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Late Onset Hypotension after Intravenous Iron Sucrose Administration

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Introduction

Hypotensive reactions to intravenous iron sucrose are not rare, they develop under ongoing infusion and resolve within 1 to 2 hours after drug administration. However, severe hypotension on the day following intravenous iron treatment is an exceptional event and thus worth to be presented.

Case

An 89-year-old woman was admitted to inpatient geriatric rehabilitation. Five days earlier she had suffered an ischemic stroke presenting as right hemiplegia and nonfluent aphasia. Workup revealed an occlusion of the M1 segment of the left middle cerebral artery. After thrombectomy and stenting of the middle cerebral artery the paralysis improved to stage Rankin 3, i.e. moderate disability, the patient was able to walk with assistance. Noteworthy in the patient's background medical history was arterial hypertension, renal failure stage 3b, and hypercholesterolemia. She was living until recently independently in the community.

On admission to the rehabilitation ward, the patient was alert, oriented, and comfortable. Her body weight was 45 kg with body mass index of 17. The bodily temperature, heart rate, respiratory rate and oxygen saturation were all within the normal range. Laboratory tests were significant for hemoglobin 9.5 g/dL, mean corpuscular volume 90 fL, eGFR 29 ml/min/1.73 m2, serum iron 10 mcg/dL, ferritin 88 ng/mL, transferrin saturation 4%. Serum vitamin B12 and thyrotropin were normal. Her daily medication treatment included aspirin 100/mg, ticagrelor 180 mg, atorvastatin 80 mg, lercanidipine 10 mg, omeprazole 80 mg, enoxaparin 40 mg and folic acid 5 mg.

Treatment with iron sucrose 200mg intravenously was

started. Two doses of iron sucrose were given without adverse reactions. Unexpectedly, on the 9th day in rehabilitation, at 10 a.m. during assisted walking at the physiotherapy treatment, the patient felt dizzy and lost consciousness; a fall was prevented by the attending physiotherapist. She was returned to bed and her consciousness recovered instantly. There were no convulsions, no dyspnea, vomiting or diarrhea, palpebral and lip edema, or skin eruption. The supine blood pressure was 115/75 mmHg, and the heart rate was 78 bpm. On physical examination there were no new findings. The electrocardiogram showed normal sinus rhythm. Of note, that morning the patient had not received iron sucrose.

All patient medications were temporarily discontinued. Later, at 2 p.m., in bed, she was comfortable. The work-up for what appeared to be a first-in-life syncopal event was resumed. A bedside postural test showed supine BP in the range 85/50 to 94/59 mmHg, heart rate 69 to 73 bpm; sitting BP 77/49 to 109/55 mmHg, heart rate 85 to 87 bpm; on standing the patient became instantly confused and lost postural tone. Lying down, her consciousness and postural tone recovered immediately, with the BP similar to prior supine levels. On the subsequent morning and the ensuing days, the patient was comfortable, had normal body temperature, heart and respiratory rate and oxygen saturation. The BP was in the range of 111/53 to 142/80 mmHg (median 125/61 mmHg). She endured prolonged sitting, before and after meals, as well as assisted walking on physiotherapy. Her prior medications were returned except for lercanidipine and iron sucrose which were permanently stopped. Blood tests five days later showed increase in serum iron to 164 mcg/dL, ferritin to 270 ng/mL, the hemoglobin remained unchanged at 10 g/dL. Among possible causes of acute-onset orthostatic hypotension the following were excluded: acute hemorrhage, dehydration, sepsis, myocardial infarction, venous thromboembolism, and anaphylaxis.

Discussion

There is an obvious association between the initiation of intravenous iron treatment and the onset of hypotension, as well as the recovery from hypotension after discontinuation of intravenous iron administration. Lercanidipine was unlikely the cause of the patient's hypotension since lercanidipine was part of her years-long medications and has been well tolerated. A causal determinism whereby intravenous iron sucrose induced the patient's hypotension, was tested with the Naranjo Adverse Drug Reaction Probability Scale [1]. The following scores of the Naranjo scale support a cause/effect relationship in this instance: the adverse event appeared after the drug was given (+2) and confirmed by objective evidence (+1); it improved when the drug was discontinued (+1); there were no other possible causes for the reaction (+2). In totalizing 6 points on the Naranjo scale, the relationship between intravenous iron sucrose treatment and the ensuing hypotensive event in this patient qualifies as probably causal. While none of the causality assessment instruments have been generally accepted, the Naranjo's algorithm is commonly and confidently used as reference [2]. Rechallenging the patient with the suspected medication is considered unethical in this geriatric case.

Hypotensive reactions under infusion of iron sucrose are not rare, they occur at the very time of the ongoing infusion and resolve with 1 to 2 hours after administration. The short-lived hypotension induced by intravenous iron is attributed to activation of complement by the labile free iron and not to a hypersensitivity reaction [3]. On the contrary, in the present case, hypotension became apparent 18 hours after administration of the last dose of iron sucrose and confined the patient to bed rest for 20 hours at least after the syncopal event. The pathophysiological mechanism of hypotension in this patient is unknown. There was no clinical evidence of fluid imbalance. Likewise, there was no indication of neurogenic dysregulation: the adequate increase in heart rate under postural challenge does not support baroreflex failure.

Indeed, a ratio of <0.5 indicates baroreflex failure while the ratio observed in this patient was 2 and 0.9, respectively [4]. Nevertheless, there are cases of isolated sympathetic failure with intact cardiovagal function that present with orthostatic hypotension but normal heart rate responses [5]. Intravenous iron treatment is associated with the risk of hypophosphatemia, and severe hypophosphatemia can impair myocardial contractility [6]. Yet, the patient’s serum phosphate was in the normal range, before and after intravenous iron treatment. Elaborate investigation of laboratory markers, such as those underlying the common, early,

short-lived hypotensive reaction under intravenous iron administration, were not available to be explored in the present case

There is notice in the literature to delayed minor adverse reactions to intravenous iron treatment, such as arthralgia and myalgia, but severe delayed reactions are scarcely documented like the delayed orthostatic hypotension described in this patient [7,8].

Conclusion

The present observation may serve as an alert to the uncommon and maybe underreported delayed adverse reaction to intravenous iron sucrose.

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