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Association of Functional and Absolute Iron Deficiency with C-Reactive Protein in Chronic Kidney Disease Patients with Anemia

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KEYWORDS

Chronic Kidney Disease, Anemia, Iron Deficiency, Functional Iron Deficiency, C-Reactive Protein, Inflammation

ABSTRACT:

Background: Chronic kidney disease (CKD) patients with anemia often experience iron deficiency, which can be classified as functional or absolute, complicating management strategies. Inflammation, marked by elevated C-reactive protein (CRP) levels, plays a crucial role in functional iron deficiency by altering iron metabolism and availability.

Objectives: To determine the occurrence of functional (FID) and absolute iron deficiency (AID) in CKD patients with anemia and their association with CRP levels.

Methods: This was a comparative, analytical cross-sectional study conducted in the Department of General Medicine and Nephrology among CKD patients between July 2023 and December 2024. Haemoglobin levels were measured, and patients classified into functional or absolute iron deficiency based on transferrin saturation (TSAT) and ferritin levels, and CRP levels measured in both groups. Analysis was done using Stata v16.

Results: Among the 100 participants, 85% had TSAT levels below 20%, with 58.8% classified as having FID and 41.2% as having AID. Haemoglobin levels were comparable between the groups (8.3 g/dL vs. 8.2 g/dL, $p = 0.759$). Serum creatinine and blood urea levels showed no significant differences between the groups. However, serum ferritin levels were significantly higher in the functional iron deficiency group (144.4 ng/dL vs. 20.7 ng/dL, $p < 0.001$), while serum iron, TIBC, and TSAT were similar. CRP levels were significantly elevated in the FID group (50.8 mg/L vs. 34.6 mg/L, $p = 0.010$), indicating a stronger inflammatory response. CRP levels showed a significant negative correlation with haemoglobin ($r = -0.341$, $p = 0.001$) and a positive correlation with ferritin ($r = 0.288$, $p = 0.004$), suggesting an inflammatory influence on iron metabolism. ROC analysis demonstrated that CRP >41.0 mg/L had moderate discriminatory power for predicting FID (AUC = 0.663, $p = 0.011$).

Conclusion: This study highlights the significant role of inflammation in functional iron deficiency among CKD patients with anemia, as evidenced by elevated CRP levels and their association with ferritin and haemoglobin.

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INTRODUCTION

Chronic kidney disease (CKD) is a global public health concern, affecting approximately 9.1% of the global population,(1) with its prevalence increasing due to aging populations and rising incidences of diabetes and hypertension.(2) Anemia is a frequent and serious complication of CKD, particularly in later stages, contributing to increased morbidity, reduced quality of life, and higher cardiovascular risks.(3) Anemia in CKD is primarily attributed to inadequate erythropoietin production by the failing kidneys, but iron deficiency also plays a critical role in its pathophysiology.(4)

Iron deficiency in CKD can be classified into two forms: absolute and functional iron deficiency.(5) Absolute iron deficiency occurs when iron stores are depleted, reflected by low serum ferritin levels and low transferrin saturation (TSAT), leading to reduced haemoglobin synthesis.(6) Functional iron deficiency, on the other hand, occurs when iron stores are adequate or elevated, but its mobilization for erythropoiesis is impaired due to increased levels of hepcidin, a liver-derived hormone regulated by inflammation.(7) Hepcidin inhibits iron absorption from the gut and iron release from macrophages and hepatocytes by degrading ferroportin, the only known cellular iron exporter.(8)

C-reactive protein (CRP) is a well-established inflammatory marker frequently elevated in CKD patients due to chronic inflammation, oxidative stress, and comorbid conditions such as infections and cardiovascular diseases.(9, 10) Inflammation, as indicated by elevated CRP levels, has been shown to be strongly associated with functional iron deficiency by promoting hepcidin synthesis, thereby impairing iron availability for erythropoiesis despite sufficient iron stores.(11) The interplay between inflammation, iron metabolism, and anemia in CKD is complex and poorly understood, necessitating further investigation into the association between CRP levels and different forms of iron deficiency in CKD patients with anemia.(12) Against this background, this study primarily aimed to assess the occurrence of functional and absolute iron deficiency in anemic CKD patients and their association with CRP levels, while also examining demographic and clinical characteristics, comparing iron deficiency types, and evaluating their relationship with CRP levels.

