Real-world efficacy and safety of intravenous ferric carboxymaltose for the management of iron deficiency anaemia in Malaysia: A single centre cohort study

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ABSTRACT

Introduction: Up to 24.2% Malaysians are estimated to be affected by anaemia. Iron deficiency is the most common nutritional deficiency leading to anaemia. Oral iron therapy may not be well tolerated or efficient. Ferric carboxymaltose (FCM), a non-dextran intravenous iron formulation, may be an appealing alternative for iron replacement therapy. This retrospective study aimed to investigate the efficacy and safety of intravenous FCM infusion for the management of iron deficiency anaemia in a single centre in Malaysia.

Materials and Methods: All patients who received at least one dose of 500 mg intravenous FCM infusion from January to December 2023 in Bukit Tinggi Medical Centre (BTMC) were identified from the electronic medical record database. Inclusion criteria were patients: (1) ≥ 14 years old and (2) with iron deficiency anaemia. The primary outcome was the mean change in haemoglobin level before treatment and 30 day after treatment. Secondary outcomes included reasons for intravenous FCM infusion, median dose, adverse drug reactions, mean change in haemoglobin levels for different subgroups and percentage of patients with normalised haemoglobin after treatment. The efficacy outcome was analysed using per-protocol analysis while the safety outcome used intention-to-treat analysis. Paired t-test was used to compare the mean difference between the haemoglobin measurements before and 30-day after treatment.

Results: A total of 144 administrations were given to 141 patients requiring intravenous iron replacement therapy during the 1-year study period in BTMC. Intravenous FCM infusion was administered for the management of iron deficiency related to: (1) increased blood loss, including menorrhagia, haemorrhoids and GI-related surgery, (2) low iron intake, including poor nutrition and gastrointestinal-related malabsorption and (3) haematological disorders, including autoimmune haemolytic anaemia, myelodysplastic syndrome, diffuse large B-cell lymphoma and idiopathic thrombocytopaenia purpura. The median dose of intravenous FCM infusion was 1000 mg. At 30 day post-infusion, the mean haemoglobin level increased significantly from 8.9 g/L to 11.6 g/L ($p < 0.05$), an increase of 2.68 g/L (95% CI: 2.45 – 2.90 g/L). No adverse drug reactions were reported. Subgroup analysis showed that patients with haematological disorders had significantly higher improvement in haemoglobin levels after intravenous iron infusion compared to those without. At 7-day, 14-day, 21-day post-infusion, 33% (33/99), 34% (34/99) and 36% (36/99) patients had a normalised haemoglobin level, respectively. The proportion of patients with a normalised haemoglobin level increased to 36% (36/99) and 42% (42/99) at 30-day and 90-day post-infusion.

Conclusion: Within the limit of this single-centre retrospective study, intravenous FCM infusion was well tolerated and effective in increasing the haemoglobin level among patients with iron deficiency anaemia.

KEYWORDS:
Iron deficiency, anaemia, efficacy, safety, ferric carboxymaltose

INTRODUCTION

In 2021, the global prevalence of anaemia across all ages was 24.3%.[1] The Malaysian population suffers a similar trend with a prevalence of 24.2%, approximating 5 million people.[2] Iron deficiency (ID) is the most common nutritional deficiency leading to anaemia, contributing to nearly 50% of all cases of anaemia.[3] ID often occurs when iron requirements are especially high (i.e. during infancy and pregnancy), when dietary iron intake is too low to meet the body's requirements (e.g. due to poor diet or malnutrition), when there is impaired iron absorption (e.g. due to a high intake of phytates or phenolic compounds, gastrointestinal disturbances such as inflammatory bowel syndrome, or chronic inflammatory conditions such as chronic kidney disease, heart failure and cancers), or when iron losses exceed iron intake over a prolonged period (e.g. from blood loss from childbirth or menstruation).[4]

Iron is a key nutrient for haemoglobin and red blood cells production. In normal situations, the body will mobilise the iron stores in the liver, spleen and bone marrow to increase haemoglobin level and thus red blood cell production. However, in the late stages of iron deficiency, when the body's iron store has been depleted, the haemoglobin level decreases below normal level, and iron deficiency anaemia (IDA) develops.[5] While iron is an essential trace mineral that plays a role in many other cellular processes including optimal mitochondrial function for respiration and energy production, iron deficiency can occur without anaemia and is

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associated with a multitude of non-specific symptoms such as fatigue and reduced exercise tolerance.[3]

