

LACTOFERRIN VERSUS FERROUS SULPHATE FOR THE TREATMENT OF IRON DEFICIENCY ANEMIA DURING PREGNANCY (A RANDOMIZED CLINICAL TRIAL)

By

Mofeed Fawzy Mohamed, Wael Soliman Taha and Mohamed Farrag Ismaeil Farag*

Obstetrics and Gynecology Department, Faculty of Medicine, Al-Azhar University, Cairo, Egypt

***Corresponding Author:** Mohamed Farrag Ismaeil Farag

Phone No.: (+2) 01064717497

E-mail: mohammad.farrag.mf@gmail.com

ABSTRACT

Background: Iron deficiency anemia (IDA) is the condition in which there is decrease in the number of red blood cells or the amount of hemoglobin in the blood. It is caused by insufficient dietary intake and absorption of iron, or iron loss from bleeding. Bleeding can be from a range of sources such as the intestinal, uterine or urinary tract. IDA develops when available iron is insufficient to support normal red cell production and is the most common type of anemia.

Objective: Comparing the efficacy and the safety of Lactoferrin versus ferrous sulphate for the treatment of iron deficiency anemia during pregnancy.

Patients and Methods: This study was done in outpatient clinic of El-Monera General Hospital, from January 2019 to March 2019, between two groups of pregnant women ranging from 20-40 years and gestational age 24-32 weeks with microcytic hypochromic anemia, mild anemia and moderate anemia for 2 months that were selected in a randomized method by the computer.

Results: Oral lactoferrin was better tolerated and more acceptable with higher increase in mean hemoglobin when compared to oral iron therapy over two months treatment.

Conclusion: Oral lactoferrin was better tolerated and more acceptable with higher increase in mean hemoglobin when compared to oral iron therapy over two months treatment.

Keywords: Lactoferrin - Ferrous Sulphate - Iron Deficiency Anemia.

INTRODUCTION

Anemia has a significant impact on the health of the fetus as well as that of the mother. It impairs the oxygen delivery through the placenta to the fetus and interferes with the normal intrauterine growth, leading to fetal loss and perinatal

deaths. Anemia is associated with increased preterm labor (28.2%), preeclampsia (31.2%), and maternal sepsis (*Hutter and Jaeggi, 2010*).

Iron homeostasis is tightly regulated through iron absorption, storage and transport (*Duck and Connor, 2016*).

Significant decreases of total serum iron and serum ferritin combined with increases of serum IL-6 have been observed in pregnant women (*Paesano et al., 2010*) and in haemodialysis patients treated with oral ferrous sulfate, these results strongly support the possibility that iron supplemented via ferrous sulfate is not exported from cells to circulation, but it is accumulated inside host cells resulting in inflammatory conditions (*Baker and Baker, 2012*). This evidence has raised serious questions regarding the safety/efficacy of oral ferrous sulfate, resulting in new approaches for treating ID and IDA and avoiding toxicities associated with iron overload (*Paesano et al., 2010*).

The oral route is the first choice to replace iron stores as this allows the normal mechanism of absorption to be used, in addition to being an inexpensive and effective treatment (*Hutter and Jaeggi, 2010*).

Lactoferrin (formerly known as lacto transferrin) is a glycoprotein, and a member of a transferrin family, thus belonging to those proteins capable of binding and transferring iron (*Nocerino et al., 2014*).

Lactoferrin is a protein found in cow milk and human milk. Colostrum, the first milk produced after a baby is born, contains high levels of lactoferrin, about seven times the amount found in milk produced later on (*Kochhar et al., 2013*). Its actions are mediated by specific receptors, by direct effect on the cellular membrane wall, competition for the iron ions or through its enzymatic function, only to mention few mechanisms through which it realizes all these activities. Its

properties are facilitated by its capacity of maintaining the iron bound in low pH environment, as well as the ability to bind to other substances, such as lipopolysaccharides, heparin, glycosaminoglycans, DNA, oxalates, carboxylates, or other metallic ions (Al^{3+} , Ga^{3+} , Mn^{3+} , Co^{3+} , Cu^{2+} , Zn^{2+}) (*Sharma and Meenakshi, 2014*).

A previous study was published in April 2010, about (Lactoferrin efficacy versus ferrous sulfate in curing iron deficiency and iron deficiency anemia in pregnant women) and found that lactoferrin represent an extremely valid natural drug which, without any adverse effects, prevents and cures IDA more effectively than ferrous sulfate (*Paesano et al., 2010*).

