INTRAVENOUS IRON ADMINISTRATION ENHANCES OXIDATIVE STRESS IN HEMODIALYZED PATIENTS

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INTRODUCTION AND OBJECTIVES

Oxidative stress (OS) is a non-traditional cardiovascular risk factor in patients affected by end-stage renal disease. Among the factors that contribute to the increased OS of this population, such as the comorbidities and the decline of the renal function, the HemoDialysis (HD) session itself plays a crucial role.[1]

During hemodialysis session, the intravenous administration of iron used to correct anemia plays a major role in increasing OS. Transferrin, indeed, is unable to saturate all the iron administered. Consequently free iron(III) oxide stimulates protein and lipid oxidation acting as a catalyst in free radical reactions.[2]

Despite this, intravenous iron administration is mostly used in HD, since oral iron has a poor intestinal absorption in HD patients.[3]

The aim of this study is to verify whether intravenous iron administration effectively influences OS in HD patients. Hence we measured two reliable biomarkers of OS, dityrosine and pentosidine, in HD patients who received or not intravenous iron.

METHODS

The plasmatic concentrations of dityrosine and pentosidine were determined with spectrofluorimetry after high performance liquid chromatography in 25 hemodialyzed patients (age 73 ± 10, dialysis vintage 6 ± 5 years) that have not taken iron supplements in the three previous months and 44 hemodialyzed patients (age 66 ± 13, dialysis vintage 5 ± 3 years) who have received intravenous sodium ferric gluconate for at least one of the three previous months. The levels of highly sensitive CRP were evaluated in order to consider the influence of inflammatory state on OS.

All patients were dialyzed with high-flux synthetic membranes and ultrapure dialysis fluid (< 0,1 CFU/mL). In our center, sodium ferric gluconate is administered once to thrice weekly according to monthly parameters of Hb and iron metabolism.

We excluded patients with clinically evident infectious processes.

RESULTS

In HD patients not receiving iron supplements the plasmatic levels of dityrosine and pentosidine were respectively 0,165 ± 0,032 AU and 0,085 ± 0,015 AU, while in HD patients receiving intravenous sodium ferric gluconate were respectively 0,195 ± 0,048 AU and 0,104 ± 0,030 AU, demonstrating a significantly higher concentration of both markers in patients taking iron supplements ($p < 0,01$). The significance of this difference is maintained also adjusting the statistical test for age, dialysis vintage and CRP levels. CRP plasmatic concentration was 0,78 ± 1,02 mg/dL in patients not receiving iron and 0,43 ± 0,42 mg/dL in patients receiving intravenous iron, resulting not significantly different between the two groups.

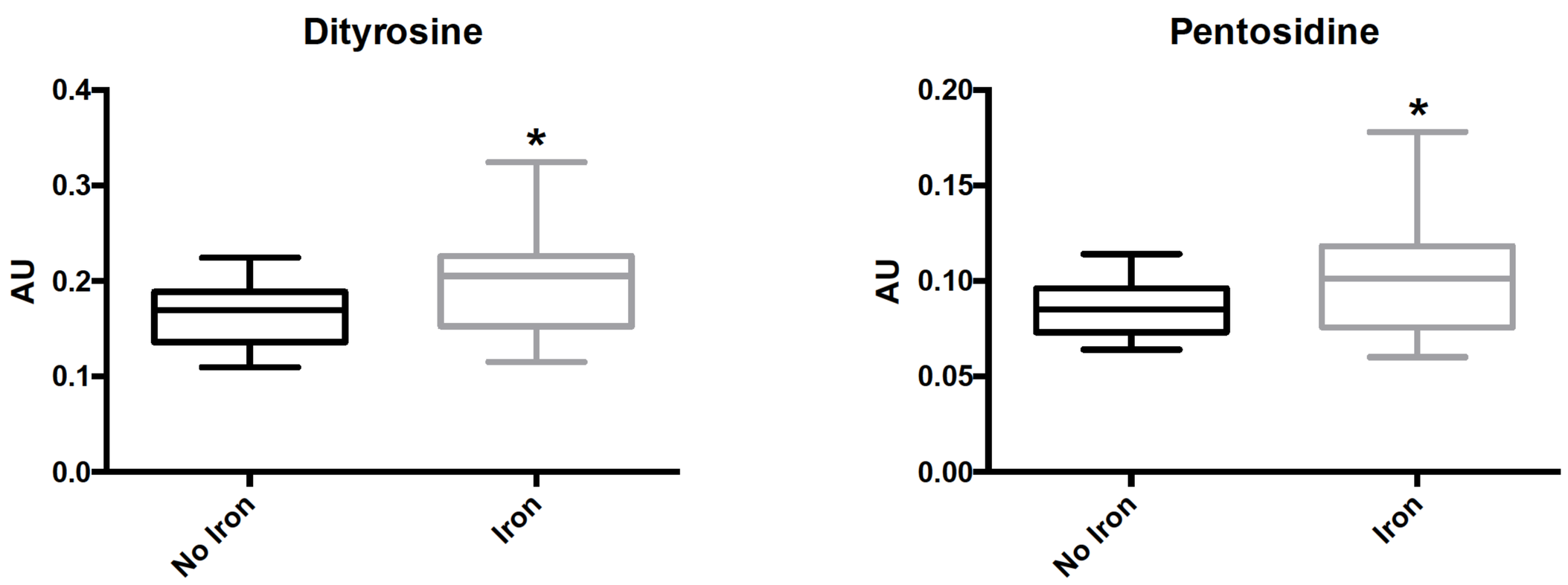

Figure. Plasmatic levels of dityrosine and pentosidine in patients who received or not intravenous iron. AU, Arbitrary Units. (* $p < 0,01$)

CONCLUSIONS

From these data, intravenous iron effectively increases OS in HD patients, irrespective of age, dialysis vintage and inflammatory status. Since OS is a major non-traditional risk factor for the development of cardiovascular disease in HD patients, iron administration must be evaluated carefully and all the strategies to prevent iron supplementation should be pursued, such as adequate diet and nutritional status, avoidance of filter clotting, adequate washing of HD lines and avoidance of useless blood sampling.

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