IDA, which is frequent in pregnancy, can adversely affect both the mother (increased risk of preterm delivery, Caesarean delivery and transfusion) and the neonate (increased risk of intensive care admission, delayed growth and development and an increase in behavioural problems that persist up to 10 years after iron repletion).[3] IDA is also prevalent among patients with gastrointestinal pathologies such as inflammatory bowel disease, gastric bypass, coeliac disease, Helicobacter pylori infection and atrophic gastritis,[3] leading to an array of clinical symptoms such as fatigue, sleeping disorders, attention deficit and agitation, that affected patients' health-related quality of life.[6] While perioperative IDA are associated with increased risk of blood transfusion, in-hospital complications, in-hospital mortality, delayed hospital discharge and poor recovery,[7] ID with and without anaemia in patients with chronic heart failure was independently associated with higher risk of all-cause and cardiovascular mortality.[8] Recent studies also suggested the association of IDA with the development of gastrointestinal (GI) cancers[9] and right-sided colorectal cancer.[10]

Due to the adverse clinical outcomes associated with IDA, the prevention and treatment of IDA become a major public health goal, especially in women, children and individuals in low-income countries.[3] Oral iron remains the first-line therapy for ID and IDA. However, the use of oral iron therapy is limited by: (1) its high incidence of gastrointestinal adverse events such as nausea, vomiting and constipation which can reduce patients' tolerance and compliance to treatment, (2) low iron absorption and bioavailability due to drug interactions and increased hepcidin release associated with chronic inflammatory conditions and (3) slow onset of action. Intravenous iron formulations may potentially solve these issues in the settings of intolerance, or refractoriness to oral iron, inflammatory conditions, need for a rapid recovery of haemoglobin such as active bleeding and severe iron-deficiency anaemia.[3]

Ferric carboxymaltose (FCM), which was approved in Malaysia in late 2021, is a single-dose non-dextran intravenous iron preparation that enables replenishment of iron at a dose of 1000 mg (20 mg iron/kg of body weight) within 15 minutes of infusion.[11,12] This allows total iron replacement in one or two infusions and encourages routine use in a busy outpatient clinic with minimal or no additional resource requirements due to its convenience to both healthcare providers and patients.[3] Moreover, since FCM is a dextran-free iron-carbohydrate complex and it does not react with dextran antibodies, iron replacement with intravenous FCM has been demonstrated to be safe and well tolerated in various disease populations, including gastrointestinal disorders, chronic kidney disease, chronic heart failure, gynaecological and obstetrics disorders.[12,13] Intravenous FCM was also shown to be effective in correcting IDA as well as improving symptom control and quality of life of different populations.[6,12-14]

Given that socioeconomic, cultural and medical conditions in low-resource settings might influence the effectiveness and safety of iron substitution modality compared with high-income countries,[13] this study aimed to evaluate the real-world efficacy and tolerability of intravenous FCM in the management of IDA among patients attending an outpatient hospital clinic setting in Malaysia, an upper middle-income multi-ethnics and multi-cultural country.

MATERIALS AND METHODS

Study Population

All patients who received at least one dose of complete 500 mg intravenous FCM infusion from January to December 2023 at Bukit Tinggi Medical Centre (BTMC) were identified from the electronic medical record database. Following the approved indication for intravenous FCM in Malaysia,[15] inclusion criteria were: patients age ≥14 years old, and diagnosed with IDA. IDA is defined by baseline haemoglobin level pre-infusion < 13 g/dL (men) and < 12 g/dL (women) and serum ferritin < 50 ng/mL (patients without chronic inflammatory conditions) or serum ferritin < 600 ng/mL with transferrin saturation < 20% (patients with chronic inflammatory conditions).[16] Exclusion criteria were: patients who did not complete the intravenous FCM infusion, those without any follow-up haemoglobin level measured post-treatment and those received blood transfusion between infusion and measurement of haemoglobin level.

Study Outcome

The primary outcome was the mean change in haemoglobin level before treatment and 30-days after treatment. Secondary outcomes included adverse drug reactions related to intravenous FCM infusion, reasons for intravenous FCM infusion, median dose of intravenous FCM infusion, mean change in haemoglobin levels for different subgroups and percentage of patients with normalised haemoglobin at 7, 14, 21, 30 days and 90 days after treatment (i.e. haemoglobin level ≥ 12g/dL for women and haemoglobin level ≥ 13 g/dL for men). Demographics and outcomes were collected through a retrospective medical record review. This study was conducted according to the principles outlined in Malaysian Good Clinical Practice, International Council for Harmonisation Good Clinical Practice, Declaration of Helsinki, and any other pertinent local and institutional guidelines. The study was approved by the ethics committee of the Ramsay Sime Darby and National Medical Research Register (RSCH ID-23-03299-8LU, NMRR ID-23-02908-MY5). Ethical permission for collection of anonymous data was granted prior to transcription from patient medical records to a case report form.

Sample Size

The minimum sample size required was 34 patients, calculated using R studio 2024.04.0 + 735 (2024.04.0 + 735) with type 1 error probability set at 0.05, power at 0.80% and effect size at 0.5 g/dL.

