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RESEARCH ARTICLE

FERRIC CARBOXYMALTOSE AND ITS EFFICACY IN ANTENATAL WOMEN WITH SICKLE CELL TRAIT WITH MODERATE TO SEVERE IRON DEFICIENCY ANAEMIA IN THE SECOND TRIMESTER: A HOSPITAL-BASED PROSPECTIVE INTERVENTIONAL STUDY

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Abstract

Background: Iron deficiency anaemia (IDA) is the commonest nutritional deficiency complicating pregnancy in India, and its coexistence with sickle cell trait (SCT), a haemoglobinopathy with carrier prevalence of up to 10–30% in tribal central India is an underexplored high-risk scenario in which iron therapy has traditionally been approached with caution. This study evaluated the efficacy and safety of intravenous ferric carboxymaltose (FCM) in antenatal women with SCT and moderate to severe IDA in the second trimester.

Methods: This prospective interventional study was conducted at Pt. J.N.M. Medical College and Dr. B.R.A.M. Hospital, Raipur, between March 2024 and February 2026. Ninety antenatal women between 14 and 28 weeks of gestation, with electrophoresis-confirmed SCT and moderate to severe IDA (haemoglobin 4.0–9.9 g/dL), were enrolled by consecutive sampling. FCM dose was calculated using the Ganzoni formula and given as a single intravenous infusion. Haematological and iron profile parameters were reassessed at 4 and 6 weeks, and maternal and perinatal outcomes recorded prospectively until delivery.

Results: Mean haemoglobin rose significantly from 7.13 ± 1.27 g/dL at baseline to 9.45 ± 1.27 g/dL at 4 weeks and 10.91 ± 1.31 g/dL at 6 weeks ($p < 0.001$ for all comparisons), an increment of 3.78 g/dL. Serum ferritin increased approximately ten-fold ($p < 0.001$), with concordant improvement in serum iron and transferrin saturation and a reciprocal fall in total iron-binding capacity.

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Blood transfusion was required in only 9 women (10.0%), significantly more often in severe than moderate anaemia (22.6% vs 3.4%; $p = 0.007$). Adverse drug reactions occurred in 14.4% of women, all mild and self-limiting, with no anaphylaxis or infusion discontinuation. Mean birth weight was 2.83 ± 0.44 kg; low birth weight was significantly more frequent in severe anaemia (32.3% vs 13.6%; $p = 0.035$). Haemoglobin improvement was statistically comparable between moderate and severe anaemia ($p = 0.508$).

Conclusion: Intravenous FCM produces rapid, substantial and uniform correction of haemoglobin and iron stores in antenatal women with sickle cell trait and moderate to severe iron deficiency anaemia, with a low transfusion requirement and a favourable safety profile, supporting its use as a preferred second-trimester therapy in this high-risk, previously underinvestigated population.

Introduction:-

Anaemia in pregnancy remains one of the most prevalent and consequential complications of gestation worldwide. The World Health Organization defines anaemia in pregnancy as a haemoglobin concentration below 11 g/dL, and global estimates suggest nearly 40% of pregnant women are affected, with the highest burden in South-East Asia. India bears a disproportionate share, the fifth National Family Health Survey (NFHS-5) reported that 52.2% of pregnant Indian women are anaemic, with only modest decline despite the Anaemia Mukht Bharat initiative. Anaemia in pregnancy is associated with maternal fatigue and heightened infection risk and, in severe form, cardiac failure and death; foetal consequences include foetal growth restriction, low birth weight, preterm birth and stillbirth.

