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Internal Medicine: Open Access

Opinion Article

The Impact and Severity of Iron Deficiency in Cardiac Diseases

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DESCRIPTION

All organ systems require iron for a variety of metabolic functions, including erythropoiesis, mitochondrial activity, oxygen transport, the metabolism of skeletal and cardiac muscle, the immunological and neurological systems, the inflammatory response, lipid metabolism, and many more. Due to the high prevalence of Iron Deficiency (ID), as well as the results of recent trials, patients with Heart Failure (HF) and decreased Ejection Fraction (HFrEF) should consider iron as a major treatment target. Nonetheless, ID seems to be prevalent in a wide variety of Cardio Vascular (CV) illnesses.

ID can be defined as reduced circulating iron, which can be linked to a persistent inflammatory state as in many CV diseases, or depleted iron stores linked to a decrease in the total body iron supply due to inadequate nutritional iron intake, impaired absorption, or chronic blood loss. Hepcidin, a type II acute phase protein produced in the liver and a key regulator of iron homeostasis, is released in greater amounts in response to inflammation. Hepcidin controls the breakdown of ferroportin, a transmembrane protein that mediates the release of recycled iron from the spleen and liver's macrophages and transfers iron taken up by food from the interior of the mucosal cells in the small intestine to the bloodstream.

Iron deficiency and anemia are significant and frequent comorbidities that frequently coexist in heart failure patients. Together or separately, these disorders are linked to lower outcomes and a poor clinical state. It is unclear if anemia and iron deficiency are only indicators of the severity of heart failure or if they actually influence the course and consequences of heart failure and should be addressed. Over the past few years, erythropoiesis-stimulating drugs have been extensively studied in the treatment of anemia in patients with heart failure.

Regretfully, these drugs increased the chance of side effects rather than improving results. Patients with heart failure may have a functional iron shortfall, which occurs when total body iron is normal or elevated but insufficient to meet the needs of target tissues due to sequestration in the storage pool, or an absolute iron deficiency, where total body iron is reduced.

Although patients with anemia due to absolute iron shortage should get iron replacement therapy, it is unclear if and how non anemic people with heart failure should be treated for absolute or functional iron deficit.

Small trials have recently shown that intravenous iron delivery improves symptoms and exercise capacity in individuals with heart failure and absolute or functional iron deficiency, with or without anemia. However, long-term results and safety data are not yet available. In this review, we go over the pathophysiology, causes, and available treatments for anemia and iron deficiency in heart failure patients.

About 30% of heart failure patients have iron shortage, which is typically categorized as chronic normocytic anemia. About one-third of patients have functional iron insufficiency. The presence of anemia is thought to be a poor prognostic factor for heart failure patients. To improve the adverse progression of organ failure or the decompensating of cardiac function, this factor can be adjusted. Numerous studies have reported varying estimates of the prevalence of the relationship between anemia and heart failure, ranging from 4% to 61%. Anemia is typically linked to a lower chance of survival and increases hospitalization expenses by reducing the ability of affected patients to exercise.

It is not advised to give oral iron supplements to patients with CHF due to the dearth of research supporting their effectiveness, the likelihood of negative gastrointestinal side effects, and the length of time required for treatment to raise hemoglobin levels.

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Received: 06-Nov-2023, Manuscript No. IME-23-29027; **Editor assigned:** 09-Nov-2023, PreQC No. IME-23-29027 (PQ); **Reviewed:** 22-Nov-2023, QC No. IME-23-29027; **Revised:** 30-Nov-2023, Manuscript No. IME-23-29027 (R); **Published:** 08-Dec-2023, DOI: 10.35248/2165-8048.23.13.441

Citation: Chanceller R (2023) The Impact and Severity of Iron Deficiency in Cardiac Diseases. Intern Med. 13:441.