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# 2039 T2* CMR in hereditary hemochromatosis

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## Background

Hemochromatosis (HH) is a disease where the intestinal absorption of iron is high and multi-organ iron overload may result with organ failure. Dilated cardiomyopathy in this condition is considered a consequence of cardiac siderosis, but there is contradictory evidence for this. Cardiovascular Magnetic Resonance (CMR) detects iron deposition in the liver and in the heart with the T2* technique. Several studies have confirmed the normal range of iron in the liver and heart for normal subjects and the abnormal value above which heart failure is a common risk. T2* is also useful to follow the chelation treatment of these patients and recent medical trials have confirmed the utility of deferiprone and deferoxamine to reduce the heart iron overload and increase the left ventricle systolic function in patients with thalassaemia. However, while the direct link between cardiac siderosis and dilated cardiomyopathy is confirmed in thalassaemia, in HH the correlation between heart iron overload and dilated cardiomyopathy requires further investigation. In this study, we hypothesized the existence of a direct relationship between myocardial iron deposition and dilated cardiomyopathy in HH.

## Methods

We retrospectively analyzed 1016 consecutive patients presenting for the evaluation of myocardial and liver iron overload from 1996 to 2006. All these patients had a confirmed diagnosis and were followed by specialized centers across the UK. Of this cohort, 53 patients had HH. Patients underwent cardiovascular magnetic resonance at 1.5 T (Siemens Sonata or Avanto, Erlangen, Germany). Each scan included the measurement of liver and heart T2*, and left and right ventricular function, volumes and mass using standard techniques with analysis using CMR-tools (Cardiovascular Imaging Solutions, London).

## Results

The CMR characteristic (LV volumes, T2* of liver and heart) of the patients are shown in Table 1. The age of the patients varied from 36 to 76 years. All patients were on medical treatment appropriate to the presenting symptoms from their HH.

Figure 1 shows the scatterplot of myocardial T2* against LV EF. Six patients (11%) had myocardial iron loading, and of these, 3 had (50%) significant LV dysfunction (EF < 56%). A further 5 patients had impaired LV function with normal myocardial T2* values, and of these 4 (80%)

**Table 1:**

| | LVEDV | LVESV | LVEF | LV mass | Liver T2* | Cardiac T2* |
|---|---|---|---|---|---|---|
| Mean 95% CI | 152 mL (138, 166) | 58.5 mL (44.7, 72.2) | 65.0% (60.7, 69.2) | 151 g (138, 165) | 11.3 ms (8.3, 4.2) | 30.0 ms (26.6, 33.6) |

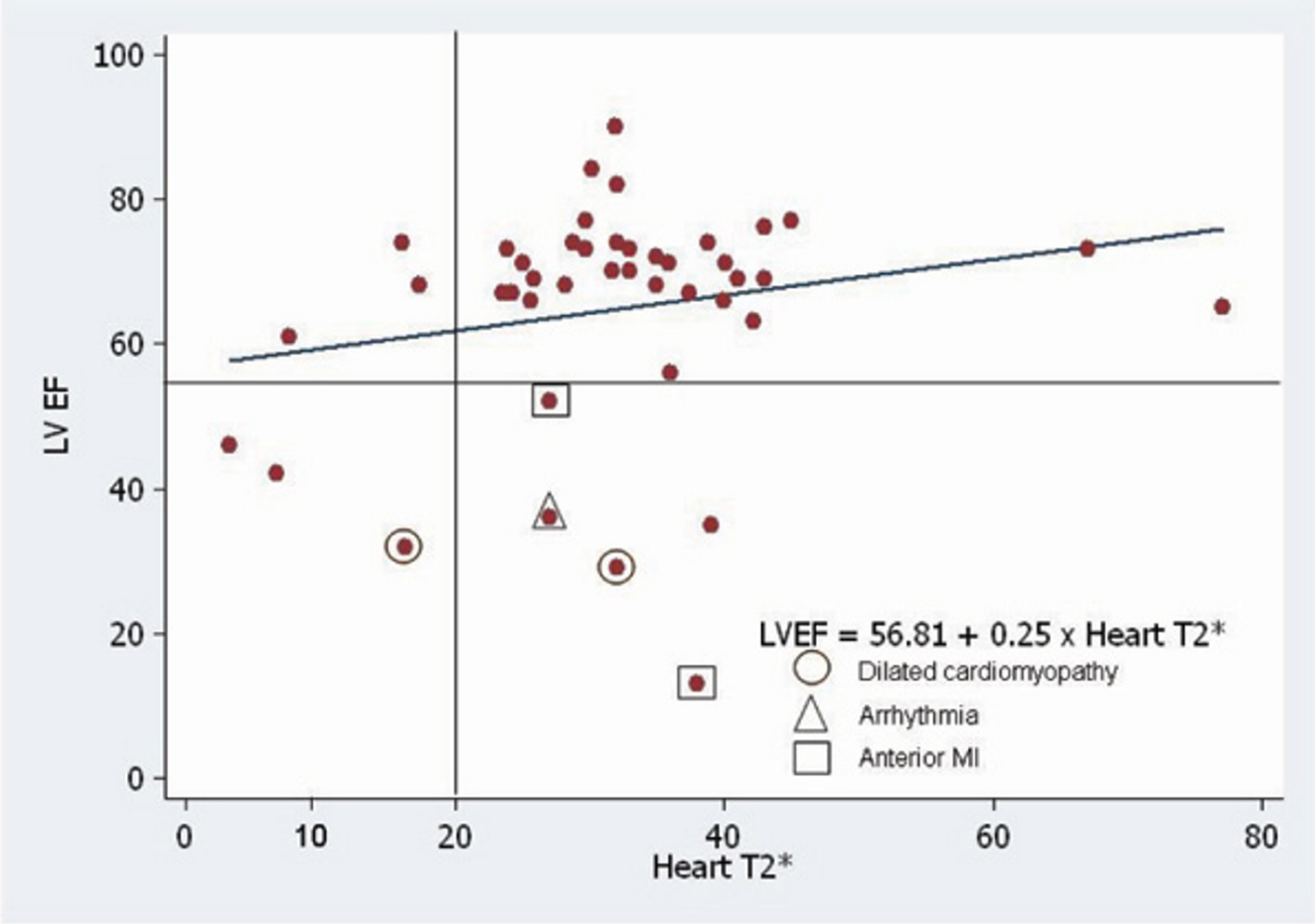


**Figure 1**

had an alternative explanation for the dysfunction (2 patients with previous anterior infarction, 1 with previously diagnosed idiopathic dilated cardiomyopathy, and 1 with arrhythmia).

## Conclusion

These findings suggest that in patients with HH, cardiac siderosis is a relatively uncommon complication (11%), which directly results in impaired LV function cardiomyopathy in half of these cases (6%).