

Key: Hb = haemoglobin; EF = ejection fraction

systolic function was similar (27% in group A and 22% in group B). Thus, anaemia may be as prevalent in HF with preserved systolic function as in reduced systolic function HF.

The cause of anaemia remains obscure in these patients. The pattern of anaemia in group A was consistent with that of the anaemia of chronic disease (low serum iron, normal or raised serum ferritin). It is not possible to rule out iron deficiency as a factor without examination of the bone marrow for iron.[25] This was carried out in only two patients. There are many reasons why HF patients may be iron-deficient, including malabsorption, nutritional deficits and their use of prophylactic aspirin. However, serum ferritin is directly related to reticuloendothelial iron stores and has been shown to be the most sensitive and specific parameter to predict bone marrow iron status.[27] At a cut-off point of 32 µg/L it has a sensitivity of 79.2% and specificity of 96.9% in the diagnosis of iron deficiency.[28] All our anaemic patients had serum ferritin levels above this cut-off point. However, since ferritin is an acute phase reactant it may not be a reliable measurement in HF patients or those with inflammatory disorders. The fact that patients who were initially anaemic (group A) were significantly more likely than patients with normal haemoglobin on admission (group B) to have both renal impairment and a past history of GI disease does not make the differentiation between iron deficiency and anaemia of chronic disease any easier.

Only two studies have looked at the extent of iron deficiency in anaemic HF patients. A retrospective hospital discharge database review of 12,065 patients with new-onset HF found a prevalence of anaemia of 17%. Of the anaemic patients, 21% had iron deficiency anaemia.[9] Of 188 patients screened in a prospective study, 10.6% were found to be anaemic (using a cut-off haemoglobin concentration of < 11.5 g/dL), all of whom had ferritin levels well within the normal range. Of these patients, 44% had some evidence of iron deficiency (low serum iron or transferrin saturation of ≤ 15%) and showed a significant increase in haemoglobin concentration in response to oral iron supplements alone over a period of three months.[29] Given the greater burden of illness and greater age in group A and taking into consideration the mean level of ferritin, anaemia of chronic disease is the most likely cause of anaemia in this population. However, iron deficiency may be an important additional contributory cause.

Gender appears to be important both in terms of level of haemoglobin concentration and in change of haemoglobin level over time. Female patients had lower mean haemoglobin concentrations at all time points and in both groups compared to mean male values. Female patients in group A showed no increase in haemoglobin concentration over time, whereas their male counterparts showed a significant increase in mean haemoglobin concentration at follow-up. Female patients in group B showed a trend towards a significant reduction in haemoglobin concentration over time, in spite of evidence-based medical therapy. We also found on multivariate analysis that female gender was a significant predictor of lower haemoglobin level at follow-up. This is consistent with findings from the ELITE 2 trial, which showed that low haemoglobin values are about twice as frequent in women as in men.[30]

NYHA class was found to be a predictor of anaemia on univariate analysis but was not found to be an independent predictor on multivariate analysis. Haemoglobin concentration at follow-up was found to be an independent predictor of severity of HF symptoms. These findings suggest a relationship between anaemia and severity of HF symptoms, consistent with other studies,[4,6,7,20] and supported by the demonstration of an independent relationship between MVO2 and haemoglobin in a study by Kalra *et al*.[31]

Indeed, HF may have at least a partially causative role in the development of the anaemia of chronic disease. One of the main theories advanced to explain anaemia of chronic disease is that inflammation causes a change in the dynamics of iron recirculation whereby iron is retained in the reticuloendothelial system rather than being released to the developing red blood cells in the marrow.[25] Pro-inflammatory cytokines (tumour necrosis factor [TNF], and interleukins [IL-1, and IL-6]) appear to cause hypoferraemia via the induction of ferritin synthesis and iron storage in macrophages and hepatocytes.[25] Cytokines have also been implicated in the impairment of erythroid progenitor cell proliferation in the marrow.[26] Thus, the anaemia of chronic disease may be a non-specific consequence of activation of the inflammatory cytokine network. That this may be relevant specifically to anaemia in HF is supported by the demonstration of elevated cytokine levels in patients with HF, especially in those with more limiting symptoms.[33-37]

In discussing the aetiology of anaemia in this group it must be mentioned that this is a specific subgroup of patients with HF, presenting with NYHA class IV symptoms and clinical signs of congestion. Subsequent fluid shifts could have influenced the analysis of haemoglobin.

That anaemia has been shown to be an independent predictor of mortality[6,8,9,38] and associated with an increased risk of rehospitalisation[8,39] underlines its importance in HF. Furthermore, the benefits seen with correction of anaemia in optimally treated HF patients by Silverberg *et al*.[4,20] and others[21] underline its clinical importance. Apart from symptomatic improvements, reduction in hospitalisations and need for diuretics, an improvement in LVEF was also noted. Similar studies in patients with renal failure show that correction of anaemia also has beneficial structural effects on the heart, including reduction in left ventricular end-systolic diameter.

Key messages

- Anaemia is prevalent in patients presenting with NYHA class IV heart failure
- Followed over time, the majority remain anaemic
- Chronic disease, in particular impaired renal function, is associated with anaemia
- Persistently low haemoglobin is associated with readmission and poor symptomatic status

In conclusion, our study confirms that anaemia is prevalent in patients who have HF with both preserved and reduced systolic function. Female gender and co-morbid conditions are independent predictors of anaemia at follow-up. Over time the presence of anaemia in patients fluctuates, yet its prevalence remains stable. The form of anaemia is predominantly that of chronic disease, but iron deficiency may play a significant contributory role. Treatment with erythropoietin may prove to be an effective additional tool in managing these patients.

Conflict of interest

None declared.

Editors' note

An editorial on low haemoglobin in heart failure can be found on pages 343–5.

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