



Does IV Iron Induce Plasma Oxidative Stress in Critically Ill Patients? A Comparison With Healthy Volunteers*

Submitted by Emmanuel Rineau on Tue, 12/18/2018 - 22:28

Titre Does IV Iron Induce Plasma Oxidative Stress in Critically Ill Patients? A Comparison With Healthy Volunteers*

Type de publication Article de revue

Auteur Lasocki, Sigismond [1], Piednoir, Pascale [2], Couffignal, Camille [3], Rineau, Emmanuel [4], Dufour, Guillaume [5], Lefebvre, Thibaud [6], Puy, Hervé [7], Duval, Xavier [8], Driss, Fathi [9], Schilte, Clementine [10]

Editeur Lippincott, Williams & Wilkins

Type Article scientifique dans une revue à comité de lecture

Année 2016

Langue Anglais

Date Mars 2016

Numéro 3

Pagination 521-530

Volume 44

Titre de la revue Critical Care Medicine

ISSN 0090-3493

Objective:

To compare the oxidative stress induced by IV iron infusion in critically ill patients and in healthy volunteers.

Design:

Multicenter, interventional study.

Setting:

Two ICUs and one clinical research center.

Subjects:

Anemic critically ill patients treated with IV iron and healthy volunteers.

Interventions:

IV infusion of 100 mg of iron sucrose.

Measurements and Main Results:

Thirty-eight anemic patients (hemoglobin, median [interquartile range] = 8.4 g/dL [7.7–9.2]) (men, 25 [66%]; aged 68 yr [48–77]; Simplified Acute Physiology Score II, 48.5 [39–59]) and 39 healthy volunteers (men, 18 [46%]; aged 42.1 yr [29–50]) were included. Blood samples were drawn before (H0) and 2, 6, and 24 hours (H2, H6, and H24) after a 60-minute iron infusion for the determination of nontransferrin bound iron, markers of lipid peroxidation—8 α -isoprostanes, protein oxidation—advanced oxidized protein product, and glutathione reduced/oxidized. Iron infusion had no effect on hemodynamic parameter in patients and volunteers. At baseline, patients had much higher interleukin-6, C-reactive protein, and hepcidin levels. 8 α -isoprostanes was also higher in patients at baseline (8.5 pmol/L [6.5–12.9] vs 4.6 pmol/L [3.5–5.5]), but the area under the curve above baseline from H0 to H6 was not different (p = 0.38). Neither was it for advanced oxidized protein product and nontransferrin bound iron. The area under the curve above baseline from H0 to H6 (glutathione reduced/oxidized) was lower in volunteers (p = 0.009). Eight patients had a second set of dosages (after the fourth iron infusion), showing higher increase in 8 α -isoprostanes.

Conclusions:

In our observation, IV iron infusion does not induce more nontransferrin bound iron, lipid, or protein oxidation in patients compared with volunteers, despite higher inflammation, oxidative stress, and hepcidin levels and lower antioxidant at baseline. In contrary, iron induces a greater decrease in antioxidant, compatible with higher oxidative stress in volunteers than in critically ill patients.

Résumé en anglais

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DOI

10.1097/CCM.0000000000001420 [12]

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Critical Care Medicine

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Publié sur *Okina* (<http://okina.univ-angers.fr>)