Statistical Analysis

While the safety analysis included all patients who all have received at least one dose of intravenous FCM (i.e. intention-to-treat analysis), efficacy analysis included patients who fulfilled the eligibility criteria pre-specified in the protocol (i.e. per-protocol analysis), which were patients aged 14 years and

Table I: Baseline demographics of study participants

Demographics	Safety analysis (n = 141)	Efficacy analysis (n = 99)
Mean age (SD)	40.6 (16.6)	42.2 (16.2)
Gender, n (%)		
Male	14 (9.9)	11 (11.1)
Female	127 (90.1)	88 (88.9)
Ethnicity, n (%)		
Malay	18 (19.9)	19 (19.2)
Chinese	49 (34.8)	36 (36.4)
Indian	64 (45.4)	44 (44.4)
Aetiology		
Increased blood loss		
Menorrhagia	115 (81.6)	85 (85.9)
Haemorrhoids	4 (2.8)	1 (1.0)
GI-related surgery	3 (2.1)	2 (2.0)
Reduced iron intake		
Malnutrition	12 (8.5)	8 (8.1)
Malabsorption	2 (1.4)	1 (1.0)
Haematological disorder		
Autoimmune haemolytic anaemia	12 (8.5)	10 (10.1)
Myelodysplastic syndrome	4 (2.8)	2 (2.0)
DLBCL	1 (0.8)	1 (1.0)
Idiopathic thrombocytopenic purpura	1 (0.8)	1 (1.0)
Comorbidities, n (%)		
Hypertension	10 (7.1)	9 (9.1)
Type 2 Diabetes	3 (2.1)	3 (3.0)
Ischaemic heart disease	3 (2.1)	3 (3.0)
Hypothyroid- related disorder	2 (1.4)	0 (0)
Hyperthyroid-related disorder	1 (0.7)	0 (0)
Liver diseases	2 (1.4)	2 (2.0)
GI-related diseases	5 (3.5)	2 (2.0)
Chronic kidney disease	1 (0.7)	1 (1.0)
Sepsis	4 (2.8)	2 (2.0)
Chronic inflammatory states	16 (11.4)	9 (9.1)
Baseline laboratory level before IV FCM infusion†		
Mean Hb level (g/dL)	8.9 (1.9)	8.9 (1.7)
Mean MCV (fL)	74.9 (11.2)	74.6 (11.2)
Mean MCH (pg)	22.5 (5.0)	22.4 (5.0)
Mean reticulocyte count (cells x 10^9/L)	61.6 (25.8)	59.9 (20.5)
Median serum iron level (µmol/L)	3.7 (2.7, 7.4)	3.6 (2.7, 6.6)
Median serum ferritin level (µg/L)	15.0 (6.0, 74.0)	13.0 (5.0, 65.0)
Median TSAT (%)	6.0 (3.0, 16.0)	5.0 (3.0, 10.5)
Mean serum phosphate level (mmol/L)	1.2 (0.3)	1.2 (0.3)
Median Vitamin D level (nmol/L)	38.0 (28.8, 46.0)	36.5 (26.0, 46.0)
Median Vitamin B12 level (pmol/L)	363.0 (274.0, 476.0)	361.5 (272.5, 467.2)
Median serum folic acid level (nmol/L)	15.9 (10.5, 22.4)	16.4 (10.1, 22.3)
Erythropoietin use	131 (92.9)	92 (92.9)
Blood transfusion	19 (13.5)	0 (0)
1 pack	2 (1.4)	
2 packs	15 (10.6)	

Data presented in number (%) unless otherwise stated. †Normally distributed data were presented in mean (SD) while skewed data were presented in median (IQR). Liver diseases included hepatitis B and cirrhosis. GI-related diseases included cholescystitis, Crohn's disease, inflammatory bowel disease, ulcerative colitis. Chronic inflammatory conditions included chronic kidney disease, chronic liver diseases, chronic GI disorder, MDS, DLBCL, sepsis.

SD: Standard deviations; IQR: Interquartile range (i.e. first quartile and third quartile). DLBCL: Diffuse large B-cell lymphoma; GI: Gastro-intestinal. TSAT: Transferrin saturation.

above, with IDA and at least one follow-up haemoglobin level after intravenous FCM infusion without blood transfusion between intravenous FCM infusion and the measurement of haemoglobin level.