The present study aimed to compare the efficacy and the safety of Lactoferrin versus ferrous sulphate for the treatment of iron deficiency anemia during pregnancy.

PATIENTS AND METHODS

The study included two groups of pregnant women that were randomized by the computer in two groups:

Group 1 (Lactoferrin plus folic acid group) included 100 pregnant women who received lactoferrin 100 (Pravotin 100 mg sachets, Hygint, Egypt) + once daily orally for 2 months.

Group 2 (Ferrous Sulphate group) included 100 pregnant women who received 150 mg of dried ferrous sulphate capsules (Ferrofol capsules, EIPICO, Egypt) once daily orally for 2 months.

Patients were assigned to take the medication orally; once daily before

breakfast, and Pravotin (100 sachets were be dissolved each in ¼ glass of water and taken before breakfast). Patients were advised to avoid the intake of tea, coffee, milk, milk products, antacids and calcium preparation within 2 hours before or after iron capsules.

Women of both groups were selected from pregnant women attending the antenatal outpatient clinic of Al-Monera General Hospital started from January 2019 to march 2019 according to the following eligibility criteria:

Inclusion criteria:

- Age: 20-40 years.
- Pregnant women with iron deficiency anemia.
- Microcytic hypochromic anemia, mild anemia (Hb 10 to 10.9 g/dl) and moderate anemia (Hb 7 to 9.9 g/dl) as per WHO guidelines and S.Ferritin levels <25 ng/dl.
- Gestational age: 24-32 weeks.
- Singleton viable pregnancy.

Exclusion criteria:

- Women with a history of anemia due to any other cause such as chronic blood loss, hemolytic anemia, and thalassemia (including thalassemia trait).
- Severe anemia (<7 g/dl) requiring blood transfusion, bronchial asthma, clinical and/or laboratory evidence of hepatic, renal, hematologic or cardiovascular abnormalities.
- History of peptic ulcer, hypersensitivity to iron preparations and treatment with any other iron preparation in the last

one month before study entry and suspected acute infection.

All women in the study were submitted to complete history taking, general examination, abdominal examination and U/S investigation. They were also subjected to complete blood count and serum ferritin.

Determine the hemoglobin difference and serum ferritin level were repeated after 1 month and 2 months after treatment.

Primary outcome:

The primary outcome was to determine the hemoglobin difference and serum ferritin level 2 months after treatment.

Secondary outcome:

- Nausea, vomiting and GIT upset.
- Compliance of the treatment.
- Constipation.
- Cost effectiveness.

Ethical Aspects:

The study protocol was in agreement to the Helsinki's Principles of Ethical Medical Research [last updated in Brazil 2013]. All women signed informed written consents before participating in the study after thorough explanation of the purpose and procedure of the study.

Sample size was calculated using Power and Sample Size Calculator, setting the power at 80% and the type-1 error at 0.05. Data from a previous very similar trial showed that the mean increase in Hb concentration after treatment was 2.28 ± 0.56 g/dl and 1.16 ± 0.42 g/dl in women who received lactoferrin and those who

received ferrous sulfate, respectively (Rezk *et al.*, 2015).

Statistical analysis was performed using Microsoft Excel version 2015 and SPSS for Windows version 20.0. Data are to be presented in terms of range, mean and standard deviation (for numeric parametric variables); range, median and interquartile range (for numeric non-parametric); or number and percentage

(for categorical variables). Difference between the two groups is to be analyzed using Mann-Whitney's U-test, and mean difference with its 95% CI (for numeric parametric variables); Mann-Whitney's U-test (for numeric non-parametric); or chi-squared test and risk ratio with its 95% CI (for categorical variables). Significance level was set at < 0.05 .

