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Oral ferrous fumarate dosing regimens in peritoneal dialysis: a single-center retrospective audit

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Background: Anemia is highly prevalent in patients with kidney failure receiving peritoneal dialysis (PD). Oral iron supplementation is frequently prescribed, but adherence may be limited by pill burden and gastrointestinal adverse effects. Emerging evidence suggests that less frequent oral iron dosing may enhance iron absorption by reducing hepcidin-mediated inhibition, yet data in PD patients are lacking. We evaluated iron utilization and compared the effectiveness of different oral iron regimens in PD patients.

Methods: This retrospective cohort study (2007-2025) included adults (≥18 years) receiving peritoneal dialysis at the Dialysis Center Groningen who were treated with oral ferrous fumarate for ≥3 months. Patients receiving intravenous iron, other iron formulations, non-standard dosing regimens, or with active gastrointestinal bleeding were excluded. Ferrous fumarate regimens included 200 mg once daily (OD), twice daily (BID), three times daily (TID), or three times weekly (TIW). Patients not receiving oral iron were evaluated for treatment eligibility. Primary outcomes were 6-month changes in hemoglobin, ferritin, and transferrin saturation. Regimen-specific trajectories were analyzed using linear mixed-effects models. Sensitivity analyses adjusted for age, sex, erythropoiesis-stimulating agent dose, and inflammation, and were restricted to incident iron users on PD.

Results: Of the 140 patients screened, 85 patients were included (median age 61 years; 57.6% male). TIW dosing resulted in hemoglobin trajectories comparable to OD dosing (P = 0.30), with similar ferritin and transferrin saturation trajectories. In contrast, BID dosing was associated with a steeper hemoglobin increase (0.013 g/dL/day; P = 0.001) than OD dosing. Sensitivity analyses yielded similar results. Among patients not receiving iron therapy, 30% (n= 9/30) met contemporaneous guideline criteria for treatment.

Conclusion: In this 18-year retrospective study of 85 PD patients, TIW and OD oral iron supplementation produced comparable hematologic outcomes, suggesting that less frequent dosing may represent an effective strategy to reduce treatment burden without compromising efficacy. Twice-daily dosing

was associated with faster hemoglobin increases, likely reflecting greater cumulative iron exposure. Iron therapy was underutilized in a substantial proportion of eligible patients. Although limited by the modest sample size, these findings highlight the need for expert consensus to address evidence gaps and guide anemia management in PD.

KEYWORDS
anemia, ferrous fumarate, hematologic outcomes, oral iron supplementation, peritoneal dialysis

Highlights

- Thrice weekly oral iron yields similar hemoglobin and iron status as daily dosing.
- Twice daily oral iron leads to a steeper rise in hemoglobin than once daily dosing.
- Less frequent oral iron dosing may be a practical alternative for PD-related anemia.

Introduction

CKD imposes a growing global health burden, with a prevalence exceeding 10% (1–3). Anemia is a common complication of CKD, with prevalence increasing across advancing disease stages and affecting up to 66-97% of patients with kidney failure, and is associated with reduced quality of life and increased cardiovascular morbidity and mortality (4–9). Anemia in CKD is a multifactorial condition, with relative reduction of erythropoietin and iron deficiency, both systemic and iron-restricted, as main contributors (4, 8, 9).

Systemic iron deficiency is characterized by low circulating iron levels and depleted total body iron stores (4, 6). Iron-restricted erythropoiesis, by contrast, occurs when iron stores are sufficient, but the iron cannot be effectively utilized for erythropoiesis, often due to inflammation (4, 9). Inflammatory processes increase hepatic production of hepcidin, which limit intestinal absorption and restrict iron release from storage sites, thereby reducing iron availability for red blood cell production (4, 10–13).

Current KDIGO (Kidney Disease: Improving Global Guidelines) guidelines recommend either intravenous iron or daily oral iron supplementation for PD patients (6). Supporting evidence is primarily derived from studies in hemodialysis and non-dialysis CKD populations, with limited data available for PD patients (14, 15). Peritoneal dialysis patients may require less iron supplementation than those on hemodialysis, as they generally experience fewer bleeding events, exhibit lower levels of chronic inflammation, and retain better residual kidney function (5). Interest in alternate-day oral iron dosing has increased following several studies on women with iron deficiency anemia, which showed that alternate-day dosing is more effective than daily administration. Specifically, daily oral iron led to higher serum hepcidin levels, thereby reducing iron absorption compared with alternate-day regimens (10, 11). Despite these differences, no studies have yet specifically investigated the use of alternate-day oral iron supplementation in the PD population.

In addition to evaluating the efficacy of different oral iron regimens, we assessed iron utilization in PD patients. Previous studies have shown that the evaluation of anemia and iron deficiency in patients with non–dialysis-dependent CKD is suboptimal and that iron therapy is often underutilized (16, 17). Whether similar patterns exist in PD patients remains unclear.

Therefore, this study examined iron utilization in PD patients and compared the efficacy of different oral iron regimens, aiming to determine whether dosing strategies differentially affect hemoglobin (Hb) levels and iron status parameters (18).