Iron deficiency anaemia (IDA), the most common nutritional cause of anaemia in pregnancy, reflects negative iron balance against the gestational requirement of approximately 1000–1100 mg, and develops progressively from depleted stores to overt anaemia; in India its high burden reflects poor dietary intake, low bioavailability, early marriage, high parity and coexisting haemoglobinopathies. Sickle cell trait (HbAS) with carrier prevalence reaching 10–30% in tribal central India, has traditionally been regarded as a clinically benign carrier state. However, the physiological stresses of pregnancy may render women with SCT more vulnerable to infection and impaired iron utilisation, and when SCT coexists with IDA, oxygen delivery may be further compromised. Historical caution around iron supplementation in haemoglobinopathy carriers, driven by concern for iron overload, is in fact unfounded in SCT, which unlike sickle cell disease is not associated with chronic haemolysis or transfusion-related iron loading.

Oral iron remains first-line treatment for IDA in pregnancy, but its efficacy in moderate to severe anaemia is limited by slow correction, gastrointestinal side effects and poor compliance. FCM a third-generation, non-dextran intravenous iron formulation, allows up to 1000 mg of elemental iron in a single short infusion, with controlled iron release minimising free-iron toxicity while supporting rapid erythropoiesis; a randomised trial has demonstrated significantly higher rates of anaemia correction with FCM than oral iron (84% vs 70%). The second trimester represents the optimal window for parenteral iron therapy, after completion of organogenesis and with sufficient time before delivery to permit full correction. No study to date has specifically evaluated FCM in antenatal women with confirmed sickle cell trait; the present study was designed to assess the efficacy of FCM in correcting moderate to severe IDA in this population in the second trimester, and its effect on transfusion requirement, adverse drug reactions, iron profile, and maternal and foetal outcomes.

Materials and Methods:-

This hospital-based, prospective interventional study was conducted in the Department of Obstetrics and Gynaecology, Pt. J.N.M. Medical College and associated Dr. B.R. Ambedkar Memorial Hospital, Raipur, Chhattisgarh, over 24 months. Pregnant women between 14 and 28 weeks of gestation with moderate to severe IDA (haemoglobin 4.0–9.9 g/dL) and sickle cell trait confirmed by haemoglobin electrophoresis were eligible; women with hypersensitivity to parenteral iron, chronic liver or renal disease, prior iron overload, associated thalassaemia, haemodynamic instability or asthma were excluded. Ninety women were enrolled by consecutive sampling, exceeding the minimum calculated sample size of 77 (based on a mean haemoglobin increment of 1.3 g/dL, pooled SD 2.49, 95% confidence level and 90% power).

IDA was confirmed by complete blood count, iron profile which includes serum ferritin, serum iron, total iron-binding capacity and peripheral smear, and SCT by haemoglobin electrophoresis/HPLC. Total FCM dose was calculated using the Ganzoni formula — total iron deficit (mg) = body weight (kg) × [target haemoglobin – actual haemoglobin (g/dL)] × 0.24 with the 500 mg depot iron excluded.

A single dose, not exceeding 1000 mg of elemental iron per week, was administered under direct medical supervision: 500 mg FCM in 100 mL normal saline infused over 6 minutes, or 1000 mg FCM in 250 mL normal saline over 15 minutes, with the infusion stopped immediately if any adverse reaction occurred.

Complete blood count and iron profile were repeated at 4 and 6 weeks post-infusion, with no further iron supplementation given during follow-up. Women were followed prospectively until delivery for maternal outcomes like mode and gestational age at delivery, transfusion requirement, adverse drug reactions, antenatal/postnatal complications and neonatal outcomes (birth weight, Apgar score, foetal growth restriction). Data were analysed in SPSS v25.0: continuous variables (mean \pm SD, normality assessed via Shapiro–Wilk) were compared using the paired t-test (pre/post) and independent samples t-test or Mann–Whitney U test (between groups); categorical variables were compared using the chi-square or Fisher’s exact test. A two-sided $p < 0.05$ was considered significant.