Descriptive statistics were used to summarise the baseline characteristics of study population. Normally distributed data were summarised in mean ± standard deviation while skewed data were summarised in median (first quartile, third quartile). Linear mixed model was used to evaluate the changes in haemoglobin level over time, adjusted for age, gender, ethnics, haematological disorders and use of erythropoietin and accounted for repeated measurements and within-patient correlations. Given the retrospective observational nature, all patients have different follow-up time. The 30-day post-treatment haemoglobin levels were

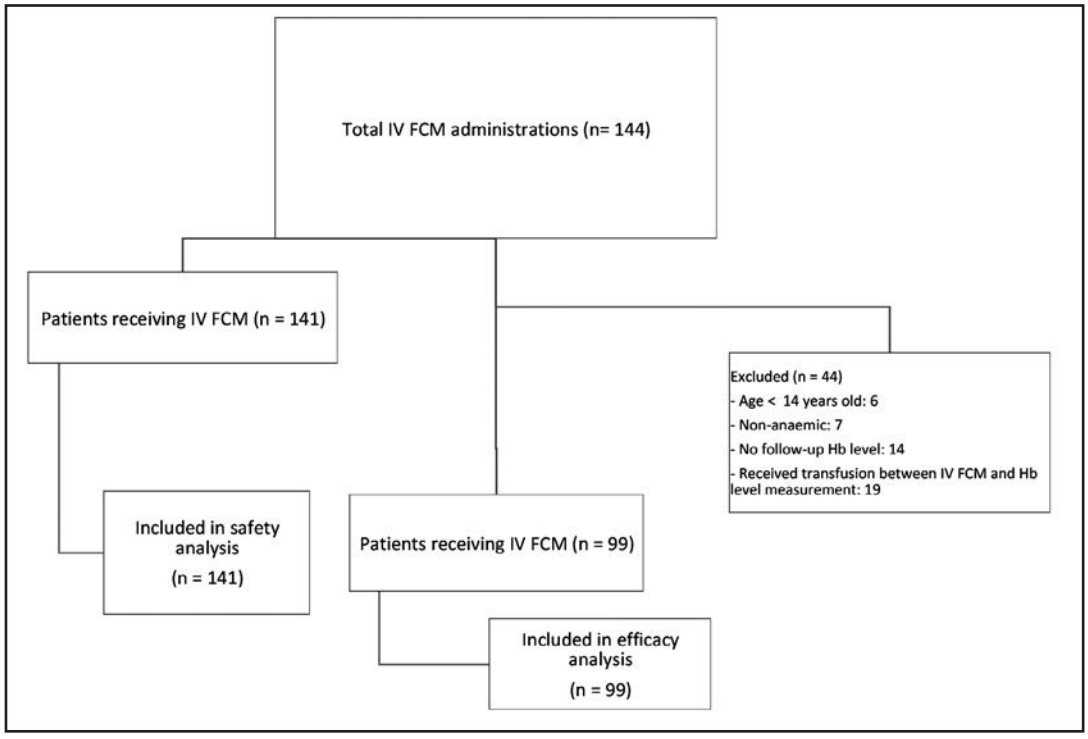

Fig. 1: Study participants flowchat.

