*Iron Deficiency and Cardiac Failure*

A 75 year old male for a Right Hemicolectomy has a history of congestive cardiac failure due to cardiomyopathy (alcohol), AF, OSA. Echo:- EF 46%, dilated left ventricle moderate mitral regurgitation. Haemoglobin currently 86 due to bowel cancer and dabigatran.
Question:- What should be the management; What preop and intraop Hb targets?
Discussion: - Stop the dabigatran – the problems of ongoing blood loss outweighs risk of embolism. Check iron stores (Ferritin) but it is almost certain that the patient is short of iron. Iron infusion should improve anaemia. In 1 week the haemoglobin would be expected to rise by more than 10grams per litre so postponing for 1 to 2 extra weeks may be worthwhile. *What should be the target haemoglobin at time of surgery?* There were differing opinions. Studies such as TRICC suggest that a transfusion threshold of 70 is appropriate but given the history of CCF a higher threshold would be supported by some. There is probably no role for EPO in preparation of this patient as the anaemia is not due to bone marrow failure.

Iron infusion should improve Hb, but may also improve heart failure directly. This was originally suggested in the IRON-HF study in 2013 (Beck-da-Silva et al), with findings replicated in other studies, but is an active area of research and controversy. There is general consensus that iron therapy in iron deficiency is valuable in improving cardiac failure with evidence of reduced hospitalisation and improved exercise capacity, independent of benefit for improving anaemia. In the absence of iron deficiency, there is no clear evidence of benefit on heart failure directly.

**SOME REFERENCES**


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*ABSTRACT: Anaemia and iron deficiency (ID) are important co-morbidities in patients with chronic heart failure (HF) and both may lead to reduced exercise capacity. METHODS: We enrolled 331 out-patients with stable chronic HF (mean age: 64 +/- 11 years, 17% female, left ventricular ejection fraction [LVEF] 35 +/- 13%, body mass index [BMI] 28.5 +/- 5.2 kg/m(2), New York Heart Association [NYHA] class 2.2 +/- 0.7, chronic kidney disease 35%, glomerular filtration rate 61.7 +/- 20.1 mL/min). Anaemia was defined according to World Health Organization criteria (haemoglobin [Hb] < 13 g/dL in men, < 12 g/dL in women). ID was defined as serum ferritin < 100 mug/L or ferritin < 300 mug/L with transferrin saturation (TSAT) < 20%. Exercise capacity was assessed as peak oxygen consumption (peak VO2) by spiroergometry and 6-minute walk test (6MWT). RESULTS: A total of 91 (27%) patients died from any cause during a mean follow-up of 18 months. At baseline, 98 (30%) patients presented with anaemia and 149 (45%) patients presented with ID. We observed a significant reduction in exercise capacity in parallel to decreasing Hb levels (r = 0.24, p < 0.001). In patients with anaemia and ID (n = 63, 19%), exercise capacity was significantly lower than in patients with ID or anaemia only. Cox regression analysis showed that after adjusting for NYHA, age, hsCRP and creatinine anaemia is an independent predictor of mortality in patients with HF (hazard ratio [HR]: 0.56, 95% confidence interval [CI]: 0.33-0.97, p = 0.04). CONCLUSION: The impact of anaemia on reduced exercise capacity and on mortality is stronger than that of ID. Anaemia remained an independent predictor of death after adjusting for clinically relevant variables.*

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AB Iron deficiency (ID) has been identified as an important co-morbidity in patients with heart failure (HF). Intravenous iron therapy reduced symptoms and rehospitalizations of iron-deficient patients with HF in randomized trials. The present multicenter study investigated the "real-world" management of iron status in patients with HF. Consecutive patients with HF and ejection fraction <=40% were recruited and analyzed from December 2010 to October 2015 by 11 centers in Germany and Switzerland. Of 1,484 patients with HF, iron status was determined in only 923 patients (62.2%), despite participation of the centers in a registry focusing on ID and despite guideline recommendation to determine iron status. In patients with determined iron status, a prevalence of 54.7% (505 patients) for ID was observed. Iron therapy was performed in only 8.5% of the iron-deficient patients with HF; 2.6% were treated with intravenous iron therapy. The patients with iron therapy were characterized by a high rate of symptomatic HF and anemia. In conclusion, despite strong evidence of beneficial effects of iron therapy on symptoms and rehospitalizations,

diagnostic and therapeutic efforts on ID in HF are low in the actual clinical practice, and the awareness to diagnose and treat ID in HF should be strongly enforced.

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**Abstract** Background: Iron deficiency is common in patients with heart failure (HF) and is associated with reduced exercise capacity and poor outcomes. Whether correction of iron deficiency with (intravenous) ferric carboxymaltose (FCM) affects peak oxygen consumption [peak $VO_2$], an objective measure of exercise intolerance in HF, has not been examined. Methods: We studied patients with systolic HF (left ventricular ejection fraction <=45%) and mild to moderate symptoms despite optimal HF medication. Patients were randomized 1:1 to treatment with FCM for 24 weeks or standard of care. The primary end point was the change in peak $VO_2$ from baseline to 24 weeks. Secondary end points included the effect on hematinic and cardiac biomarkers, quality of life, and safety. For the primary analysis, patients who died had a value of 0 imputed for 24-week peak $VO_2$. Additional sensitivity analyses were performed to determine the impact of imputation of missing peak $VO_2$ data. Results: A total of 172 patients with HF were studied and received FCM (n=86) or standard of care (control group, n=86). At baseline, the groups were well matched; mean age was 64 years, 75% were male, mean left ventricular ejection fraction was 32%, and peak $VO_2$ was 13.5 mL/min/kg. FCM significantly increased serum ferritin and transferrin saturation. At 24 weeks, peak $VO_2$ had decreased in the control group (least square means -1.19±0.389 mL/min/kg) but was maintained on FCM (-0.16±0.387 mL/min/kg; P=0.020 between groups). In a sensitivity analysis, in which missing data were not imputed, peak $VO_2$ at 24 weeks decreased by -0.63±0.375 mL/min/kg in the control group and by -0.16±0.373 mL/min/kg in the FCM group; P=0.23 between groups). Patients' global assessment and functional class as assessed by the New York Heart Association improved on FCM versus standard of care. Conclusions: Treatment with intravenous FCM in patients with HF and iron deficiency improves iron stores. Although a favorable effect on peak $VO_2$ was observed on FCM, compared with standard of care in the primary analysis, this effect was highly sensitive to the imputation strategy for peak $VO_2$ among patients who died. Whether FCM is associated with an improved outcome in these high-risk patients needs further study.