RESULTS

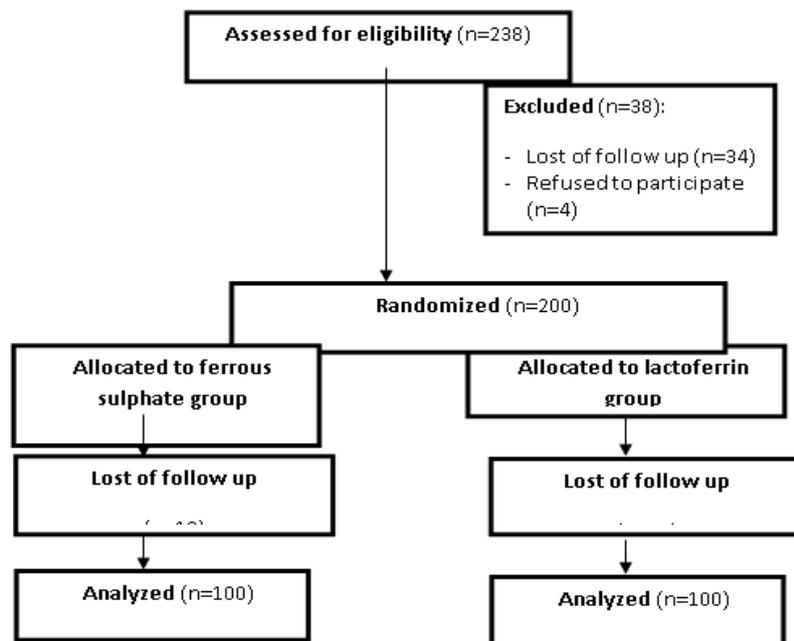


Figure (1): Consort, Patient flow chart

No significant difference between ferrous sulphate and lactoferrin groups regarding demographic characteristics.

Table (1): Demographic characteristics among the studied groups

Variables	Measures	Lactoferrin (N=100)	Ferrous sulphate (N=100)	P
Age (years)	Mean±SD	30.7±4.4	29.5±4.7	>0.05
	Range	20.0–39.0	21.0–39.0	
BMI (kg/m ²)	Mean±SD	28.7±1.6	28.3±1.8	>0.05
	Range	26.0–31.6	25.3–31.4	
Parity (n, %)	Primigravida	24 (24.0%)	28 (28.0%)	>0.05
	Multigravida	76 (76.0%)	72 (72.0%)	
GA (weeks)	Mean±SD	18.9±2.8	19.3±2.9	>0.05
	Range	13.0–26.0	13.0–26.0	
Anemia grade (n, %)	Mild	18 (18.0%)	16 (16.0%)	>0.05
	Moderate	82 (82.0%)	84 (84.0%)	

No significant difference between ferrous sulphate and lactoferrin groups regarding basal hemoglobin. Hemoglobin significantly increased in ferrous sulphate and lactoferrin groups at month-1 and at month-2 after treatment. Hemoglobin at month-1 and at month-2 after treatment

was significantly higher in lactoferrin group than in ferrous sulphate group. Hemoglobin elevation at month-1 and at month-2 after treatment was significantly higher in lactoferrin group than in ferrous sulphate group.

Table (2): Hemoglobin (g/dl) among the studied groups

Time	Measures	Lactoferrin (N=100)	Ferrous sulphate (N=100)	^P
Basal	Mean±SD	8.9±0.9	9.2±0.9	>0.050
	Range	7.3–10.5	7.2–10.7	
Month-1	Mean±SD	10.1±0.9	9.7±0.9	<0.002
	Range	8.4–12.1	7.5–11.5	
Month-2	Mean±SD	11.4±1.1	10.2±1.2	<0.001
	Range	9.3–13.8	7.6–13.1	
Change At Month-1	Mean±SD	1.1±0.2	0.5±0.2	<0.001
	Range	0.7–1.6	0.1–0.9	
	#P	<0.001*	<0.001*	
Change At Month-2	Mean±SD	2.5±0.4	1.1±0.6	<0.001
	Range	1.6–3.5	0.0–2.6	
	#P	<0.001*	<0.001*	
Impact of lactoferrin over ferrous sulphate on Hb elevation				
Time	Mean±SE	95% CI		
Month-1	0.6±0.1	0.5–0.7		
Month-2	1.4±0.1	1.2–1.7		

^Mann-Whitney test, #Wilcoxon Rank test, CI: Confidence interval

No significant difference between ferrous sulphate and lactoferrin groups regarding basal serum ferritin. Serum ferritin significantly increased in ferrous sulphate and lactoferrin groups at month-1 and at month-2 after treatment. Serum ferritin at month-1 and at month-2 after

treatment was significantly higher in lactoferrin group than in ferrous sulphate group. Serum ferritin elevation at month-1 and at month-2 after treatment was significantly higher in lactoferrin group than in ferrous sulphate group.

