

# Evaluating the Impact of Iron Chelation on Fibroid-Related Symptom Severity in Premenopausal Women with Elevated Serum Ferritin: A Double-Blind Randomized Trial

Aseel Mosa Jabber\*

## ABSTRACT

Iron overload has been associated with uterine fibroids with available treatments options carry limitation whether hormonal or surgical. This study explored the role of using iron chelation intervention to improve fibroid-related symptoms and quality of life in premenopausal women with elevated ferritin levels. To evaluate the clinical impact of Deferasirox, an oral iron chelator, on symptom severity and inflammatory biomarkers in women with MRI-confirmed fibroids and high serum ferritin. A double-blind, randomized, placebo-controlled trial was conducted with 100 women aged 30–45 years received Deferasirox (15mg/kg/day) or placebo for 8 weeks. Outcomes included UFS-QOL symptom severity scores, health-related quality of life (HRQoL), and levels of serum ferritin, IL-6, and estradiol. After week 8, the interventional therapy, there were significant reduction in symptom severity ( $28.94 \pm 12.76$  versus  $45.46 \pm 10.09$ ,  $p < 0.001$ ) and higher HRQoL ( $77.94 \pm 16.22$  versus  $54.28 \pm 14.57$ ,  $p < 0.001$ ) than controls. Ferritin and IL-6 levels declined significantly in the intervention group (ferritin:  $191.18 \pm 82.66$  versus  $270.04 \pm 67.47$  ng/mL,  $p < 0.0001$ ; IL-6:  $29.30 \pm 9.96$  versus  $42.50 \pm 10.44$  pg/mL,  $p < 0.0001$ ). Estradiol levels remained statistically unchanged ( $p = 0.6232$ ). Multivariate regression confirmed that treatment group was the only significant predictor of both improved symptom severity and HRQoL. Iron chelation therapy reduced fibroid-related symptom and improved the quality of life via anti-inflammatory mechanisms, providing a promising non-hormonal treatment for symptomatic uterine fibroids.

**Keywords:** Iron chelation, Deferasirox, Ferritin, Iron overload, Uterine fibroids.

## INTRODUCTION

Iron overload has significant impact on the progression of uterine fibroids through multiple integrated mechanisms. Elevated serum ferritin and tissue iron accumulation in fibroid tissues can intensify oxidative damage by generating reactive oxygen species, leading to DNA damage, cell proliferation, and extracellular matrix accumulation<sup>1</sup>, encouraging fibroid growth and alter the gene expression forms linked with fibroid pathogenesis<sup>2</sup>. Systemic increase in iron concentration stimulate estrogen receptor in fibroid cells, thereby promoting proliferation<sup>3</sup>. Additionally, localized increased concentration of iron directs macrophage differentiation toward a pro-inflammatory M1 phenotype, promoting to fibrogenesis via enhancing of cytokines like IL-6 and TNF- $\alpha$ <sup>2</sup>.

Elevated IL-6 detected in fibroid tissues with iron overload, activating inflammation signalling and enhancing estrogen-driven pathways<sup>3,4</sup>. These together (IL-6 and ferritin) cooperate, where inflammation increases ferritin, and high ferritin levels encourage cytokine release<sup>5,6</sup>. Iron weaken endogenous antioxidant systems raising pro-oxidant milieu that encourage fibroid progression<sup>7,8</sup>. Current therapy for uterine fibroids include as GnRH analogs and selective progesterone receptor modulators (SPRMs). Myomectomy and hysterectomy are alternative options for weak or no response. GnRH analogs effectively reduce fibroid size and bleeding, but long-term therapy limited hypoestrogenic side effects, such as, bone loss and hot flashes<sup>9</sup>. Alternatively, long term use of SPRMs, such as, ulipristal acetate limited by liver toxicity<sup>10,11</sup>. Fibroids recur after myomectomy despite uterus-sparing<sup>11,12</sup>, nevertheless, hysterectomy is eradicating fibroid but eliminates fertility<sup>9,13</sup>, both hormonal and surgical options are costly

and not suitable for all patients, particularly those aiming to preserve fertility<sup>14,15</sup>. The objectives of the study were framed to explore a novel therapeutic approach to managing uterine fibroid symptoms by targeting iron overload, through the administration of Deferasirox, could lead to a measurable improvement in symptom severity among premenopausal women diagnosed with uterine fibroids and presenting with elevated serum ferritin levels.