Results:-

A total of 90 antenatal women with haemoglobin electrophoresis-confirmed sickle cell trait and moderate to severe IDA were enrolled. Mean age was 24.8 ± 4.4 years, with 41.1% aged 23–27 years; most women were enrolled between 19 and 22 weeks of gestation (40.0%) and were primigravida (40.0%). At baseline, 59 women (65.6%) had moderate anaemia (Hb 7.0–9.9 g/dL) and 31 (34.4%) had severe anaemia (Hb 4.0–6.9 g/dL) (Table 1).

Table 1. Baseline demographic and obstetric profile of the study cohort (n = 90).

Variable	n	%
Age group (years)		
18–22	33	36.6
23–27	37	41.1
28–32	15	16.7
33–37	5	5.6
Body mass index (kg/m²)		
Underweight (< 18.5)	22	24.4
Normal (18.5–24.9)	41	45.6
Overweight (25.0–29.9)	22	24.4
Obese (> 30.0)	5	5.6
Gestational age at enrolment (weeks)		
14–18	20	22.2
19–22	36	40.0
23–28	34	37.8
Gravida		
Primigravida (G1)	36	40.0
Gravida 2	32	35.6
Gravida 3	17	18.8
Gravida \geq 4	5	5.6
Grade of anaemia at baseline (WHO criteria)		
Moderate (Hb 7.0–9.9 g/dL)	59	65.6
Severe (Hb 4.0–6.9 g/dL)	31	34.4

Variable	n	%
Total	90	100.0

Mean (\pm SD) age was 24.8 ± 4.4 years. This age group is reflecting marriage and child bearing pattern in central India.

Baseline complete blood count revealed a mean haemoglobin of 7.13 ± 1.27 g/dL with reduced MCV, MCH and MCHC, confirming a microcytic hypochromic pattern, and a low reticulocyte count ($0.79 \pm 0.29\%$) indicating a blunted marrow response. Peripheral smear showed a microcytic hypochromic picture in 57.8% and target cells attributable to SCT in 23.3%. Iron studies confirmed depleted stores, with markedly reduced serum ferritin and transferrin saturation and elevated TIBC (Table 2).

Table 2. Baseline haematological and iron profile of the study cohort (n = 90).

Parameter	Unit	Mean \pm SD
Complete blood count		
Haemoglobin (Hb)	g/dL	7.13 ± 1.27
Packed cell volume (PCV)	%	22.29 ± 3.95
Mean corpuscular volume (MCV)	fL	68.49 ± 6.54
Mean corpuscular haemoglobin (MCH)	pg	21.06 ± 3.11
MCHC	g/dL	28.40 ± 1.75
White blood cell count	$\times 10^3/\mu\text{L}$	10.02 ± 2.14
Platelet count	$\times 10^3/\mu\text{L}$	206.44 ± 38.24
Reticulocyte count	%	0.79 ± 0.29
Iron profile		
Serum ferritin	ng/mL	8.11 ± 2.54
Serum iron	$\mu\text{g/dL}$	40.42 ± 8.86
Total iron-binding capacity (TIBC)	$\mu\text{g/dL}$	421.11 ± 36.24
Transferrin saturation	%	10.63 ± 2.88

MCHC = mean corpuscular haemoglobin concentration. All women met laboratory criteria for moderate to severe iron deficiency anaemia at enrolment.