Materials and methods

Study design

This study is a clinical audit conducted at Dialysis Centrum Groningen (DCG), utilizing data from patients who received treatment between March 2007 and December 2025. DCG provides comprehensive renal replacement therapies, including center hemodialysis, home hemodialysis and PD, to patients across the three northern provinces of the Netherlands. In 2025, the center treated a total of 170 patients, 21 of whom were managed using home-based modalities (home hemodialysis or PD) (19). In 2021, the prevalence of kidney failure in the Netherlands was 43.9 per 100.000 adults, reflecting the population burden of kidney failure within the country (20). The primary objective was to assess the effectiveness of different iron supplementation regimens in achieving and maintaining adequate Hb levels and iron status in patients with kidney failure undergoing PD. This study was approved by the Medical Ethics Committee of the University Medical Center Groningen (UMCG). The need for participant consent was waived by the committee in accordance with the Dutch Medical Research Involving Human Subjects Act (WMO). This study was written in accordance with the guidelines for Strengthening the Reporting of Observational studies in Epidemiology (21).

Study population and inclusion/exclusion criteria

Eligible participants were adults (≥18 years) with kidney failure who had been undergoing PD for at least three months. Exclusion

criteria included PD duration <3 months, iron supplementation <3 months, the use of iron formulations other than ferrous fumarate, oral iron dosages not consistent with the standard 200 mg regimen, the use of intravenous iron, and any episodes of active gastrointestinal bleeding during the study period. A total of 140 patients were screened for eligibility. After application of the predefined exclusion criteria, 85 patients were included in the final study cohort, as can be seen in Figure 1. Patients were subsequently categorized according to their prescribed oral iron regimen at baseline. Four treatment groups were defined based on the dosing schedule of ferrous fumarate 200 mg: once-daily (OD), twice-daily (BID), three-times-daily (TID), or three-times-weekly (TIW) (86 mg a day). During database creation it became apparent that a significant number of patients (n=30) did not receive any form of iron supplementation. This group was analyzed separately to explore whether the lack of treatment was clinically justified.

Data collection

Data were retrospectively extracted from Electronic Patient Records. All PD patients were screened, and for those meeting the inclusion criteria, baseline characteristics, laboratory measurements and changes in iron- or erythropoietin-stimulating agents-dosage (ESA) were collected. Baseline characteristics were defined using data from the outpatient clinic visit closest to the start date of iron therapy or closest to start PD when patients were already using iron. Laboratory values were obtained as close as possible to this date, aiming to collect a complete iron status, with a maximum allowable interval of three months.

Treatment regimens and follow-up

Patients were treated according to routine clinical practice at the discretion of the treating nephrologists. Follow-up began at initiation of iron supplementation or, for patients already using iron, at the start of PD. Standard follow-up was six months, with a minimum of three months accepted if observation ended earlier due to death, transfer to hemodialysis, or other clinical circumstances. Changes in iron and ESA dosing were documented throughout follow-up.

Laboratory measurements were obtained as part of routine care, leading to variable timing and frequency depending on clinic visits and clinical condition. Iron regimens varied over time, reflecting evolving practice and guideline changes. Oral iron OD became standard from 2012 onward. TID dosing was mainly prescribed between 2012 and 2016, while BID dosing was commonly used between 2012 and 2019. TIW dosing was introduced experimentally between 2022 and 2025 and is not currently included in guideline recommendations for PD patients.

Outcomes

The outcomes were changes in Hb levels (g/dL), serum ferritin (µg/L), and transferrin saturation (TSAT, %). Because TSAT was not consistently reported, it was calculated, using the formula: 100 x serum iron/total iron binding capacity (TIBC) (22).

Statistical analysis

Data were analyzed using IBM SPSS Statistics version 30.0. Baseline characteristics were summarized as median with interquartile range (IQR) for skewed variables. Normality of baseline continuous variables was assessed by visual inspection of histograms and Q–Q plots. Categorical variables were summarized as counts and corresponding percentages. Baseline differences between the oral iron regimen groups were assessed using the Kruskal–Wallis test for continuous variables and Fisher's exact test for categorical variables. Patients not using iron supplementation were excluded from these analyses *a priori* because they addressed a different research question. When the Kruskal–Wallis test indicated a statistically significant difference ($P < 0.05$), *post-hoc* pairwise comparisons were performed using the Mann–Whitney U test with Holm's correction for multiple testing. To ensure a consistent and robust analytical approach across variables, non-

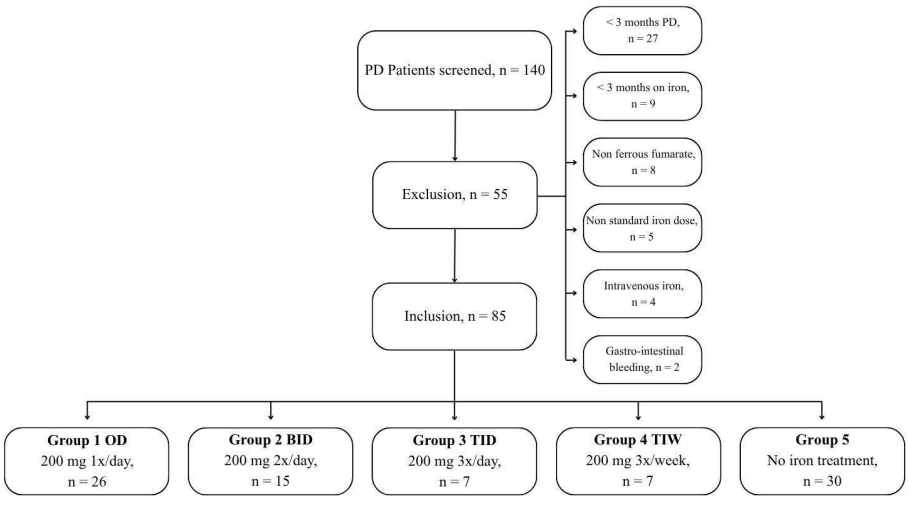


FIGURE 1
Flowchart patient inclusion and exclusion. PD, peritoneal dialysis; OD, once-daily, BID, twice-daily; TID, three-times-daily; TIW, three-times-weekly.