Table (3): Serum ferritin (ng/dl) among the studied groups

Time	Measures	Lactoferrin (N=100)	Ferrous sulphate (N=100)	^P
Basal	Mean±SD	9.4±1.7	10.0±1.9	<0.001
	Range	6.0–13.0	5.7–15.0	
Month-1	Mean±SD	14.1±2.2	13.2±2.3	<0.001
	Range	9.9–19.3	7.4–18.1	
Month-2	Mean±SD	18.4±2.6	15.5±3.0	<0.001
	Range	12.9–23.5	9.1–23.1	
Change At Month-1	Mean±SD	4.7±0.7	3.2±0.7	<0.001
	Range	3.3–6.7	0.7–4.4	
	#P	<0.001	<0.001	
Change At Month-2	Mean±SD	9.0±1.3	5.5±1.7	<0.001
	Range	5.7–11.5	2.1–9.4	
	#P	<0.001	<0.001	
Impact of lactoferrin over ferrous sulphate on ferritin elevation				
Time		Mean±SE		95% CI
Month-1		1.5±0.1		1.2–1.8
Month-2		3.5±0.3		2.9–4.1

^Mann-Whitney test, #Wilcoxon Rank test, CI: Confidence interval

Maternal poor compliance and side effects were significantly more frequent in lactoferrin group than in ferrous sulphate group.

Table (4): Maternal compliance and side effects among the studied groups

Findings	Lactoferrin (N=100)	Ferrous sulphate (N=100)	^P	RR (95% CI)
Abdominal pain	18 (18.0%)	68 (68.0%)	<0.001	0.291 (0.159–0.532)
Gastric upset	14 (14.0%)	58 (58.0%)	<0.001	0.289 (0.146–0.575)
Nausea	12 (12.0%)	46 (46.0%)	<0.001	0.334 (0.160–0.697)
Vomiting	6 (6.0%)	38 (38.0%)	<0.001	0.226 (0.078–0.658)
Constipation	14 (14.0%)	56 (56.0%)	<0.001	0.302 (0.152–0.600)
Black stool	2 (2.0%)	46 (46.0%)	<0.001	0.065 (0.009–0.443)
Poor compliance	4 (4.0%)	24 (24.0%)	0.001	0.256 (0.070–0.936)

^Chi square test, RR: Relative risk, CI: Confidence interval

Cost effectiveness at month-2 after treatment was significantly lower (more expensive) in lactoferrin group than in ferrous sulphate group. This based on the cost of total course of lactoferrin 280 Egyptian pounds, while that of ferrous sulphate is 12 Egyptian pounds.

Table (5): Cost effectiveness (Egyptian pound/Hemoglobin gm elevation) among the studied groups

Measures	Lactoferrin (N=100)	Ferrous sulphate (N=100)	^P
Mean±SD	232.0±43.4	18.5 ± 6.4	<0.001
Range	160.0–350.0	12–24	
Impact of lactoferrin over ferrous sulphate on cost elevation			
Time		Mean±SE	95% CI
Month-2		213.5±4.387	204.849–222.151

^Mann-Whitney test, CI: Confidence interval

DISCUSSION

Iron deficiency anemia (IDA) is the condition in which there is decrease in the number of red blood cells or the amount of hemoglobin in the blood. It is caused by insufficient dietary intake and absorption of iron, or iron loss from bleeding. Bleeding can be from a range of sources such as the intestinal, uterine or urinary tract. IDA develops when available iron is insufficient to support normal red cell production and is the most common type of anemia (*Stedman's Medical Dictionary, 2006*).

Anemia has a significant impact on the health of the fetus as well as that of the mother. It impairs the oxygen delivery through the placenta to the fetus and interferes with the normal intrauterine growth, leading to fetal loss and perinatal deaths. Anemia is associated with increased preterm labor, preeclampsia, and maternal sepsis (*Hutter and Jaeggi, 2010*).

Ferrous sulfate is the most widely used iron preparation throughout the world. However, despite efficacy and low cost, this drug is associated with high rate of side effects, mainly affecting the gastrointestinal system (*Tolkien et al., 2015*).

Lactoferrin was first identified in milk and then in other human epithelial secretions and barrier body fluids. Many different functions have been attributed to lactoferrin, including protection from iron induced lipid peroxidation, immunomodulation and cell growth regulation and transcriptional activation of specific DNA sequences (*Kochhar et al., 2013*).