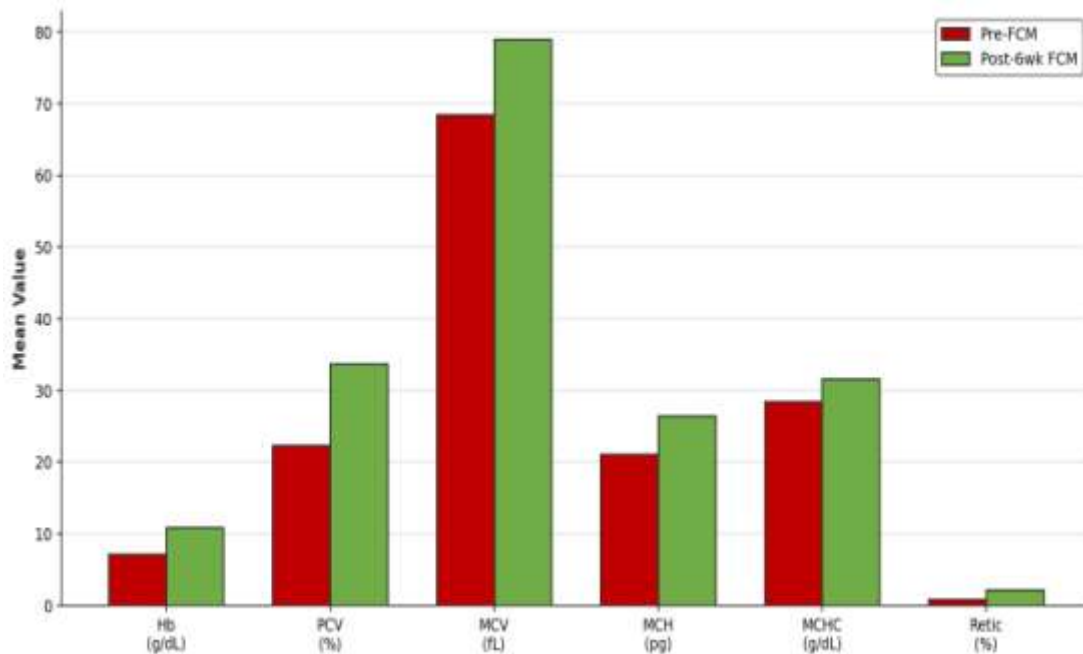
Following FCM administration, mean haemoglobin rose progressively from 7.13 ± 1.27 g/dL at baseline to 9.45 ± 1.27 g/dL at 4 weeks and 10.91 ± 1.31 g/dL at 6 weeks ($p < 0.001$ for all comparisons), an overall increment of 3.78 g/dL, with concordant improvement across all CBC indices including a 2.7-fold rise in reticulocyte count (Table 3).

Table 3. Haemoglobin and complete blood count parameters before and after FCM administration (paired t-test, n = 90).

Parameter	Pre-FCM Mean \pm SD	Post-6-week Mean \pm SD	Mean difference	p-value
Hb (g/dL)	7.13 ± 1.27	10.91 ± 1.31	+3.78	< 0.001
PCV (%)	22.29 ± 3.95	33.73 ± 4.04	+11.44	< 0.001
MCV (fL)	68.49 ± 6.54	78.97 ± 5.29	+10.48	< 0.001
MCH (pg)	21.06 ± 3.11	26.47 ± 2.72	+5.40	< 0.001
MCHC (g/dL)	28.40 ± 1.75	31.63 ± 1.68	+3.22	< 0.001

Parameter	Pre-FCM Mean \pm SD	Post-6-week Mean \pm SD	Mean difference	p-value
WBC ($\times 10^3/\mu\text{L}$)	10.02 \pm 2.14	8.90 \pm 1.65	-1.12	< 0.001
Platelet ($\times 10^3/\mu\text{L}$)	206.44 \pm 38.24	229.96 \pm 31.44	+23.51	< 0.001
Reticulocyte (%)	0.79 \pm 0.29	2.16 \pm 0.47	+1.37	< 0.001

Paired t-test, df = 89. Mean haemoglobin at the interim 4-week assessment was 9.45 \pm 1.27 g/dL (increment +2.32 g/dL from baseline, p < 0.001).



Iron profile parameters mirrored the haematological response: serum ferritin increased approximately ten-fold, serum iron and transferrin saturation increased nearly two-and-a-half-fold and three-fold respectively, and TIBC fell reciprocally as transferrin binding sites became saturated (Table 4).

Table 4. Iron profile parameters before and after FCM administration (paired t-test, n = 90).