parametric tests were used for group comparisons even when approximate normality was observed. As a descriptive analysis, baseline Hb, ferritin and TSAT values were compared with the last available measurement obtained while patients remained on their initial iron regimen. These analyses were intended to describe absolute changes and did not account for treatment switches. Longitudinal associations between oral iron supplementation regimen and Hb, ferritin, and TSAT were analyzed using linear mixed-effects models to account for the correlation of repeated measurements within patients. Hb was analyzed on the original scale. Ferritin and TSAT were natural log-transformed; the adequacy of these transformations was assessed by visual inspection of histograms and Q–Q plots. For all outcomes, a random intercept for each patient was included to account for individual-level variability in baseline values and within-patient correlation of repeated measurements. Fixed effects included treatment group, time, the treatment group × time interaction, iron use at PD initiation (yes or no), and the interaction between treatment group and iron use at PD initiation. Patients were allowed to contribute data to different treatment groups if they switched iron regimens during follow-up. For the longitudinal mixed-effects analyses, observations were assigned to the regimen being used at the time of measurement. If a patient switched to a regimen outside the primary research question, follow-up was censored at time of switch. These analyses were complemented by sensitivity analyses to assess the robustness of the primary results. In the first analysis, inflammation count and current ESA dose were included as time-varying covariates, while age and sex were included as time-invariant fixed effects. C-Reactive Protein (CRP) values were available in only a minority of patients across all groups. Because of this limited availability, leukocyte counts were chosen as the proxy marker of inflammation. The detailed numbers at baseline and follow-up for each group are summarized in Supplementary Table S1. In a second sensitivity analysis, the primary model specification (including group, time and group × time interaction) was retained, but the analysis was restricted to patients who initiated iron supplementation during their PD period. Estimated marginal means with 95% confidence intervals were calculated for each group at each time point to facilitate interpretation and pairwise comparisons of longitudinal outcomes. Model-based longitudinal prediction plots were generated in R (version 4.5.2) based on the linear mixed-effects models. Statistical significance was defined as $P < 0.05$.

Results

Baseline characteristics

We included 85 PD patients, with a median age of 61 (IQR 45-71) years, and 57.6% of these patients were male (n=49). Overall, the groups were largely comparable in terms of demographic and clinical characteristics. The vast majority of patients were Caucasian (n=76, 89.4%). Hypertensive nephropathy and glomerular diseases were the most common causes of CKD (cumulatively accounting for 42.4%, n =36), and the prevalence of hypertension and diabetes mellitus was 72.9% and 11.8% (n = 62 and 10, respectively). The median Hb level was 10.5 (IQR 9.7-11.8) g/dL (reference range: >13 g/dL for males, >12 g/dL for females) (23), with a median ferritin of 220 (IQR 122–328) µg/L and a median TSAT of 21 (IQR 15–30) % (reference range: ferritin <100 µg/L and TSAT <40% or ferritin ≥ 100 µg/L and <300 µg/L and TSAT <25%) (23). Significant differences between the groups were identified for Hb ($P = 0.047$). *Post-hoc* comparisons showed that baseline Hb was significantly lower in the BID and TIW group compared to the OD group. Aside from these differences, the groups were comparable with respect to other baseline characteristics, as can be seen in Table 1.

Baseline and follow-up Hb and iron status according to different iron regimes

The frequency of laboratory assessments varied across parameters and treatment groups. Hb was measured most consistently, with a median of 6–7 assessments per patient, whereas ferritin and TSAT were measured less frequently (2 to 4 assessments). Iron dose adjustments occurred in 20–43% of patients, and ESA dose adjustments in 43–77% (Supplementary Tables S2, S3). Median follow-up was approximately six months in all groups.

TABLE 1 Baseline characteristics of the 85 included patients according to their iron treatment regime.

	All patients (n = 85)	Group 1 OD, (n = 26)	Group 2 BID, (n = 15)	Group 3 TID, (n = 7)	Group 4 TIW, (n = 7)	Group 5 no iron, (n = 30)
Demographic characteristics						
Sex n, (% male)	49 (57.6)	16 (61.5)	7 (46.7)	4 (57.1)	4 (57.1)	18 (60.0)
Ethnicity n, (% Caucasian)	76 (89.4)	25 (96.2)	12 (80.0)	7 (100)	7 (100)	25 (83.3)
Age (years)	61 (45-71)	63 (34-72)	67 (45-73)	60 (52-65)	66 (60-70)	59 (43-70)
Cause of CKD						
Hypertensive nephropathy n, (%)	14 (16.5)	6 (23.1)	4 (26.7)	1 (14.3)	0 (0.0)	3 (10.0)
Diabetic kidney disease n, (%)	3 (3.5)	1 (3.8)	1 (6.7)	0 (0.0)	0 (0.0)	1 (3.3)
Glomerular disease n, (%)	22 (25.9)	4 (15.4)	1 (6.7)	2 (28.6)	4 (57.1)	12 (36.7)
Tubulointerstitial disease n, (%)	8 (9.4)	1 (3.8)	2 (13.3)	2 (28.6)	1 (14.3)	2 (6.7)

(Continued)