MATERIALS AND METHODS

This was a hospital based, comparative, analytical cross-sectional study conducted in the outpatient department and/or inpatient wards of the Department of General Medicine and Nephrology, Aarupadai Veedu Medical College and Hospital, Puducherry, India for a duration of 18 months between July 2023 and December 2024. The study was approved by the Institutional Human Ethics Committee (IHEC) with reference number AV/IHEC/2023/057 dated 25/05/2023. The participants were given the Participant Information Sheet (PIS) in their native language, and its contents were verbally explained to ensure their understanding and satisfaction. Enrolment into the study proceeded upon receipt of written informed consent. Patients more than or equal to 18 years of age, of both gender, diagnosed with CKD according to the National Kidney Foundation – Kidney Disease Outcomes Quality Initiative (NKF KDOQI) guidelines, with anemia, defined as haemoglobin levels <12 g/dL for women and <13 g/dL for men were included. However, patients with acute infections; history of cancer or autoimmune diseases; history of liver disease or gastrointestinal bleeding; history of blood transfusions within the past 6 months; history of iron or erythropoiesis-stimulating agent (ESA) therapy within the past 3 months; pregnant or breastfeeding women; on haemodialysis; and with other types of anemia were excluded.

The sample size was calculated based on an expected anemia prevalence of 86.7% in CKD patients,(13) with a 7% relative precision and a 95% confidence level (Z = 1.96), resulting in a minimum required sample size of 100 participants. We used nonprobability sampling technique – purposive sampling/consecutive enumeration to enrol patients. Haemoglobin levels were measured in all recruited CKD patients to confirm the presence of anemia. Following this, demographic and clinical data, including relevant medical history and laboratory findings, were collected. Iron studies were performed on all anaemic patients, which included the assessment of serum iron, total iron-binding capacity, transferrin, and ferritin levels. Based on these iron study results anaemic CKD

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patients were classified into two groups: functional iron deficiency (transferrin saturation <20% and ferritin 100–500 ng/mL)(14) and absolute iron deficiency (transferrin saturation <20% and ferritin less than 100 ng/mL)(15). CRP levels were then measured in both groups.

Statistical analysis: The data collected was manually entered in Microsoft Excel, coded, recoded, and analysed using Software for Statistics and Data Science (Stata) v16 (StataCorp, 2019). Descriptive analysis was presented using numbers and percentages for categorical variables; mean and standard deviation (SD) for continuous variables (based on data normality tested using Kolmogorov–Smirnov test and the Shapiro–Wilk test). Appropriate graphs were used. Chi square test of significance (two-sided) for categorial and independent 't' tests (two-sided) for continuous variables was applied to test for association between independent and dependent study variables. Pearsons's correlation coefficient was estimated to assess the correlation between CRP levels and independent study variables. Receiver operating characteristics (ROC) analysis was conducted to determine the area under the curve of CRP levels to predict functional iron deficiency. Statistical significance was considered at $p<0.05$.

RESULTS

The study included 100 participants with a mean age of 55.2 years (SD: 11.9), the majority (63%) aged between 31 and 60 years, and 34% above 60 years. Males comprised 56% of the study population. Diabetes was prevalent in 82% of patients, while hypertension was observed in 57%. None had liver disease, received iron supplements, or were on dialysis. The mean haemoglobin level was 8.4 g/dL (SD: 1.6), and serum creatinine averaged 5.8 mg/dL (SD: 3.2). Blood urea had a mean value of 96.3 mg/dL (SD: 44.7). Iron study parameters showed a mean serum ferritin level of 82.0 ng/dL (SD: 67.8), serum iron of 31.3 µg/dL (SD: 13.4), total iron-binding capacity of 509.9 µg/dL (SD: 87.1), and transferrin saturation of 17.5% (SD: 8.2). C-reactive protein (CRP) levels averaged 38.5 mg/L (SD: 28.2). The majority (85%) had transferrin saturation below 20%. Regarding iron deficiency classification, 58.8% had functional iron deficiency, while 41.2% had absolute iron deficiency.