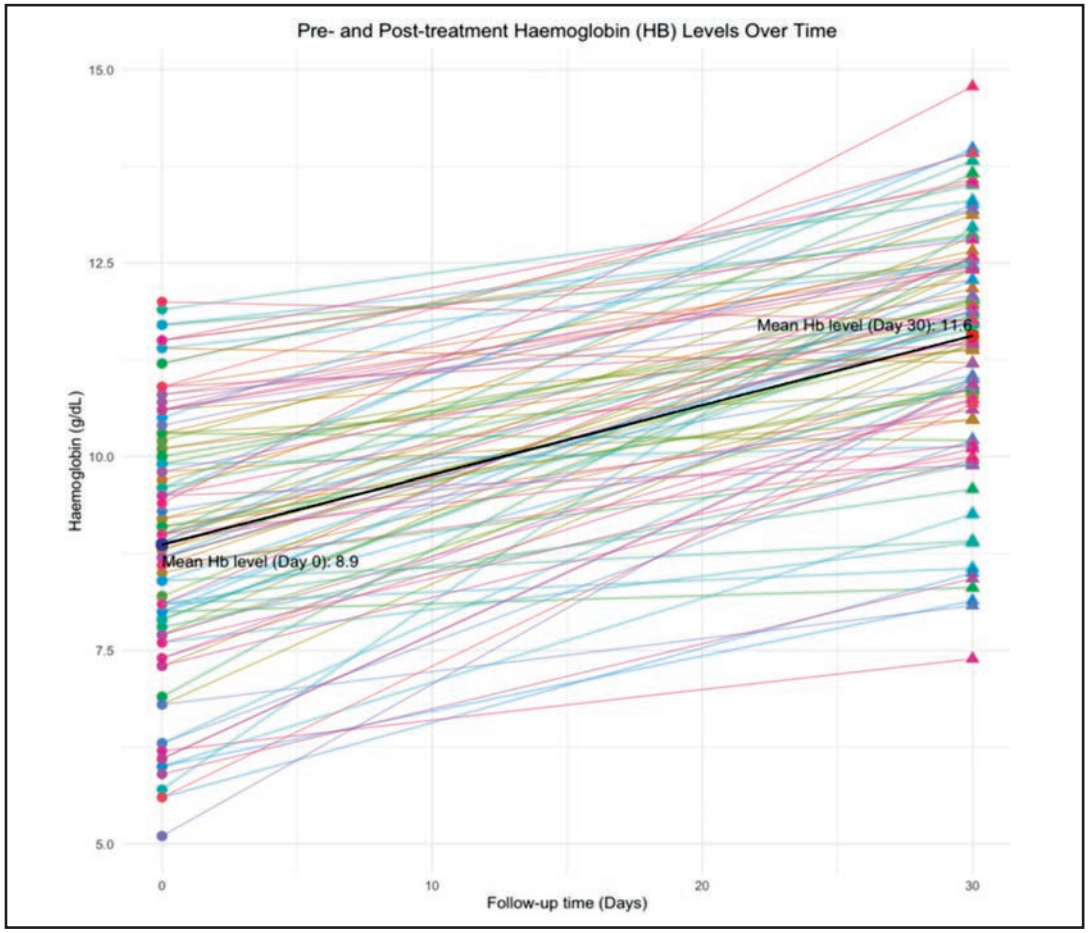

Fig. 2: Model prediction for individual patients at 30-day follow-up.

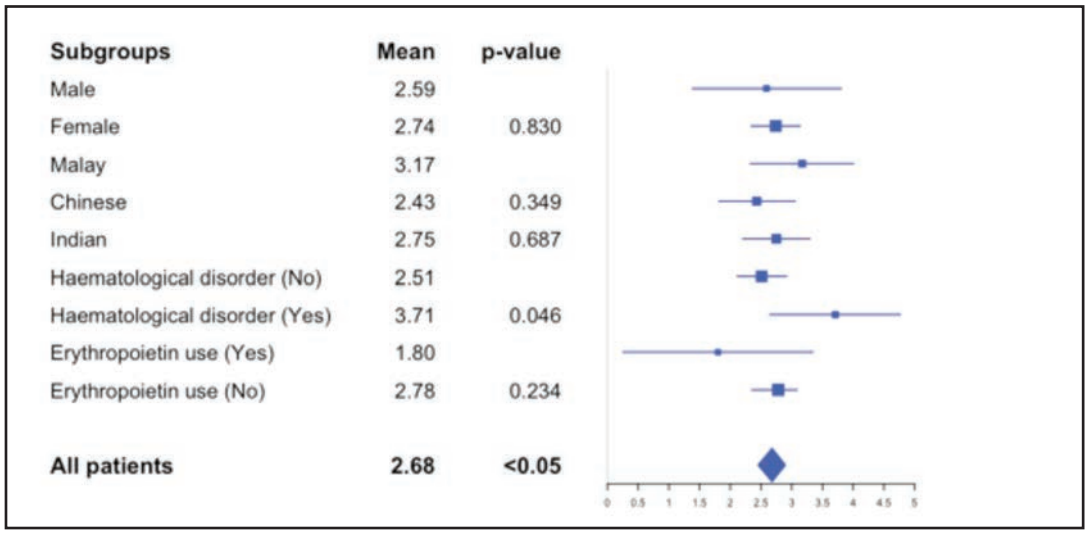

Fig. 3: Subgroup analysis for the mean change in haemoglobin.

