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Effects of intravenous iron in chronic kidney disease and heart failure

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The heart is subject to a number of adaptive and subsequently maladaptive changes in patients with chronic kidney disease (CKD). There are structural changes with both concentric and eccentric hypertrophy in part linked to FGF-23; changes in energetic with a switch from fatty acids to glucose metabolism; ischemic vulnerability from both iron and erythropoietin deficiency; oxidative stress and changes in calcium cycling within the mitochondria. However, the key components in the process are the adjustments in mitochondrial function which serve as the powerhouse for all tissues. Iron plays a pivotal role in oxygen uptake, transport, storage and metabolism in both skeletal and cardiac muscle. In models of stressed hearts with CKD, there is an increase in stage 4 respiration in addition to an increase in uncoupling proteins leading to mitochondrial dysfunction and an increase in transition pore formation leading to impaired contractile function of cardiomyocytes. Therefore a deficiency of iron may lead to impaired mitochondrial function via effects on transition pore opening and subsequent inhibition of the pro-survival pathway and activation of apoptotic pathways. Also chronic iron deficiency may cause structural abnormalities in cardiac myocytes leading to reduced exercise capacity and performance. However, both *in vitro* and *in vivo* data suggest that there may be an increase in oxidative stress with intravenous iron administration whilst clinical data suggest improved functional capacity. Few studies have examined the effects of intravenous iron in patients with heart failure, anemia, iron deficiency and renal dysfunction and none have examined whether a single large dose of iron may lead to improvements in functional capacity without adverse effects. Therefore we have been examining the effect of iron and whether it leads to improvement in mitochondrial function and hence symptom improvement and cardiac function independent of hemoglobin in patients with CKD. In addition we are assessing if intravenous iron therapy affect markers of kidney injury and oxidative stress via generation of labile or catalytic iron. The results of these studies will provide an insight into the mechanisms underlying the conundrum with the benefits of iron related to incorporation into the iron sulphur clusters as part of the electron transport chain and TCA cycle, the changes in mitochondrial function and the potential iron related increase in oxidative stress and subsequent adverse effects.

Biography

Ahmed Zeidan is currently pursuing Doctorate in Renal Medicine under Prof. Bhandari's supervision at Hull York Medical School. He has obtained BSc (Hons) in Biomedical Science from University of Newcastle upon Tyne and Medical degree from University of Sheffield, UK. He has been practicing clinical medicine for five years and is involved in research at a national level.

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