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TABLE 1 Continued

	All patients (n = 85)	Group 1 OD, (n = 26)	Group 2 BID, (n = 15)	Group 3 TID, (n = 7)	Group 4 TIW, (n = 7)	Group 5 no iron, (n = 30)
Cause of CKD						
Vascular disease n, (%)	8 (9.4)	2 (7.7)	2 (13.3)	1 (14.3)	1 (14.3)	2 (6.7)
Obstructive nephropathy n, (%)	10 (11.8)	7 (26.9)	1 (6.7)	0 (0.0)	0 (0.0)	3 (10.0)
Cystic kidney disease n, (%)	8 (9.4)	3 (11.5)	1 (6.7)	1 (14.3)	0 (0.0)	3 (10.0)
Other causes n, (%)	12 (14.1)	2 (7.7)	3 (20.0)	0 (0.0)	1 (14.3)	6 (20.0)
Comorbidities						
Diabetes Mellitus n, (%)	10 (11.8)	2 (7.7)	3 (20.0)	1 (14.3)	0 (0.0)	4 (13.3)
Hypertension n, (%)	62 (72.9)	20 (76.9)	12 (80.0)	5 (71.4)	6 (85.7)	19 (63.3)
Anthropometrics and vital signs						
Systolic BP (mmHg)	146 (130-162)	139 (131-165)	146 (134-172)	139 (138-153)	155 (121-179)	147 (118-156)
Diastolic BP (mmHg)	78 (73-88)	77 (72-92)	76 (67-80)	87 (75-90)	74 (72-84)	82 (74-89)
BMI[a] (kg/m^2)	24.3 (22.8-27.0)	24.1 (22.7-25.7)	24.0 (23.3-27.5)	24.2 (23.6-29.6)	29.0 (24.5-33.1)	24.3 (21.9-27.4)
Dialysis-related characteristics						
Residual diuresis n, (%)	79 (92.9)	21 (80.8)	16 (100)	6 (85.7)	7 (100)	29 (96.7)
Dialysis vintage (months)	0 (0-0)	0 (0-0)	0 (0-0)	0 (0-4.0)	6 (0-40.0)	–
On iron before start PD n, (%)	49 (57.6)	21 (80.8)	12 (80.0)	5 (71.4)	3 (42.9)	8 (26.7)
Previous HD n, (%)	31 (36.5)	9 (34.6)	4 (26.7)	0 (0.0)	4 (57.1)	14 (46.7)
Laboratory parameters						
Hb (g/dL)	10.5 (9.7-11.8)	**10.9 (10.1-12.2)**	**9.5 (8.7-10.8)**	11.1 (9.2-12.1)	**9.7 (9.2-10.0)**	10.6 (9.8-11.6)
Ht (%)	32.0 (29.0-35.5)	33.0 (30.0-37.3)	30.0 (27.0-33.0)	34.0 (28.0-38.0)	30.0 (29.0-30.0)	32.0 (29.0-36.0)
Reticulocytes[b] (‰)	17.1 (12.4-21.7)	17.0 (12.8-22.5)	15.3 (12.3-16.7)	20.0 (11.0-22.0)	20.4 (15.6-22.7)	18.0 (11.9-27.2)
MCV[c] (fL)	91.8 (88.8-95.5)	91.5 (88.9-96.3)	92.2 (89.0-94.1)	88.4 (85.6-92.3)	93.5 (92.3-98.1)	91.7 (88.8-97.7)
Leukocytes ($\times 10^9$/L)	7.2 (6.1-9.2)	7.2 (5.7-9.6)	7.0 (6.4-8.1)	7.1 (5.4-9.6)	6.8 (4.9-9.2)	7.6 (6.3-9.6)
Ferritin[c] (µg/L)	220 (122-328)	220 (112-361)	213 (129-289)	198 (142-281)	102 (31-252)	259 (149-416)
Transferrin[d] (g/L)	2.1 (1.8-2.3)	2.1 (1.9-2.5)	2.2 (1.9-2.7)	2.0 (1.6-x)	2.1 (1.8-2.5)	2.0 (1.7-2.3)
TSAT[e] * (%)	21 (15-30)	21 (15-31)	22 (15-29)	31 (17-47)	16 (14-19)	23 (15-30)
Serum iron[e] (µg/L)	10.8 (7.9-15.0)	10.1 (7.5-15.4)	10.9 (8.0-13.6)	15.5 (7.7-22.5)	8.5 (7.6-10.1)	11.3 (7.8-15.5)
TIBC[e] (µmol/L)	51 (46-58)	52 (46-56)	49 (48-57)	47 (42-52)	52 (46-62)	51 (45-58)
eGFR ($ml/min/1{,}73m^2$)	6.7 (4.9-8.7)	5.9 (4.4-8.2)	6.7 (5.4-8.6)	7.5 (5.3-9.2)	6.4 (3.9-8.7)	6.8 (4.9-8.9)
Medication						
Use of Darbepoetin α n, (%)	65 (76)	19 (73)	15 (100)	5 (71)	6 (86)	20 (67)
Darbepoetin α (UG/week)	20 (9-40)	20 (0-40)	30 (10-50)	15 (0-40)	30 (13-40)	18 (0-40)
Use of PPI n, (%)	41 (48.2)	8 (30.8)	7 (46.7)	4 (57.1)	4 (57.2)	18 (60.0)
Use of anticoagulation n, (%)	23 (27.1)	6 (23.1)	4 (26.7)	2 (28.6)	5 (71.5)	6 (20.0)
Use of ACE-inhibitors n, (%)	17 (20.0)	6 (23.1)	2 (13.3)	1 (14.3)	2 (28.6)	6 (20.0)

Variables with a skewed distribution are presented as median (IQR). Categorical variables as numbers with corresponding percentages. The bold results differ significantly from each other. OD, once-daily; BID, twice-daily; TID, three-times-daily; TIW, three-times-weekly; CKD, chronic kidney disease BP, blood pressure; HD, hemodialysis; Hb, hemoglobin; Ht, hematocrit; MCV, mean corpuscular volume; TSAT, transferrin saturation; TIBC, total iron binding capacity; PTH, parathyroid hormone; PPI, Proton Pump Inhibitor; ACE-inhibitor, Angiotensin-Converting Enzyme inhibitor.