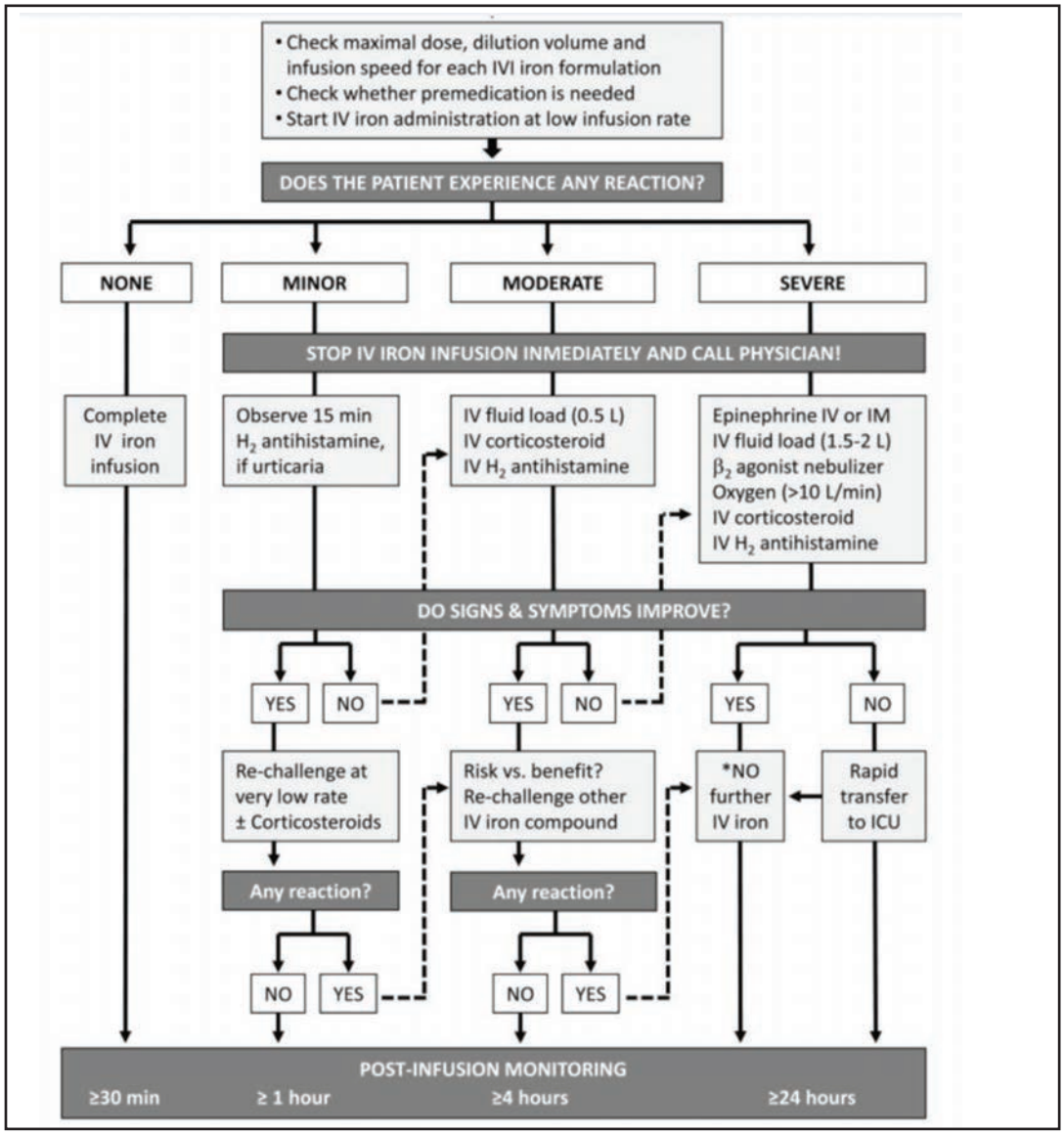

Fig. 4: An algorithm for prevention and management of reactions to intravenous iron administration (Adapted from Gómez-Ramírez et al)[21].

predicted for all per-protocol study population and the mean haemoglobin levels before and after intravenous FCM infusion was compared using paired t-test. Subgroups were compared using paired t-test with Kenward-Roger adjustment. All analyses were performed using R version 4.2.2.

RESULTS

A total of 144 administrations were given to 141 patients requiring intravenous iron replacement therapy (mean age of 40.6 ± 16.6 years old, predominantly Indian and female) during the 1-year study period in BTMC. Intravenous FCM infusion was administered for the management of ID related to: (1) increased blood loss, including menorrhagia, haemorrhoids and GI-related surgery, (2) low iron intake, including poor nutrition and gastrointestinal-related malabsorption and 3) haematological disorders, including autoimmune haemolytic anaemia, myelodysplastic syndrome, diffuse large B-cell lymphoma and idiopathic thrombocytopaenia purpura (Table I). The median dose of intravenous FCM infusion was 1000 mg. No adverse drug reactions were observed among all the 141 patients who received at least one complete intravenous FCM infusion (age ranged from 14 to 88 years old).

The efficacy analysis included 99 patients receiving intravenous FCM infusion with a median follow-up of 47 days (Figure 1). The mean haemoglobin level at pre-infusion was 8.9 ± 1.7 g/dL. At 30-day post-infusion, the model predicted that the mean haemoglobin level increased significantly from 8.9 g/dL to 11.6 g/dL ($p < 0.05$), an increase of 2.68 g/dL (95% CI: 2.45 – 2.90 g/dL) (Figure 2). Post-hoc analysis (Table III) showed no subgroup difference in the change of haemoglobin level except patients with haematological disorders had significantly higher improvement in haemoglobin levels after intravenous iron infusion compared to those without.