## PATIENTS AND METHODS

**Study design:** This randomized clinical placebo controlled trial enrolled over all 50 premenopausal females aged 30–45 years old, diagnosed with uterine fibroids in the last six months and free from any other systemic chronic diseases and used no hormonal therapy in the three months. The baseline serum ferritin levels of above 200 ng/ml. Excluded participants are those being pregnant or intending to become a pregnant during the study period and this was determined during pre-enrollment screening. Participants were also excluded in case of having hypersensitivity with previous use of iron chelators, procedures (surgery) to treat fibroids, recent transfusion or current supplementation by iron. Participants with chronic diseases were also excluded, such as, liver, kidneys or blood diseases.

**Sample size and justified calculation:** The sample size was calculated on 0.6 effect size based on Cohen effect ( $d = 0.6$ ), power ( $1 - \beta$ ) of 0.80 and the level of significance ( $\alpha$ ) of 0.05 will reduce the occurrence of Type II error as much as possible. The following formula was used for estimating the required sample size for comparing two independent means:

$$n = [(Z_{1-\alpha/2} + Z_{1-\beta})^2 \times 2 \times \sigma^2] / \Delta^2$$

\* Department of Gynecology and Obstetrics  
College of Medicine, University of ThiQar  
ThiQar, Iraq.  
Email: aseel-m@utq.edu.iq

The required sample size was 44 participants for each per group, we enrolled 50 participants per group, participants allocated based on computer-generated random sequence. **Intervention group treated by** oral administration of Deferasirox 15 mg/kg body weight lasted eight weeks, while control group received placebo therapy for the same time period.

**Assessment of symptom severity:** The assessment of the degree of symptoms based on the Uterine Fibroid Symptom and Quality of Life (UFS-QOL) questionnaire that is consisted of 37 items grouped into two major items: the Symptom Severity Scale (SSS) and the subscales of the Health-Related Quality of Life (HRQL). The SSS had items about the severity and frequency of the most common fibroid symptoms which were heavy menstrual bleeding, pelvic pressure or discomfort, bloating among others. HRQL section further consisted of six subdomains, i.e. concern, activities, energy and mood, control self-consciousness and sexual function. UFS-QOL raw scores were translated into improved faith system scores (based on 0-100 scale) by accepted methods of scoring.

**Biochemical analysis:** Blood samples were collected via venipuncture at baseline and at the end of the study, serum separated, and aliquot prepared, and serum aliquots were immediately stored at  $-80^{\circ}\text{C}$  until analysis. Serum ferritin concentrations were measured using the Crystal Chem Human Ferritin ELISA Kit (Cat# 80964), which required only 20  $\mu\text{L}$  of serum. Interleukin-6 levels were quantified using the R&D Systems Quantikine Human IL-6 Immunoassay (Kit D6050), a solid-phase sandwich ELISA designed for serum and plasma. Estradiol was analyzed using a homogeneous chemiluminescence immunoassay (CLIA) kit from Poclight Bioscience, validated for precise measurement of estradiol in human serum.

**Ethical considerations:** This study was conducted in accordance with the Declaration of Helsinki and written informed consent was obtained from each participant before sample collection.

**Statistical analysis:** All statistical analyses were performed using IBM SPSS V28.0.1.0.USA). Variables were expressed as means and standard deviations. Comparisons of continuous variables were

assessed using independent samples t-tests, with Welch's correction for unequal variances as needed. To control for baseline covariates or after 8 weeks and evaluate the independent contribution of predictors, multivariate linear regression was conducted. Statistical significance was defined as a two-sided p-value  $< 0.05$ .

## RESULTS

The demographic parameters of participants revealed a non-significant ( $p < 0.05$ ) differences existed between intervention group regarding age  $38.87 \pm 4.67$  versus  $39.80 \pm 5.08$  years or BMI  $30.11 \pm 5.25$  versus  $30.50 \pm 5.36$  (Table 1).

These intervention and control groups were comparable at the beginning of the research based on the symptom severity and HRQoL with non-significant differences ( $p = 0.531$  and  $p = 0.291$ , respectively). After 8 weeks, there was a significant difference in the mean score of symptom severity between the intervention group and the control group with the former showing a much larger change in the 8 week measure of  $59.81 \pm 13.10$  to  $28.94 \pm 12.76$  at mid-point and  $58.38 \pm 9.29$  to  $45.46 \pm 10.09$  in the control group, ( $p < 0.001$ ). The HRQoL score was found to have increased to  $77.94 \pm 16.22$  at week 8 as compared to  $42.27 \pm 16.06$  at baseline. Conversely, the mean score of HRQoL among the control group rose to  $54.28 \pm 14.57$  as opposed to the  $39.11 \pm 13.55$  at baseline stage. The 8-week change in HRQoL showed also statistically significant improvement ( $p < 0.001$ ). After eight weeks of intervention, ferritin and IL-6 were reduced. Ferritin and IL-6 significantly ( $p < 0.0001$ ) dropping to  $191.18 \pm 82.66$  and  $29.30 \pm 9.96$  versus control group  $270.04 \pm 67.47$  and  $42.50 \pm 10.44$ , respectively. In contrast, estradiol levels remained stable in both groups over the eight-week period, with no statistically significant differences observed at week 8 ( $p = 0.6232$ ), (Table 2).