[a]Data available in 82 patients. [b]Data available in 81 patients. [c]Data available in 83 patients. [d]Data available in 59 patients. [e]Data available in 84 patients. [f]Data available in 75 patients.

*TSAT was calculated using the formula: 100 x serum iron/TIBC (22).

Descriptive changes within the initial treatment groups are presented in Table 2. Hb, ferritin, and TSAT values varied between treatment groups at baseline and follow-up. Median Hb remained stable in the OD group (10.9 g/dL) over follow-up. In contrast, Hb increased in the other groups, with the largest absolute difference in the BID group (from 9.5 at baseline to 11.6 g/dL at last follow-up), followed by the TIW group (from 9.7 to 10.4 g/dL), and TID group (11.4 to 11.9 g/dL). Median ferritin and TSAT values showed heterogeneous patterns between regimens without a consistent direction of change across groups. Ferritin increased most in the OD group (from 220 to 293 µg/L), and in the TIW group which started with the lowest ferritin (from 102 to 112 µg/L), while it

remained stable in the BID group (from 213 to 209 µg/L) and TID group (from 198 to 195 µg/L). TSAT also showed an increase in the OD and TIW group (from 21 to 28%, and from 16 to 30%, respectively), and a decline in the TID group (31 to 23%).

Linear mixed models regarding oral iron regimens

Hemoglobin

In the reference group receiving OD oral iron supplementation, Hb levels showed a non-significant decline over time (β= -0.002 g/dL per day; 95% CI -0.007 to 0.002; $P < 0.30$) (Table 3; Figures 2A–D). Compared with this group, patients receiving BID had significantly lower baseline Hb levels (β = −1.22 g/dL; 95% CI −1.77 to −0.67; $P < 0.001$) but demonstrated a significantly steeper increase in Hb over time (β = 0.013 g/dL per day; 95% CI 0.006 to 0.020; $P < 0.001$ for the group-by-time interaction). Interestingly, patients receiving oral iron TIW did not differ from the OD dosing in average baseline Hb levels (β = −0.54 g/dL; 95% CI −1.29 to 0.22; $P = 0.16$), and Hb trajectories over time were also comparable ($P = 0.30$ for the group-by-time interaction).

Patients who were not using iron supplementation prior to the initiation of PD had consistently lower Hb levels throughout follow-up (β = −0.79 g/dL; 95% CI −1.20 to −0.38; $P < 0.001$).

Ferritin

No significant differences in ferritin levels were observed between dosing groups on average at baseline (Table 3; Figures 2E–H). In the OD group, ferritin showed a non-significant upward trend over time (β = 0.002 per day; 95% CI 0.000 to 0.005; $P = 0.07$). The BID dosing exhibited a different ferritin trajectory compared with OD dosing, characterized by a relative decrease over time (β = −0.005 per day; 95% CI −0.009 to −0.001; $P = 0.01$). Although ferritin trajectories were not identical between the TIW and OD dosing groups, no statistically significant differences were observed. Patients not receiving iron supplementation prior to the initiation of PD had lower ferritin levels throughout follow-up (β = −0.80; 95% CI −1.13 to −0.46; $P < 0.001$).

TSAT

No statistically significant differences in average TSAT levels were observed between oral iron dosing regimens across follow-up (Table 3; Figures 2I–L). TSAT remained stable over time in the OD group, and no significant group-by-time interactions were detected, indicating similar TSAT trajectories across dosing regimens. TSAT levels at baseline and trajectories over time in patients receiving BID or TIW dosing were comparable to those observed with OD dosing. Prior iron supplementation before the initiation of PD was not associated with TSAT levels.

Sensitivity analyses

As first sensitivity analysis, we additionally adjusted the mixed models for time-varying inflammation count, current ESA dose, age, and sex. The direction and magnitude of the estimated group-by-time interactions for Hb, ferritin, and TSAT were comparable to those observed in the primary analyses (Supplementary Table S4). Accordingly, these additional adjustments did not alter the overall conclusions. The Hb trajectory changed modestly, from a slight decline to a slight increase compared with the primary analyses (-0.003 to 0.001 g/dL per day). For ferritin the relative decrease over time in the BID group persisted.

Second, we restricted a sensitivity analysis to patients who initiated iron supplementation during PD. The effect estimates differed slightly from those in the primary analysis, with changes in the direction of associations observed for some dosing groups (Supplementary Table S5). Group sizes were small (n= 5, 3, 2 and 4, respectively), therefore these analyses are inherently exploratory and underpowered. Consequently, the resulting *P*-values should be interpreted with caution, as they carry a substantial risk of spurious findings. For the BID group, the previously observed steeper increase in Hb over time disappeared. For TSAT, a significant increase over time was observed in the OD dosing group (0.004% per day on the logarithmic scale; 95% CI 0.001 to 0.008; $P = 0.03$). BID and TID dosing showed a significant decrease in TSAT over time compared to OD dosing (-0.006% per day on the logarithmic scale for BID dosing; 95% CI -0.011 to 0.000; $P=0.05$ and -0.005% per day on the logarithmic scale for TID dosing; 95% CI -0.010 – 0.000; $P=0.05$).