At 7-day, 14-day, 21-day post-infusion, 33% (33/99), 34% (34/99) and 36% (36/99) patients had a normalised haemoglobin level, respectively. The proportion of patients with a normalised haemoglobin level increased to 36% (36/99) and 42% (42/99) at 30-day and 90-day post-infusion. There was one patient (1%) who required a repeat intravenous FCM infusion after of 174 days (5.8 months) of first infusion. The drop in haemoglobin level is related to severe menorrhagia. Mean haemoglobin level before the second infusion was 9.9 g/dL, which increased 11.1g/dL and 13.1g/dL after 14 days and 51 days after infusion.

DISCUSSION

This is the first retrospective study that examined the real-world efficacy and safety of intravenous FCM infusion for the treatment of IDA in Malaysia, a multi-ethnicity and multi-cultural upper-middle-income setting. This study provided solid evidence, supporting the safe use of the currently available parenteral iron formulation (i.e. intravenous FCM infusion) in an outpatient setting of a tertiary hospital in Malaysia while overcoming the long-standing clinicians' fear against intravenous iron administration related to the reports of severe adverse events including anaphylaxis, hypotension and shock. This study also shed light on the real-world efficacy of intravenous FCM infusion in improving haemoglobin level for patients with IDA.

Safety

Consistent with many systematic reviews[17-19] and real-world studies,[20,21] our study confirmed that intravenous FCM infusion therapy was well tolerated among patients requiring intravenous iron replacement therapy, without signs of infusion-related reactions (also known as fish-bane reaction) and hypersensitivity. This could be explained by the fact that FCM is a robust and stable molecule with low labile iron, minimising the release of free iron during its administration and allowing greater iron delivery to tissues and a faster repletion of iron stores.[12] Nevertheless, it should be emphasised that adverse events related to intravenous iron infusion can occur. Although our study did not observe any infusion-related reactions such as flushing, headache, dizziness and nausea, these reactions which are due to vascular reaction to labile iron and not hypersensitivity are mild and often self-limiting. They usually abate spontaneously within 5 to 10 min of infusion pause without any intervention and rarely recur upon re-challenge at a lower infusion rate, especially if the patient has been pre-medicated with steroid prior to re-challenging. Skin discoloration from extravasation is also a possible complication and patients should be well informed of this particular risk.[22]

Although hypophosphatemia is frequently reported in intravenous FCM clinical trials,[23] this study did not observe any symptomatic hypophosphatemia. Monitoring serum phosphate levels is recommended in symptomatic patients, particularly in those who require repeated infusions, or in those at higher risk for low phosphate levels (e.g. patients treated with renal replacement therapy, those with chronic diarrhoea and those who have undergone a parathyroidectomy secondary to end-stage renal disease), or in those on medications associated with low absorption or increased excretion of phosphate (antacids, phosphate binders, niacin, acetazolamide, imatinib and sorafenib).[22]

Acute hypersensitivity reactions, which are believed to be caused by complement activation-related pseudo-allergy (CARPA),[22] are uncommon (≥1/1,000 to < 1/100).[11] It is believed that fast infusion of IV iron results in the production rate of anaphylotoxin exceeding its clearance rate from the blood. These anaphylotoxins activate mast cells and basophils, which produce secretory products (i.e. histamine, thromboxanes, leukotrienes and PFA) to trigger hypersensitivity reactions such as bronchospasm, laryngeal oedema, tachycardia, hypo- or hypertension and hypoxia. Given that rapid infusion rate is one risk factor for hypersensitivity, hypersensitivity can be prevented by reducing the rate of infusions. In severe cases, though exceedingly rare (< 1:250,000 administrations), CARPA can lead to anaphylaxis which are life-threatening if not promptly treated and can result in loss of consciousness, shock and cardio-respiratory arrest.[22]

As a preventive measure, it is important to highlight that intravenous iron should be administered only at facilities where staff is trained to identify patients with increased risk of hypersensitivity (e.g., previous mild-to-moderate reactions to IV iron, other drug allergies, severe asthma, eczema, mastocytosis, respiratory or cardiac disease, treatment with hypotensive drugs) or contraindications for intravenous iron (e.g. previous severe reaction to other IV iron, severe hepatic disease, iron overload, first trimester of pregnancy, active infection). Moreover, the staff should be adequately competent to evaluate and manage different types of adverse reactions (Figure 4). Appropriate pharmacological interventions and equipment should be immediately available at the administration sites to manage serious hypersensitivity reaction. Apart from that, before starting intravenous FCM infusion, patients should be well informed about potential adverse events.[22]

During administration, staff should be familiar with and be adherent to the appropriate maximum dose, dilution volume and infusion speed for each intravenous iron formulation, as recommended by the manufacturer, though it is advisable to start all infusions at low rates (< 50% of recommended rate), increasing this after a few minutes if no infusion reaction occurs (Figure 4). An even lower initial infusion rate (10% of the recommended rate during the first 10 to 15 min) is suggested in patients at risk of hypersensitivity reactions. A test dose is no longer recommended, as it does not accurately predict reactions to the subsequent intravenous iron infusion and has never been shown to alter the therapeutic plan. Close monitoring during and at least 30-minutes post-infusion remains a crucial step to ensure patient safety and exclude possibilities of late manifestation of hypersensitivity reactions and anaphylaxis.[22]