The comparison between functional and absolute iron deficiency groups revealed no significant differences in demographic characteristics, including age, gender, diabetes, and hypertension status. The mean haemoglobin levels were comparable between the two groups (8.3 g/dL vs. 8.2 g/dL, $p = 0.759$). Similarly, serum creatinine, blood urea, serum iron, total iron-binding capacity, and transferrin saturation levels did not show significant variations. However, serum ferritin was significantly higher in the functional iron deficiency group (144.4 ng/dL vs. 20.7 ng/dL, $p < 0.001$). Additionally, C-reactive protein (CRP) levels were significantly elevated in patients with functional iron deficiency compared to those with absolute iron deficiency (50.8 mg/L vs. 34.6 mg/L, $p = 0.010$), suggesting a potential association between inflammation and functional iron deficiency.

The correlation analysis between CRP levels and various laboratory parameters revealed a significant negative correlation with haemoglobin levels ($rp = -0.341$, $p = 0.001$), indicating that higher CRP levels were associated with lower haemoglobin levels. Serum ferritin levels showed a significant positive correlation with CRP ($rp = 0.288$, $p = 0.004$), suggesting an association between increased inflammation and elevated ferritin levels. Serum creatinine exhibited a weak positive correlation with CRP levels ($rp = 0.189$), but this was not statistically significant ($p = 0.060$). Similarly, blood urea levels showed no significant correlation with CRP ($rp = 0.034$, $p = 0.739$). Serum iron levels had a weak negative correlation with CRP ($rp = -0.095$, $p = 0.346$), while TIBC had a weak positive correlation ($rp = 0.061$, $p = 0.547$), neither of which were statistically significant. Transferrin saturation also demonstrated a weak negative correlation with CRP ($rp = -0.183$), but this was not statistically significant ($p = 0.069$).

The ROC analysis evaluating the predictive ability of CRP levels for functional iron deficiency demonstrated an AUC of 0.663 (95% CI: 0.549–0.778), indicating a moderate discriminatory power. The optimal CRP cutoff value for predicting functional iron deficiency was determined to be >41.0 mg/L, with a sensitivity of 60.0% and a specificity of 65.7%. The association was statistically significant ($p = 0.011$).

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DISCUSSION

The present study aimed to investigate the occurrence of functional and absolute iron deficiency and their association with CRP levels in chronic kidney disease patients with anemia. Among the 100 participants, 85% had transferrin saturation levels below 20%, with functional iron deficiency (58.8%) being more common than absolute iron deficiency (41.2%). This high prevalence of iron deficiency aligns with previous findings that anemia in CKD patients is often multifactorial, with iron metabolism playing a crucial role.(16) The mean age of patients with functional and absolute iron deficiency did not significantly differ, and the age distribution across both groups was similar, reinforcing that both types of iron deficiency affect CKD patients across different age groups. Gender distribution was identical in both groups (60% male, 40% female), indicating no significant gender predisposition to functional or absolute iron deficiency in CKD patients. These findings contrast with some prior studies suggesting that male CKD patients may have a higher risk of iron deficiency due to increased erythropoiesis demand and inflammation.(12) However, in this study, the equal distribution suggests that other factors, such as inflammation and ESA therapy, may play a more significant role. Diabetes was present in 86% of functional iron deficiency patients and 82.9% of absolute iron deficiency patients, with no significant difference. Hypertension was observed in 60% of both groups. These findings are consistent with studies indicating that diabetes and hypertension are the leading causes of CKD and contribute to the development of anemia through inflammatory mechanisms, increased oxidative stress, and erythropoietin resistance.(17) Given the similar distribution of these comorbidities in both iron deficiency groups, their presence does not seem to be a distinguishing factor between functional and absolute iron deficiency in CKD patients.