No iron supplementation

During screening, it became apparent that 30 of 85 patients (35%) did not receive iron therapy during their period on PD. Of these 30 patients, 14 had previously been treated with hemodialysis. Among these 14 patients, 8 had received iron therapy during their hemodialysis period, but no iron was restarted after initiation of PD.

TABLE 2 Hemoglobin and iron status parameters at baseline and end of follow-up across the initial iron regimes.

	Group 1 OD, n = 26		Group 2 BID, n = 15		Group 3 TID, n = 7		Group 4 TIW, n = 7	
	Baseline	Follow-up	Baseline	Follow-up	Baseline	Follow-up	Baseline	Follow-up
Median Hb (g/dL)	10.9 (9.2-12.1)	10.9 (9.0-11.8)	9.5 (8.9-10.6)	11.6 (10.5-12.4)	11.4 (10.9-12.1)	11.9 (11.1-12.6)	9.7 (9.2-10.0)	10.4 (9.3-11.1)
Median ferritin (µg/L)	220 (122-328)	293 (176-405)	213 (129-289)	209 (93-258)	198 (142-281)	195 (131-292)	102 (31-252)	112 (81-202)
Median TSAT* (%)	21 (15-31)	28 (18-36)	22 (15-29)	22 (17-25)	31 (17–47)	23 (18-30)	16 (14-19)	30 (13-35)

Variables with a skewed distribution are presented as median (IQR). Values are presented according to the initial iron regimen. Treatment switches during follow-up were not accounted for in this descriptive analyses.
OD, once-daily; BID, twice-daily; TID, three-times-daily; TIW, three-times-weekly; Hb hemoglobin; TSAT transferrin saturation. *TSAT was calculated using the formula: 100 x serum iron/TIBC (22).

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TABLE 3 Linear mixed-effects models for longitudinal hemoglobin, ferritin and TSAT levels.

Hb levels (g/dL)			
Parameter	β	95% CI	p-value
Intercept	11.48	9.341 – 13.619	**<0.001**
BID vs OD	-1.22	-1.77 – -0.67	**<0.001**
TID vs OD	-0.31	-0.50 – 1.11	0.45
TIW vs OD	-0.54	-1.29 – 0.22	0.16
Time (days)	-0.002	-0.007 – 0.002	0.30
BID * time	0.013	0.006 – 0.020	**<0.001**
TID * time	0.000	-0.009 – 0.009	0.94
TIW * time	0.001	-0.008 – 0.010	0.85
On iron before start PD (no)	-1.12	-1.73 – -0.51	**<0.001**
On iron before start PD (no) * time	0.005	-0.002 – 0.012	0.20
Ferritin (µg/L)			
Para meter	β	95% CI	p-value
Intercept	5.440	4.501 – 6.379	**<0.001**
BID vs OD	-0.131	-0.471 – 0.209	0.45
TID vs OD	0.208	-0.264 – 0.679	0.39
TIW vs OD	-0.349	-0.782 – 0.084	0.11
Time (days)	0.002	0.000 – 0.005	0.07
BID * time	-0.005	-0.009 – -0.001	**0.01**
TID * time	-0.001	-0.006 – 0.005	0.76
TIW * time	-0.001	-0.006 – 0.004	0.61
On iron before start PD (no)	-0.796	-1.133 – -0.459	**<0.001**
On iron before start PD (no) * time	0.000	-0.004 – 0.004	0.88
TSAT (%)			
Para meter	β	95% CI	p-value
Intercept	3.090	2.397 – 3.783	**<0.001**
BID vs OD	-0.106	-0.358 – 0.146	0.41
TID vs OD	0.156	-0.206 – 0.517	0.40
TIW vs OD	-0.026	-0.347 – 0.295	0.87
Time (days)	0.001	-0.001 – 0.003	0.45
BID * time	-0.001	-0.004 – 0.002	0.41
TID * time	-0.002	-0.006 – 0.002	0.39
TIW * time	0.001	-0.003 – 0.005	0.63
On iron before start PD (no)	-0.138	-0.390 – 0.114	0.28
On iron before start PD (no) * time	0.002	-0.001 – 0.005	0.18

Models include a random intercept for each patient. OD dosing was used as the reference category. Time was modeled as a continuous variable (days). Ferritin and TSAT were analyzed on the natural logarithmic scale. Group * time interaction terms represent differences in longitudinal change over time between treatment group and OD. The interaction between time and iron use at PD initiation represents whether the longitudinal trajectory differs between patients who initiated iron supplementation during PD and those who were already receiving iron at PD initiation.
Hb, hemoglobin; CI, confidence interval; OD, once daily; BID, twice daily; TID, three times daily; TIW, three times weekly; PD, peritoneal dialysis; TSAT, transferrin saturation.
Bold values represent statistical significance for the corresponding parameter within the model.