Parameter	Pre-FCM Mean \pm SD	Post-6-week Mean \pm SD	Change	p-value
Serum ferritin (ng/mL)	8.11 \pm 2.54	88.75 \pm 18.62	+80.64 \uparrow	< 0.001
Serum iron ($\mu\text{g/dL}$)	40.42 \pm 8.86	97.96 \pm 17.18	+57.54 \uparrow	< 0.001
TIBC ($\mu\text{g/dL}$)	421.11 \pm 36.24	314.59 \pm 24.62	-106.52 \downarrow	< 0.001
Transferrin saturation (%)	10.63 \pm 2.88	31.07 \pm 7.94	+20.44 \uparrow	< 0.001

Paired t-test, df = 89; all parameters p < 0.001. No participant developed clinical or biochemical evidence of iron overload.

Only 9 of 90 women (10.0%) required blood transfusion following FCM administration, near term before delivery, significantly more often among women with severe anaemia (22.6%) than moderate anaemia (3.4%) (Fisher's exact

test, OR = 0.120, p = 0.007). Adverse drug reactions(ADR) occurred in 13 women (14.4%), most commonly itching (5.6%) and rash (3.3%); all were mild and self-limiting, with no anaphylaxis, hypotension, bronchospasm or infusion discontinuation (Table 5).

Table 5. Blood transfusion requirement and adverse drug reaction profile following FCM administration (n = 90).

Outcome	n	%
Blood transfusion required		
Yes	9	10.0
No	81	90.0
Transfusion by grade of anaemia		
Moderate anaemia (n = 59)	2	3.4
Severe anaemia (n = 31)	7	22.6
Adverse drug reactions (ADR)		
Itching	5	5.6
Rash	3	3.3
Body pain	3	3.3
High fever	2	2.2
Patients with any ADR	13	14.4
Patients with no ADR	77	85.6

Fisher's exact test for transfusion requirement by grade of anaemia: OR = 0.120, p = 0.007. No anaphylaxis, hypotension, bronchospasm or infusion discontinuation occurred.

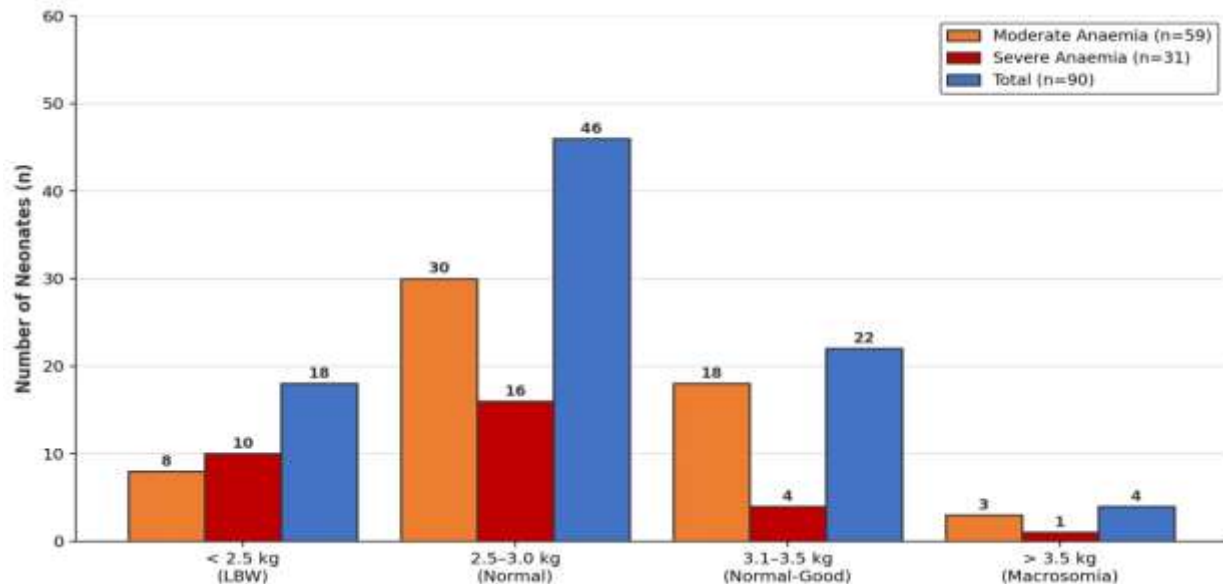
Vaginal delivery occurred in 57 women (63.3%); the remaining 33 (36.7%) underwent caesarean section, most commonly for meconium-stained liquor, foetal distress or prior caesarean. Most women (77.7%) delivered at term and 16.7% preterm. Mean birth weight was 2.83 ± 0.44 kg, with 20.0% of neonates classified as low birth weight; Apgar scores were reassuring (82.2% scoring 7–10 at 1 minute, rising to 92.2% at 5 minutes), and foetal growth restriction was noted in 13.3% (Table 6).