For symptom severity at week 8, the intervention group was associated with a highly significant reduction in symptom severity compared to the control group, as indicated by the negative coefficient and a p-value less than 0.001. No other variables, including ferritin, IL-6, estradiol, BMI, or age, showed significant associations with symptom severity at week 8. For HRQoL at week 8, the intervention group

**Table 1.** Comparison of baseline characteristics between intervention and control groups.

| Demographic parameters | Intervention     | Control          | p value |
|------------------------|------------------|------------------|---------|
| Age                    | $38.87 \pm 4.67$ | $39.80 \pm 5.08$ | 0.34    |
| BMI                    | $30.11 \pm 5.25$ | $30.50 \pm 5.36$ | 0.71    |

**Table 2.** Comparison of symptom severity and health-related quality of life between intervention and control groups

| Measured parameters |          | Intervention             | Control                 | p value |
|---------------------|----------|--------------------------|-------------------------|---------|
| Symptom Severity    | Baseline | $59.81 \pm 13.10$        | $58.38 \pm 9.29$        | 0.531   |
|                     | Week 8   | $28.94 \pm 12.76$        | $45.46 \pm 10.09^{*}\#$ | 0.001   |
| HRQoL               | Baseline | $42.27 \pm 16.06$        | $39.11 \pm 13.55$       | 0.291   |
|                     | Week 8   | $77.94 \pm 16.22^{*}\#$  | $54.28 \pm 14.57^{*}$   | 0.001   |
| Ferritin            | Baseline | $306.50 \pm 76.86$       | $292.17 \pm 65.99$      | 0.3196  |
|                     | Week 8   | $191.18 \pm 82.66^{*}\#$ | $270.04 \pm 67.47^{*}$  | 0.00001 |
| IL-6                | Baseline | $46.22 \pm 9.25$         | $45.51 \pm 9.65$        | 0.7064  |
|                     | Week 8   | $29.30 \pm 9.96^{*}\#$   | $42.50 \pm 10.44^{*}$   | 0.00001 |
| Estradiol           | Baseline | $88.18 \pm 23.80$        | $86.47 \pm 25.09$       | 0.7280  |
|                     | Week 8   | $83.23 \pm 23.42$        | $85.65 \pm 25.52$       | 0.6232  |

Data expressed as mean  $\pm$  SD, two-sample t-test used to compare between intervention and control group.

\*# indicate significant differences at p values less than 0.05.

\*as compared to baseline in the same group

#as compared to the other group.

was associated with significant increase in HRQoL compared to the control group ( $p=0.008$ ). Age approached statistical significance as a negative predictor, suggesting a trend toward lower quality of life with increasing age, but this did not reach the conventional significance threshold ( $p=0.052$ ). None of the other variables were significantly associated with HRQoL at week 8, (Table 3).

For serum ferritin at week 8, the analysis found that none of the predictors significantly associated with ferritin levels after adjustment for other variables. The intercept was significant, indicating a baseline ferritin value, but the negative coefficient for the intervention group suggested a trend toward lower ferritin levels in this group that did

not reach conventional significance ( $p=0.179$ ). For IL-6 at week 8, the intervention group showed a significant negative association with IL-6 levels ( $p=0.005$ ), meaning that participants in the intervention group had significantly lower IL-6 levels at week 8 compared to controls, even after adjusting for all other variables in the model. No other predictors were significantly associated with IL-6 levels at week 8. In the case of estradiol at week 8, none of the predictors, including group assignment, symptom severity, HRQoL, BMI, or age, showed a statistically significant association. The intercept was significant, indicating a baseline estradiol value, but the intervention group did not differ from the control group in terms of week 8 estradiol levels ( $p=0.839$ ), (Table 4 and Figure 1).

