**IRON-OVERLOAD CARDIOMYOPATHY: PATHOPHYSIOLOGY, DIAGNOSIS AND TREATMENT**

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Background: The prevalence of primary (hereditary) hemochromatosis and secondary iron-overload (hemosiderosis) is reaching epidemic levels worldwide. Iron-overload leads to excessive iron deposition in a wide variety of tissues, including the heart and endocrine tissues.

Methods and Results: Iron-overload cardiomyopathy is the primary determinant of survival in patients with secondary iron overload while being a leading cause of morbidity and mortality in patients with primary hemochromatosis. Iron-induced cardiovascular injury also occurs in acute iron toxicosis (iron poisoning), myocardial ischemia-reperfusion injury, cardiomyopathy associated with Freiderich ataxia, and vascular dysfunction. The mainstay therapies for iron-overload associated with primary hemochromatosis and secondary iron-overload is phlebotomy and iron chelation therapy, respectively. L-type Ca2+ channels provide a high capacity pathway for ferrous (Fe2+) uptake into cardiomyocytes in iron-overload conditions and calcium channel blockers may represent a new therapeutic tool to reduce the toxic effects of excess iron. Conclusions: Iron-overload cardiomyopathy is a an important and potentially reversible cause of heart failure at an international scale and involves diastolic dysfunction, increased susceptibility to arrhythmias and a late-stage dilated cardiomyopathy. The early diagnosis of iron-overload cardiomyopathy is critical since the cardiac dysfunction is reversible if effective therapy is introduced before the onset of overt heart failure.