Lactoferrin also seems to be involved with regulation of bone marrow function (myelopoiesis), and it seems to be able to boost the body's defense (immune) system. Lactoferrin is a multifunctional protein exhibiting both dependent and independent biological activity based upon its iron binding capacity (*Paesano et al., 2010*).

Its actions are mediated by specific receptors, by direct effect on the cellular membrane wall, competition for the iron ions or through its enzymatic function (*Sharma and Meenakshi, 2014*).

Its proprieties are facilitated by its capacity of maintaining the iron bound in low pH environment, as well as the ability to bind to other substances, such as lipopolysaccharides, heparin, glycosaminoglycans, DNA, oxalates, carboxylates, or other metallic ions (Al³⁺, Ga³⁺, Mn³⁺, Co³⁺, Cu²⁺, Zn²⁺) (*Sharma and Meenakshi, 2014*).

We aimed in this study to compare the efficacy and the safety of Lactoferrin versus ferrous sulphate for the treatment of iron deficiency anemia during pregnancy.

This study was done in outpatient clinic of Al-Monira General Hospital between two groups of pregnant women ranging from 20-40 years and gestational age 24-32 weeks with microcytic hypochromic anemia, mild anemia and moderate anemia for 2 months who were selected in a randomized method by the computer.

All patients were subjected to full history taking, full clinical examination, obstetric history, symptoms of anemia,

past history and General Examination, doing CBC and serum ferritin.

Our study show that no significant difference between ferrous sulphate and lactoferrin groups regarding demographic characteristics.

In our study, the results show that total increase in hemoglobin after 2 months with lactoferrin was higher compared to ferrous sulfate.

Indeed, during the 60 days' period of administration we observed no significant difference between ferrous sulphate and lactoferrin groups regarding basal hemoglobin.

In the present study we found that Hemoglobin elevation at month-1 and at month-2 after treatment was significantly higher in lactoferrin group than in ferrous sulphate group.

Serum ferritin at month-1 and at month-2 after treatment was significantly higher in lactoferrin group than in ferrous sulphate group.

Our data were in a good agreement with those previously reported by *Paesano et al. (2010)* who showed that treatment with bovine lactoferrin is slightly more efficient in reestablishing iron storage.

Our results agree with the results of *Rezk et al. (2015)* that reported increased hemoglobin and total serum iron values to a greater extent in women treated with bLf (bovine lactoferrin) than those observed in women treated orally for 30 days with ferrous sulfate, independently of the trimester of pregnancy and concluded that oral administration of partially iron-saturated bLf enhances intestinal iron delivery better than ferrous sulphate with

the absence of side effects resulted in very high compliance among treated women.

We found also that no significant difference between ferrous sulphate and lactoferrin groups regarding basal serum ferritin.

Also our results disagree with *Nappi et al. (2009)* who showed that bovine lactoferrin probably have the same effect as ferrous sulfate on hematological parameters (Hb, serum iron, serum ferritin rise and TIBC decreases) with significantly fewer gastrointestinal side-effects. This due to his study was done for 30 days only.

We also investigated gastrointestinal side effects of both treatments and observed a higher tolerability of bovine lactoferrin in comparison with ferrous sulfate.

The occurrence of abdominal pain, nausea, vomiting and constipation, in fact, was significantly higher in patients receiving ferrous sulfate in comparison with those receiving lactoferrin. This agreed with *Rezk et al. (2015)* that reported that gastrointestinal adverse events occurred more frequently with ferrous sulphate than lactoferrin group. Also it agreed with *Nappi et al. (2009)* who observed a higher tolerability of bovine lactoferrin in comparison with ferrous sulfate.

In the present study, we found that cost effectiveness at month-2 after treatment was significantly more expensive in lactoferrin group than in ferrous sulphate group. This based on the cost of total course of lactoferrin 280 Egyptian pounds, while that of ferrous sulphate is 12 Egyptian pounds.

This lower incidence of gastrointestinal side effects is due to the different metabolism of the compounds and to the need of administering higher doses of ferrous sulfate. Indeed, fractional iron absorption after oral intake amounts to 10-20% or less. Thus, 80-90% of ingested iron remains in the gut lumen and may cause considerable discomfort. These gastrointestinal effects seem to be due to mucosal irritation and to altered gastrointestinal motility and depend on the labile iron concentration in the lumen.

