

**2009 Annual Meeting of the  
American Society of Hematology**

**Highlights Report**

**Vitamin C Status in Transfusionally Iron-Overloaded  
Patients On Long-Term Deferasirox and Its  
Relationship to Myocardial Iron Removal**

***Maciej W Garbowski, Margarete Fabre, Patricia  
Evans and John B Porter***

***Abstract 2005***

**Vitamin C Status in Transfusionally Iron-Overloaded Patients On Long-Term Deferasirox and Its Relationship to Myocardial Iron Removal (Abstract # 2005)**

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Iron overloaded patients often become deficient in vitamin C (ascorbic acid, AA) because of an increased rate of AA oxidation in the presence of iron, and vitamin C has been shown to increase DFO-induced urinary iron excretion by increasing the availability of chelatable iron.<sup>1,2</sup> During DFO therapy vitamin C administration to correct this deficiency has been shown to increase iron excretion by up to 30%.<sup>3</sup> Whilst these effects are less well established for the oral iron chelator deferasirox, it has previously been reported that fasting plasma ascorbate (FPA) levels are significantly lower in patients receiving deferasirox than normal controls, with deficiency present in 72% of those who do not receive AA supplementation.<sup>3</sup>

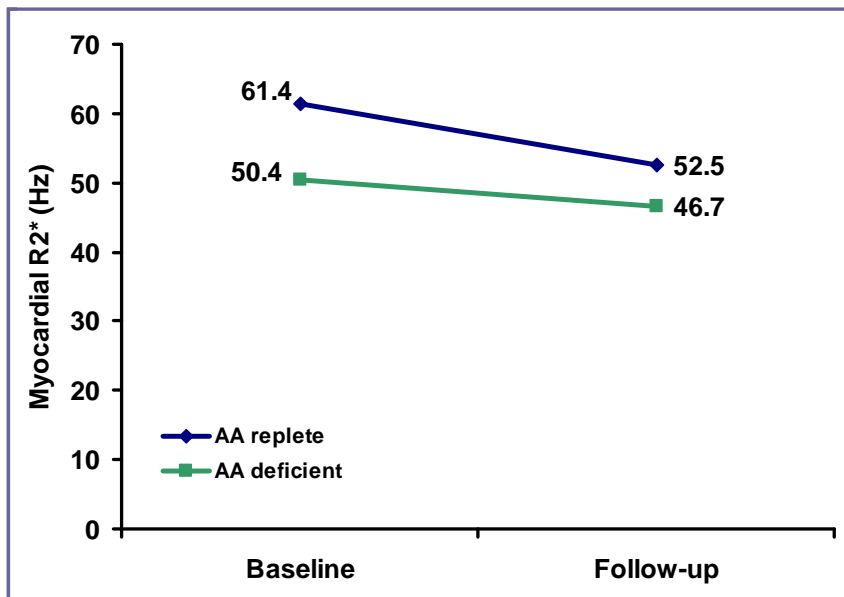
This study examined FPA levels and markers of iron overload in patients receiving deferasirox therapy over approximately 1 year to assess the impact of AA supplementation in this group. 10 patients with a variety of transfusion-dependent anemias received AA supplementation (2–3 mg/kg/day) and 11 did not. Patients who became AA replete over the course of study were compared with those who did not.

In AA supplemented patients, FPA increased significantly, correcting the deficiency in 3/5 scorbutic patients; an improvement was also seen in non-AA supplemented patients, which correlated with decreased LIC consistent with observations that iron overload speeds oxidation of AA.

In 13 AA-replete patients, myocardial R2\* improved significantly from 61.4 to 52.5 Hz ( $P=0.008$ , T2\* of 16.2 to 19.0 ms) and LIC also decreased from 10.2 to 7.0 mg Fe/g dw ( $P=0.02$ ). In patients who remained AA deficient a less-pronounced myocardial improvement from 50.4 to 46.7 was observed ( $P=ns$ , T2\* of 19.8 to 21.4 ms) (Figure 1) and LIC did not change significantly (6.6 to 4.8 mg Fe/g dw).

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**Figure 1. Changes in myocardial iron in AA-replete and AA-deficient patients**



These findings confirm previous studies indicating that AA deficiency is a significant risk in iron overloaded patients on long-term deferasirox therapy. Additionally, these data suggest that improved heart and liver iron is more likely in AA replete individuals than in those with low levels of AA. In the absence of randomized control trial data, FPA levels should be monitored and supplements considered for deficient transfusional iron overloaded patients receiving deferasirox.

**References**

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2. Porter JB. Practical management of iron overload. *Br J Haematol* 2001;115:239-252.
3. Sarantos K, Evans P, Garbowski M *et al*. Vitamin C deficiency in patients on long-term deferasirox without supplementation. *Blood* 2008;112(11):abst 1858.