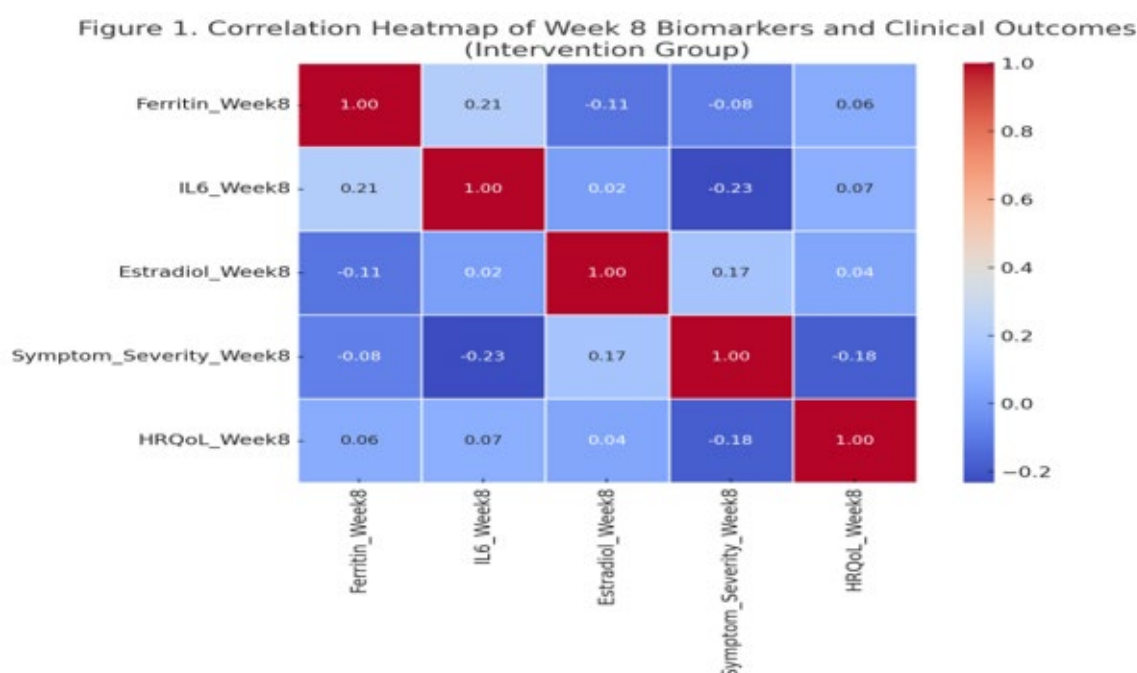
**Table 3.** Multivariate regression analysis of predictors at baseline and week 8 in the studied groups.

| Predictors |          | UFS-QOL, severity |         | HRQoL       |         |
|------------|----------|-------------------|---------|-------------|---------|
|            |          | Coefficient       | P value | Coefficient | P value |
| Ferritin   | Baseline | 0.066             | 0.367   | -0.065      | 0.512   |
|            | Week 8   | -0.062            | 0.378   | 0.069       | 0.466   |
| IL6        | Baseline | 0.145             | 0.647   | 0.105       | 0.806   |
|            | Week 8   | -0.287            | 0.347   | -0.163      | 0.692   |
| Estradiol  | Baseline | 0.051             | 0.801   | 0.219       | 0.421   |
|            | Week 8   | 0.036             | 0.859   | -0.223      | 0.415   |
| BMI        |          | -0.034            | 0.885   | 0.044       | 0.889   |
| Age        |          | 0.264             | 0.288   | -0.656      | 0.052   |

UFS-QOL=Uterine fibroid symptom and quality of life,  
HRQoL= Health-related quality of life

**Table 4.** Multivariate regression analysis of biochemical parameters on quality of life and severity of symptoms in the studied groups.

|            |                  |          | Biochemical at week 8 |       |        |       |           |       |
|------------|------------------|----------|-----------------------|-------|--------|-------|-----------|-------|
|            |                  |          | Ferritin              |       | IL6    |       | Estradiol |       |
| Predictors | Symptom severity | Baseline | -0.616                | 0.671 | 0.180  | 0.354 | -0.568    | 0.219 |
|            |                  | Week 8   | 0.133                 | 0.925 | -0.275 | 0.148 | 0.812     | 0.073 |
|            | HRQoL            | Baseline | 1.361                 | 0.320 | 0.058  | 0.749 | -0.558    | 0.200 |
|            |                  | Week 8   | -1.046                | 0.432 | -0.090 | 0.611 | 0.462     | 0.275 |
|            | BMI              |          | 0.582                 | 0.697 | 0.063  | 0.752 | 0.041     | 0.932 |
|            | Age              |          | -0.367                | 0.828 | 0.172  | 0.446 | -0.010    | 0.986 |



**Figure 1.** Correlation heatmap of week 8 biomarkers and clinical outcomes in the intervention group.

## DISCUSSION

The present study evaluated the effects of iron chelation therapy using Deferasirox on symptom severity and health-related quality of life (HRQoL) in premenopausal women with uterine fibroids and elevated ferritin levels. Over an eight-week period, the intervention group demonstrated reduction in symptom severity and increased HRQoL, compared to the control group, which exhibited more modest improvements.