In this subgroup, the median Hb level in this group was 10.6 (IQR 9.8–11.6) g/dL, ferritin was 259 (IQR 149–416) µg/L, and TSAT was 23 (IQR 15–30) %. One third of the patients did not use ESA. According to the Dutch guideline on anemia in CKD from 2015, based on the KDIGO 2012 clinical practice guideline, a trial of oral or intravenous was advised in patients with Hb ≤ 10.0 g/dL and/or renal anemia with symptoms and ferritin <no><200</no> µg/L and TSAT <25% for non-dialysis patients (24). Among the 30 patients, 9 patients (30%) met both ferritin and TSAT criteria for iron therapy.

Discussion

In patients undergoing PD, we showed that TIW and OD oral iron supplementation resulted in similar Hb and iron parameter trajectories, suggesting no additional hematologic benefit from higher dosing frequency. Interestingly, BID dosing was associated with lower baseline Hb levels but a steeper increase over time. Although this finding does not demonstrate superiority of BID therapy, it raises questions about how dosing frequency influences iron absorption in patients with CKD. This is particularly relevant given the altered hepcidin regulation and iron homeostasis characteristics of CKD, which may modify the expected relationship between oral iron dosing frequency and iron absorption.

The rationale for TIW dosing is grounded in hepcidin physiology. Oral iron intake induces a rapid increase in hepcidin, which suppresses subsequent iron absorption (12). Randomized trials in iron-deficient healthy women have shown higher absorption of iron with TIW dosing compared with OD dosing, whereas BID dosing reduced absorption efficiency due to repeated hepcidin stimulation (11, 12). However, patients with CKD exhibit chronically elevated hepcidin levels due to reduced renal clearance and persistent low-grade inflammation (12, 25). In this context, the relative impact of additional hepcidin induction after oral iron intake may be attenuated. The lack of significant differences between TIW and OD dosing in our cohort may reflect this altered regulatory environment, in which increasing dosing frequency does not proportionally enhance effective iron absorption (11, 12).

Descriptive analyses further showed that the TIW group experienced rising Hb levels accompanied by a marked increase in TSAT and only modest ferritin increases, suggesting preferential use of iron for erythropoiesis. In contrast, OD dosing was associated with more pronounced ferritin increases and smaller TSAT changes without a corresponding rise in Hb, indicating greater iron storage. Although these findings should be interpreted cautiously, they are consistent with the hypothesis that less frequent dosing may optimize functional iron utilization in iron deficiency anemia (11, 12). From a clinical perspective, TIW dosing may be particularly relevant, as oral iron therapy is frequently associated with gastrointestinal side effects that impair adherence (12, 26). Reduced dosing frequency lowers pill burden and may improve tolerability without compromising hematologic outcomes, while potentially reducing treatment costs (26, 27). This may be particularly relevant in PD patients, as constipation is a frequent side-effect of oral iron supplementation and can adversely affect PD function and treatment outcomes (26–28).

Interestingly, the steeper increase in Hb in the BID group is not fully explained by existing hepcidin based models. One might

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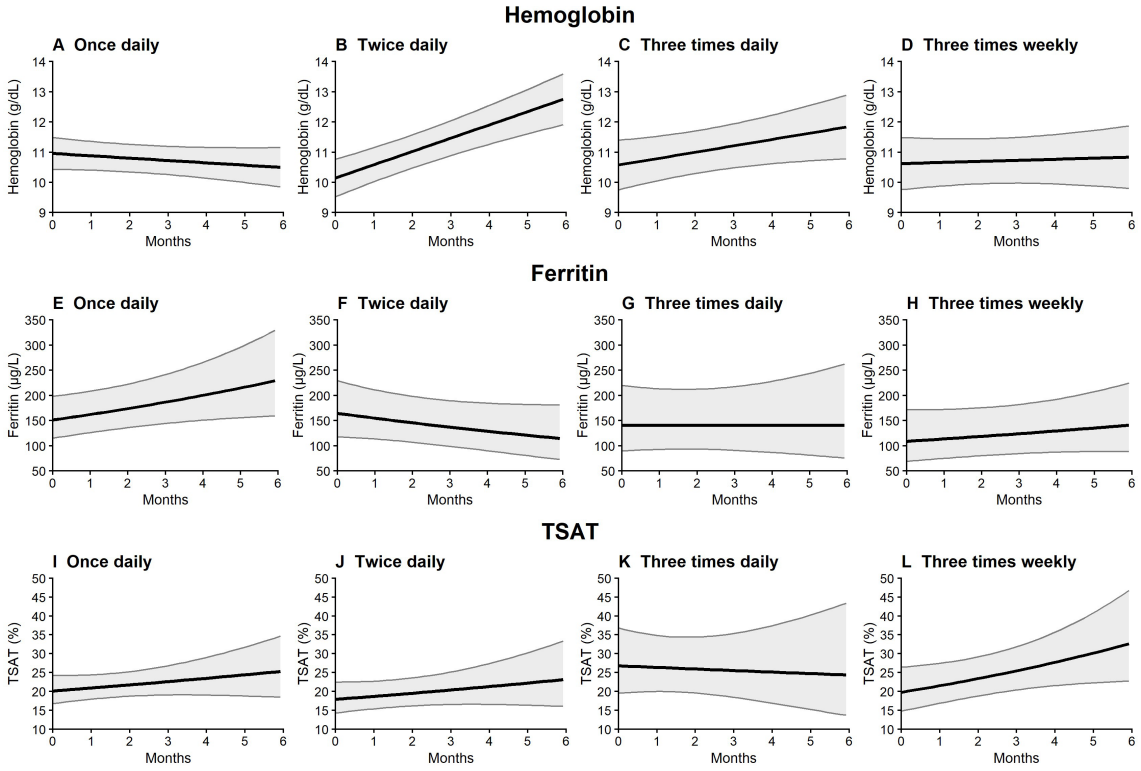