Table 6. Maternal and perinatal outcomes (n = 90).

Outcome	n	%
Mode of delivery		
Vaginal delivery	57	63.3
Lower segment caesarean section (LSCS)	33	36.7
Gestational age at delivery		
Preterm (< 37 weeks)	15	16.7
Term (37–40 weeks)	70	77.7
Post-term (> 40 weeks)	5	5.6
Birth weight		
Low birth weight (< 2.5 kg)	18	20.0
Normal (2.5–3.0 kg)	46	51.1

Outcome	n	%
Normal–good (3.1–3.5 kg)	22	24.4
Macrosomia (> 3.5 kg)	4	4.4
Apgar score at 1 minute		
7–10 (good)	74	82.2
4–6 (moderate)	14	15.6
< 4 (poor)	2	2.2
Foetal growth restriction(FGR)		
Present	12	13.3
Absent	78	86.7

Mean (\pm SD) birth weight was 2.83 ± 0.44 kg. Apgar score ≥ 7 at 5 minutes was recorded in 92.2% of neonates.



When stratified by baseline severity, low birth weight was significantly more common with moderate anaemia (32.3% vs 13.6%; $\chi^2 = 4.441$, $p = 0.035$), though preterm delivery did not differ significantly (25.8% vs 11.9%; $\chi^2 = 2.844$, $p = 0.092$). The mean haemoglobin increment with FCM was statistically comparable between severity groups (3.64 ± 0.64 vs 3.51 ± 0.58 g/dL; $p = 0.508$), indicating uniform efficacy of FCM irrespective of baseline anaemia severity (Table 7).

Table 7. Maternal and foetal outcomes stratified by grade of anaemia.

Outcome	Moderate (n = 59)	Severe (n = 31)	Test statistic	p-value
Low birth weight, n (%)	8 (13.6)	10 (32.3)	$\chi^2 = 4.441$	0.035
Preterm delivery, n (%)	7 (11.9)	8 (25.8)	$\chi^2 = 2.844$	0.092 (NS)
Blood transfusion required, n (%)	2 (3.4)	7 (22.6)	OR = 0.120	0.007
Mean Hb increment at 6 weeks (g/dL)	3.64 ± 0.64	3.51 ± 0.58	$t = -0.665$	0.508 (NS)

NS = not significant. Chi-square test for categorical comparisons; Fisher's exact test for transfusion requirement; independent samples t-test for haemoglobin increment.

Discussion:-

In this prospective study of 90 antenatal women with confirmed sickle cell trait and moderate to severe IDA, intravenous FCM produced a rapid, substantial and statistically uniform rise in haemoglobin and iron stores, with a low transfusion requirement and a favourable safety profile: mean haemoglobin rose by 3.78 g/dL ($p < 0.001$), serum ferritin increased roughly ten-fold, and only 10.0% of women required transfusion. These findings address a previously underexplored gap, since iron supplementation in haemoglobinopathy carriers has historically been approached with caution, despite SCT unlike sickle cell disease carrying no inherent risk of iron overload.