Efficacy

Our study underscores the efficacy of intravenous FCM infusion in significantly improving haemoglobin level by 2.7g/dL at 30 days after infusion for patients with IDA at a mean baseline level of 8.9g/dL, consistent with a systematic review of ten observational studies by Srimathi et al17 which found that haemoglobin level rises by 1.3 to 2.5g/dL at 4 weeks post intravenous FCM infusion. This improvement, which was independent of blood transfusion, might be important gain for pregnant mothers, particularly in the third trimester. Pregnant mothers in the third trimester require rapid increase in haemoglobin level before delivery, to prevent both maternal and neonatal complications, including premature birth, maternal death and low birth weight.[3] After 4 weeks of intravenous FCM infusion, while Srimathi et al.[17] estimated that haemoglobin rises up to 3.6g/dL, the effect of intravenous FCM infusion is expected to continue increasing the haemoglobin level beyond 30 days in this study and therefore provide benefits to mothers postnatal such as reducing the risks of depression, fatigue and impaired cognition associated with postpartum anaemia.[24]

Notably, intravenous FCM infusion rapidly corrected anaemia after 7 days of infusion in one-third of patients in this study, suggesting rapid replenishment of iron stores by intravenous FCM for the production of red blood cells. While Van Wyck et al.[25] also reported an increase of haemoglobin by 2 g/dL within 7 days, administering intravenous FCM therapies in patients who require rapid correction of anaemia, including patients with active bleeding and those who will be undergoing surgeries, could potentially reduce the need for blood transfusion and the associated transfusion-related reactions, as well as complications related to peri-operative IDA.[26]

Besides that, intravenous FCM seems to be an appealing therapeutic option compared to the conventional oral iron therapies for patients with menorrhagia as the correction of anaemia was found to be durable, with 42% patients had a sustained correction of anaemia for up to 90 days post-infusion and only one patient required a subsequent repeat infusion for body iron store replenishment, which observed continuous haemoglobin improvement at second infusion without refractories to prior infusion. Those patients who did not sustain the anaemia correction might need more than 1000 mg for iron repletion, require treatment continuation with oral iron or require treatment for the underlying disease that caused IDA.

In this study, 9% patients had chronic inflammatory conditions. In the event when oral iron cannot be absorbed due to hepcidin-mediated blockade associated with chronic inflammation, intravenous FCM is a viable option to increase the haemoglobin level and treat chronic anaemia due to iron deficiency. While patients with underlying haematologic disorders, including autoimmune haemolytic anaemia, myelodysplastic syndrome, diffuse large B-cell lymphoma and idiopathic thrombocytopenic purpura, often requires blood transfusion to correct anaemia, our subgroup analysis suggested that iron therapies significantly improve their haemoglobin levels by up to 3.7g/dL at 30 days after infusion and could potentially reduce the need for blood transfusion.

Given that FCM is a parenteral iron formulations that is strongly bound to carbohydrates (carboxymaltose) with minimal amount of labile iron, it allows rapid administration of total dose of iron in an outpatient setting, requiring fewer hospital visits and reducing treatment costs to the patients or the payers.[22] The routine use of total-dose iron infusion such as intravenous FCM is particularly important in a low-resource setting with high patients load.

Study Limitations

First, this study only included patients receiving intravenous FCM in a single centre, primarily females and Indians, limiting the generalisability of safety and efficacy of intravenous FCM to the entire Malaysian population. Secondly, all patients in this study received pre-medications of hydrocortisone and antihistamine, we cannot exclude the possibility of infusion-related and other adverse reactions in patients not given these medications. Additional monitoring of side effects and precautions are encouraged if pre-medications are not administered. Lastly, most patients received subcutaneous erythropoietin in this study. Although we have adjusted for erythropoietin use in the generalised linear mixed model, we cannot exclude that the improvement in haemoglobin level is in some part due to the erythropoietin use as well as the treatment of the underlying disease not related to the iron replacement therapy only.

CONCLUSION

Within the limits of this single-centre retrospective study, intravenous ferric carboxymaltose (FCM) infusion was well tolerated and effective in increasing the haemoglobin level among patients with iron deficiency anaemia (IDA). Intravenous FCM infusion is an appealing therapeutic option for iron replacement therapy in patients requiring significant increase in haemoglobin levels and rapid correction of anaemia related to iron deficiency, reducing the need for blood transfusion.

DECLARATIONS

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Ethics Approval: RSCH ID-23-03299-8LU, NMRR ID-23-02908-MY5

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