In the upper part of the small intestine, those effects are directly related to the ingested iron dose. Colonic effects correlate less well with the ingested dose, as differences in absorption, intestinal transit time, and binding to dietary ligands interfere with the availability of iron ions.

On the other hand, lactoferrin is thought to be internalized through endocytosis. Iron is then released from Lactoferrin-Fe complex in intestinal cells and lactoferrin is degraded.

The released iron is then transported through the basolateral membrane into the circulation by transferrin. This proposed apical-to-basolateral Lactoferrin-Fe transport mechanism via a specific receptor in the intestinal cells provides an efficient mechanism for iron uptake.

In anemic pregnant women, daily treatment with bovine lactoferrin has a more effect than ferrous sulfate in restoring iron deposits and in contrasting iron deficiency anemia but it causes less gastrointestinal side effects. Considering that one of the major problems of oral supplementation with ferrous sulfate is the lack of compliance because of the high incidence of gastrointestinal side effects,

bovine lactoferrin seems to be an appealing alternative strategy in pregnant women with iron deficiency anemia.

Inabilities to design a double blind clinical trial and to record the obstetric outcome of women with IDA were a major limitation of our study.

Future research should address obstetric outcome in terms of gestational age at delivery, mode of delivery, maternal complications (postpartum hemorrhage and defective lactation) and neonatal outcome (neonatal weight, admission to neonatal intensive care unit and neonatal death).

According to the results obtained in this clinical trial, oral lactoferrin was better tolerated and more acceptable with higher increase in mean hemoglobin when compared to oral iron therapy over two month treatment. Oral lactoferrin can be used as a good substitute to oral iron therapy in mild to moderate IDA during pregnancy.

CONCLUSION

- Oral lactoferrin was better tolerated and more acceptable with higher increase in mean hemoglobin when compared to oral iron therapy over two month treatment.
- Oral lactoferrin can be used as a good substitute to oral iron therapy in mild to moderate IDA during pregnancy.

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دراسة عشوائية لمقارنة تأثير اللاكتوفيرين مقابل كبريتات الحديدوز لعلاج فقر الدم بسبب نقص الحديد خلال فترة الحمل

مفيد فوزي محمد، وائل سليمان طه، محمد فراج إسماعيل فرج

قسم امراض النساء والتوليد، كلية الطب، جامعة الأزهر، القاهرة، مصر

خلفية البحث: فقر الدم الناجم عن نقص الحديد هو الحالة التي يحدث فيها إنخفاض في عدد خلايا الدم الحمراء أو كمية الهيموجلوبين في الدم. وينتج عن عدم كفاية المدخول الغذائي وإمتصاص الحديد، أو فقدان الحديد من النزيف ويمكن أن يكون النزيف من مجموعة من المصادر مثل الأمعاء أو الرحم أو المسالك البولية وتحدث انيميا نقص الحديد عندما يكون الحديد المتاح غير كافٍ لدعم الإنتاج الطبيعي للخلايا الحمراء وهو أكثر أنواع فقر الدم شيوعاً.

الهدف من البحث: مقارنة فعالية وسلامة اللاكتوفيرين مقابل كبريتات الحديد لعلاج فقر الدم الناجم عن نقص الحديد أثناء الحمل.

المرضى وطرق البحث: أجريت هذه الدراسة في العيادات الخارجية في مستشفى المنيرة العام بين مجموعتين من النساء الحوامل تتراوح أعمارهن بين 20-40 سنة وعمر الحمل 24-32 أسبوعاً اللواتي يعانين من فقر الدم الناقص الكريات، لكل من فقر الدم الخفيف والمعتدل لمدة شهرين واللواتي تم اختيارهم بطريقة عشوائية من قبل الكمبيوتر.

نتائج البحث: كان التحمل عن طريق الفم للاكتوفيرين أفضل وأكثر قبولاً مع زيادة أعلى في الهيموجلوبين بالمقارنة مع العلاج عن طريق الفم بكبريتات الحديدوز لمدة شهرين.

الاستنتاج: كان التحمل عن طريق الفم للاكتوفيرين أفضل وأكثر قبولاً مع زيادة أعلى في الهيموجلوبين بالمقارنة مع العلاج عن طريق الفم بكبريتات الحديدوز لمدة شهرين.