The cohort's age (24.8 ± 4.4 years) and predominantly lean body mass index (24.4% underweight, 45.6% normal) closely parallel earlier Indian and international FCM-in-pregnancy series, reflecting both the early-marriage and early-childbearing pattern of central India and a chronic-undernutrition, rather than obesity-driven, contributor to IDA in this population. Enrolment predominantly at 19–22 weeks mirrors the second-trimester windows used in major trials, a deliberate choice that avoids first-trimester teratogenic concerns while leaving sufficient time before delivery, unlike later third-trimester administration. Baseline anaemia grading (65.6% moderate, 34.4% severe) and mean haemoglobin (7.13 g/dL) were broadly comparable to, though somewhat lower than, prior Indian and Western cohorts, consistent with the combined burden of nutritional deficiency and underlying SCT in this central Indian population.

The 3.78 g/dL haemoglobin increment observed exceeds several previously reported FCM responses and approaches the augmented response seen when FCM is combined with vitamin B12 and folic acid in severe anaemia in Maitri et al, suggesting that correcting coexisting micronutrient deficiency may further enhance the haematological response; concordant improvements in MCV, MCH and MCHC and a meta-analysis by Shin et al confirming FCM's superiority over iron sucrose. The 10.0% transfusion rate, concentrated among women with severe anaemia, is consistent with FCM's superior anaemia-correction rate over oral iron (84% vs 70%) and with expert consensus favouring FCM specifically to reduce transfusion dependency in moderate-to-severe pregnancy anaemia. The 14.4% rate of mild, self-limiting adverse reactions, with no anaphylaxis or infusion discontinuation, mirrors the favourable tolerability reported across diverse obstetric populations.

Mean birth weight (2.83 ± 0.44 kg) and the 20.0% low-birth-weight rate were broadly consistent with prior FCM perinatal data showing no significant adverse effect on neonatal parameters despite significantly improved maternal iron stores. The significantly higher rate of low birth weight in severe versus moderate anaemia, despite an equivalent haemoglobin response to FCM, suggests that some component of foetal growth restriction may already be established by the time severe anaemia is corrected, reinforcing the rationale for early, second-trimester intervention. The 63.3% vaginal delivery rate was more favourable than rates generally reported among women with sickle cell disorders, possibly reflecting both early anaemia correction and the comparatively benign nature of trait, as opposed to disease, status.

A distinctive strength of this study is its exclusive enrolment of haemoglobin electrophoresis-confirmed sickle cell trait carriers, a population not specifically addressed by the major randomised trials and meta-analyses of FCM in pregnancy, extending the evidence base to a group of substantial public health relevance in Chhattisgarh, where SCT prevalence reaches 10–30% in tribal communities. Limitations include the absence of a comparator arm (oral iron or iron sucrose), the single-centre design, and follow-up restricted to 6 weeks without extension into the postpartum period, which precluded capture of longer-term benefits demonstrated elsewhere with extended follow-up. Future multicentre randomised trials in sickle cell trait carriers, incorporating a comparator arm and extended postpartum follow-up, are warranted to build on the evidence generated by the present study.

Conclusion:-

Intravenous FCM produced rapid, substantial and statistically uniform correction of haemoglobin and iron stores in antenatal women with sickle cell trait and moderate to severe iron deficiency anaemia in the second trimester, with a low transfusion requirement, a favourable safety profile and acceptable perinatal outcomes. These findings extend the global and Indian literature on FCM in pregnancy to a previously underinvestigated, high-risk population and support the incorporation of FCM into routine second-trimester antenatal care for women with sickle cell trait and significant iron deficiency anaemia, particularly in regions such as central India where the two conditions frequently coexist.

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Conflict of Interest:-

The authors declare that they have no conflicts of interest relevant to this work.

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