The mean haemoglobin levels were 8.3 g/dL in the functional iron deficiency group and 8.2 g/dL in the absolute iron deficiency group, with no significant difference. These values indicate moderate anemia, which is expected in CKD patients.(18) The lack of significant differences suggests that both iron deficiency types contribute similarly to anemia severity. Serum creatinine levels were slightly higher in the absolute iron deficiency group (6.6 mg/dL) compared to the functional iron deficiency group (5.7 mg/dL), though not statistically significant. This trend may indicate that worsening kidney function is more associated with absolute iron deficiency, as reduced kidney function can impair erythropoiesis and iron utilization.(19) However, given the lack of statistical significance, further studies with larger sample sizes are needed to confirm this trend. Blood urea levels were similar in both groups, reinforcing that iron deficiency subtypes do not distinctly impact nitrogenous waste accumulation. This finding is in agreement with prior research indicating that uraemia does not directly differentiate between functional and absolute iron deficiency but rather affects overall anemia progression.(20)

Serum ferritin levels were significantly higher in the functional iron deficiency group (144.4 ng/dL) compared to the absolute iron deficiency group (20.7 ng/dL, $p < 0.001$). This is expected, as ferritin serves as an acute-phase reactant and is elevated in inflammation, even in the presence of iron-restricted erythropoiesis.(18) Absolute iron deficiency, characterized by low ferritin levels, occurs due to true iron depletion, which explains the stark contrast in ferritin levels between the two groups. Serum iron levels were slightly higher in the functional iron deficiency group (32.7 µg/dL) compared to the absolute iron deficiency group (29.0 µg/dL), though the difference was not statistically significant ($p = 0.222$). Similarly, TIBC was comparable between groups. These findings align with previous studies that suggest functional iron deficiency is not characterized by overt iron depletion but rather by impaired iron availability due to inflammation-induced hepcidin elevation.(21)

Transferrin saturation was identical in both groups (14.6%), reinforcing that this parameter alone does not differentiate between functional and absolute iron deficiency in CKD patients. Similar findings have been reported in studies indicating that transferrin saturation levels remain consistently low in both conditions due to limited iron availability for erythropoiesis, whether due to depletion (absolute deficiency) or inflammatory sequestration (functional deficiency).(22)

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CRP levels were significantly higher in the functional iron deficiency group (mean = 50.8 mg/L) compared to the absolute iron deficiency group (mean = 34.6 mg/L, p = 0.010). This finding supports the concept that functional iron deficiency is closely linked to inflammation, as inflammatory states elevate hepcidin levels, restricting iron availability for erythropoiesis despite adequate iron stores.(18) The significantly lower CRP levels in the absolute iron deficiency group suggest that true iron depletion is less influenced by systemic inflammation, reinforcing the pathophysiological distinction between these two types of iron deficiency.(19) The strong association between CRP and functional iron deficiency aligns with previous research highlighting the role of inflammation in anemia management. Bárány et al.(23) (2001) emphasized that inflammatory cytokines, particularly interleukin-6 (IL-6), stimulate hepcidin production, leading to iron sequestration in macrophages and hepatocytes. This process reduces iron bioavailability for erythropoiesis, a hallmark of functional iron deficiency. Given these findings, elevated CRP can serve as an indirect marker of inflammation-driven iron restriction in CKD patients.

A significant negative correlation was observed between CRP levels and haemoglobin levels (rp = -0.341, p = 0.001), indicating that higher CRP levels were associated with lower haemoglobin levels. This finding is consistent with prior studies suggesting that inflammation suppresses erythropoiesis through increased hepcidin production and reduced erythropoietin responsiveness.(24) Panichi et al.(25) (2001) demonstrated that inflammatory markers such as CRP and IL-6 negatively correlate with renal function and haemoglobin levels, further supporting the present study's results. Inflammation not only limits iron availability but also impairs erythropoietin production and erythroid progenitor cell proliferation, exacerbating anemia in CKD patients. Serum ferritin levels showed a significant positive correlation with CRP (rp = 0.288, p = 0.004), suggesting that inflammation contributes to elevated ferritin levels. This finding is expected, as ferritin is an acute-phase reactant that increases in response to inflammation. The positive association between CRP and ferritin has been well documented in CKD patients, as inflammatory states cause increased hepatic ferritin synthesis, complicating the interpretation of iron status.(23) In functional iron deficiency, ferritin levels remain elevated despite iron-restricted erythropoiesis, leading to a paradox where high ferritin does not necessarily indicate adequate iron availability. Elmenyawi et al.(26) (2017) observed a similar relationship, where CKD patients undergoing haemodialysis exhibited elevated ferritin levels despite concomitant anemia and iron deficiency. This underscores the necessity of incorporating inflammatory markers like CRP when assessing iron status in CKD patients, as relying solely on ferritin may lead to misleading interpretations.