This novel therapeutic mechanism aligns with previous studies linking iron overload and inflammation to uterine fibroid pathophysiology. However, the present study's strategy of directly lowering ferritin via iron chelation diverges from most existing literature, which has primarily focused on hormonal modulation or surgical interventions. For instance, the use of gonadotropin-releasing hormone (GnRH) agonists like leuprolide acetate has shown efficacy in shrinking fibroid size and improving anemia but is limited by cost and side effects, such as bone density loss<sup>16</sup>.

Alternatively, Seid et al., demonstrated that injectable iron not only improved hemoglobin levels but also enhanced HRQoL in women with abnormal uterine bleeding and iron deficiency anemia<sup>17</sup>. Although their approach directed on iron add-on therapy rather than chelation, confirming that correcting iron physiology improve symptom relief and functional outcomes. Vannuccini et al. reported that fibroid-induced heavy menstrual bleeding resulted in iron deficiency, contributing to patient systemic fatigue and diminished HRQoL<sup>18</sup>.

Nevertheless, Morales-Borges demonstrated that iron coadministration with flavonoids improved anemia in women with abnormal uterine bleeding, but iron management alone may be inadequate without direct fibroid-targeting therapy<sup>19</sup>. Moreover, large-scale study reported that hormonal therapies are the mainstay of symptom control, Stewart et al confirmed that hormonal modulators are useful options for reducing fibroid symptoms and improving patient outcomes<sup>20</sup>. Similarly, Dahiya et al. demonstrated that selective progesterone receptor modulators (e.g. ulipristal acetate) improved bleeding and symptom control and highlighting the efficacy of endocrine modulation<sup>21</sup>. Nonetheless, liver toxicity should be considered during ulipristal therapy<sup>22</sup>.

Results of the current study confirmed that improvement in HRQoL scores based on patient-centered approaches since it was pointed out by Laberge et al. that the enhanced management of fibroid symptoms requires consideration of fertility, and comorbidity<sup>23</sup>.

The present study demonstrated that Deferasirox led to reduction in serum ferritin and interleukin-6 (IL-6) levels after 8-week of treatment of uterine fibroids, while estradiol remained stable, explaining that the therapeutic benefits in uterine fibroid due to anti-inflammatory mechanisms rather than through modulation of estrogenic pathways<sup>24</sup>. Earlier study conducted by Aali et al. (2021) reported that DIBI, an iron chelator, inhibited IL-6 and IL-8 production in cystic fibrosis airway cells exposed to inflammatory stimuli which provided evidence that iron chelation has this effect in non-hematologic tissues<sup>25</sup>. Bruzzese et al. (2023) performed the review of iron chelation agents such as Deferasirox and mentioned their systemic anti-inflammatory and cytoprotective effects in different forms of iron overload<sup>26</sup>. Vlahakos et al. (2020) reported that iron chelation therapy attenuated hyperferritinemia and IL-6 in COVID-19 patients<sup>18</sup>. Susanah et al. (2020) proved that iron overload and the increase in serum ferritin levels is very fast in patients with  $\beta$ -thalassemia, and early initiation of chelation treatment substantially helps to decrease the iron burden before reaching dangerous levels<sup>27</sup>. Conversely, iron chelation of fetal intestinal epithelial cells by Desferoxamine elevated the level of IL-6, reflecting that properties of chelation may depend on the type of cells<sup>28</sup>.

Thuma et al. reported no changes in IL-6 after receiving Desferoxamine in cases of children with cerebral malaria<sup>29</sup>. Cabantchik et al. (2013) reported that deferiprone declined serum ferritin with no impacts on hematological indicators<sup>30</sup>.

## CONCLUSION

**Iron chelation therapy with Deferasirox was associated with a clinically and statistically significant reduction in uterine fibroid-related symptoms and systemic inflammation, as measured by UFS-QOL scores and IL-6 levels, without impacting estradiol levels. These findings support the hypothesis that iron overload contributes to fibroid symptomatology via non-hormonal pathways, particularly inflammation. Deferasirox offers a promising, fertility-preserving alternative to conventional hormonal and surgical interventions. Future research should explore long-term outcomes and the potential for integrating chelation therapy into personalized treatment strategies for uterine fibroid management.**

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**Potential Conflicts of Interest:** None

**Competing Interest:** None

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