FIGURE 2
Estimated mean trajectories of hemoglobin, ferritin, and transferrin saturation over time according to oral iron regimen. **(A–D)** Show hemoglobin trajectories, **(E–H)** ferritin trajectories, and **(I–L)** transferrin saturation trajectories for once-daily, twice-daily, three-times-daily, and three-times-weekly dosing regimens, respectively. Ferritin and transferrin saturation were modeled on the log scale and are presented as back-transformed estimates on the original scale. Shaded areas indicate 95% confidence intervals.

expect split dosing to reduce fractional absorption due to repeated hepcidin elevations within a single day (12). In PD patients, however, chronic inflammation and reduced renal clearance contribute to sustained elevations in baseline hepcidin levels (12, 25). It is therefore conceivable that the relative increase in hepcidin following an additional daily dose is proportionally smaller when baseline levels are already elevated. In such a context, BID dosing might provide greater cumulative iron exposure without proportionally worsening hepcidin-mediated absorption blockade. Given the observational design and absence of direct hepcidin measurements, these interpretations remain speculative.

This study has several strengths. First, it focuses on patients undergoing PD, an underrepresented population in iron supplementation research, as current guidelines are largely extrapolated from hemodialysis or non–dialysis-dependent CKD populations. Second, the longitudinal design and use of linear mixed-effects models allow appropriate analysis of repeated measurements over time while accounting for irregular follow-up and varying number of measurements per individual. Third, consistent results across sensitivity analyses support robustness. In addition, the study reflects clinical practice over an extended period, enhancing external validity and generalizability to routine PD care. Finally, inclusion of multiple iron-related parameters enables a comprehensive assessment of hematologic response (22).

These findings should be interpreted with consideration of several limitations. The relatively small sample size within the individual dosing groups may have reduced the ability to detect modest differences between regimens. Therefore, these findings cannot be directly generalized or immediately applied to clinical decision making. In addition, this was a single-center, non-randomized study in which iron dosing was determined by the treating nephrologist as part of routine clinical care, potentially introducing treatment-selection bias and residual confounding despite statistical adjustment. As an observational study, the observed associations therefore cannot be interpreted as causal effects of the different dosing regimens. Laboratory measurements were obtained during routine care rather than a standardized protocol, leading to variable follow-up intervals; however, mixed-effects models were used to address this variability. Inflammatory status was assessed using leukocyte count rather than CRP, which may only partially reflect inflammation-related iron regulation, and hepcidin levels were not available, limiting direct mechanistic interpretation (29). Furthermore, adherence to oral iron therapy could not be assessed, which may have contributed to variability in outcomes, although this reflects real-world treatment. Finally, this study primarily assessed laboratory-based outcomes, and therefore the impact on clinically relevant outcomes such as ESA requirements, quality of life and patient reported side-effects could not be established.

Consequently, these findings suggest that an expert consensus process, such as a Delphi process, is a crucial next step to identify gaps in evidence. Incorporating inflammatory markers, hepcidin measurements, and patient-reported outcomes will be essential to better define optimal iron supplementation strategies benefit. Notably, a considerable proportion of patients did not receive iron, despite approximately one-third meeting treatment criteria, suggesting potential underuse consistent with previous CKD studies (16, 17). This highlights that, beyond optimizing dosing strategies, improving identification and initiation of iron therapy may represent an additional opportunity to enhance anemia management in PD patients.

In conclusion, TIW oral iron supplementation resulted in hematologic outcomes comparable to OD dosing, suggesting no added benefit from higher dosing frequency. TIW dosing may represent a more patient-friendly strategy by reducing pill burden without compromising efficacy. BID dosing was associated with faster Hb increases, likely reflecting higher cumulative iron exposure; however, given the observational design, these findings should be interpreted with caution. In addition, iron appears underutilized in this population, highlighting an important opportunity for improvement.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

The studies involving humans were approved by Medical Ethics Committee of the University Medical Center Groningen (UMCG). The studies were conducted in accordance with the local legislation and institutional requirements. The ethics committee/institutional review board waived the requirement of written informed consent for participation from the participants or the participants' legal guardians/next of kin because the need for participant consent was waived by the committee in accordance with the Dutch Medical Research Involving Human Subjects Act (WMO). The manuscript presents research on animals that do not require ethical approval for their study.

Author contributions

RW: Writing – review & editing. IH: Writing – review & editing. NM: Writing – review & editing. AA: Writing – review & editing. IN: Writing – review & editing, Formal analysis. YY: Writing – review & editing. AT: Conceptualization, Data curation, Visualization, Investigation, Writing – review & editing, Formal analysis, Writing – original draft. ME: Conceptualization, Data curation, Visualization, Investigation, Writing – review & editing, Formal analysis, Supervision, Writing – original draft. PR: Writing – review & editing.

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Conflict of interest

Dr. Eisenga has received speaker/consultancy fees from Vifor Pharma, Astellas, Cablon Medical, CSL Behring, GlaxoSmithKline, Medice and Pharmacosmos; has served on the Advisory Board for Cablon Medical, GlaxoSmithKline and Medice; and has received grant support from Cablon Medical and Astellas (all to employer in all instances).

The remaining author(s) declared that this work was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

The author ME declared that they were an editorial board member of Frontiers, at the time of submission. This had no impact on the peer review process and the final decision.

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Supplementary material

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fneph.2026.1989068/full#supplementary-material

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