Serum iron levels showed a weak negative correlation with CRP (rp = -0.095, p = 0.346), and TIBC exhibited a weak positive correlation (rp = 0.061, p = 0.547), neither of which were statistically significant. These findings are consistent with previous studies indicating that inflammation-mediated iron sequestration does not always manifest as significant changes in serum iron or TIBC levels.(24) Instead, inflammatory suppression of iron release from macrophages and hepatocytes primarily impacts iron utilization rather than systemic iron concentrations. Transferrin saturation also demonstrated a weak negative correlation with CRP (rp = -0.183, p = 0.069), though not statistically significant. This suggests that while inflammation may reduce iron availability, transferrin saturation alone may not be a strong predictor of inflammation-driven iron restriction in CKD patients.

The ROC analysis evaluating CRP as a predictor of functional iron deficiency demonstrated an AUC of 0.663 (95% CI: 0.549–0.778), indicating a moderate discriminatory power. The optimal CRP cutoff value for predicting functional iron deficiency was determined to be >41.0 mg/L, with a sensitivity of 60.0% and specificity of 65.7% (p = 0.011). This suggests that while CRP alone is not a perfect diagnostic tool, it can provide valuable insights into the likelihood of functional iron deficiency in CKD patients. These findings align with Pramugaria et al.(27) (2020), who examined CRP as a potential marker for inflammation-driven anemia and reported similar moderate predictive capabilities. Given that functional

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iron deficiency is driven by inflammatory processes, incorporating CRP measurement in clinical practice could help refine anemia management strategies. However, due to its moderate sensitivity and specificity, CRP should be used alongside other biomarkers, such as hepcidin, to enhance diagnostic accuracy.

The present study has several limitations that should be considered when interpreting the findings. First, the cross-sectional design prevents the establishment of causal relationships between inflammation, iron deficiency, and anemia in CKD patients. A longitudinal study would be necessary to assess the temporal dynamics of these associations. Second, the study was conducted at a single centre which may limit the generalizability of the results to broader CKD populations with diverse demographic and clinical characteristics. Third, while CRP was used as a marker of inflammation, other inflammatory markers such as IL-6 and hepcidin were not assessed, which could have provided a more comprehensive understanding of the mechanisms underlying functional iron deficiency. Additionally, the study did not evaluate erythropoietin levels, which play a crucial role in anemia management and could have further clarified the interaction between inflammation and erythropoiesis. Finally, potential confounding factors such as nutritional status and medication use were not extensively analysed, which may have impacted the study outcomes.

CONCLUSION

The findings highlight that functional iron deficiency is more prevalent than absolute iron deficiency in this population, with significantly higher CRP and ferritin levels, reinforcing the role of inflammation in disrupting iron metabolism. The study also establishes a significant negative correlation between CRP and haemoglobin levels, further emphasizing the impact of inflammation on anemia severity. Moreover, the ROC analysis suggests that CRP levels above 41.0 mg/L can serve as a potential indicator of functional iron deficiency, offering a useful clinical tool for distinguishing between iron deficiency subtypes in CKD patients. However, due to its moderate sensitivity and specificity, CRP should be used alongside other biomarkers, such as hepcidin, to enhance diagnostic accuracy.

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Table 1: Sociodemographic, clinical and laboratory parameters among CKD patients with anemia

		Frequency (N = 100) (n)	Percentage (%)
Age (in years), Mean (SD)		55.2 (11.9)	
Age (in years)	≤30	3	3.0
	31 to 60	63	63.0
	>60	34	34.0
Gender	Male	56	56.0
	Female	44	44.0
Diabetes	Present	82	82.0
	Absent	18	18.0
Hypertension	Present	57	57.0
	Absent	43	43.0
Liver diseases	Present	0	0.0
	Absent	100	100
Iron supplements	Taken	0	0.0
	Not taken	100	100
Dialysis	Present	0	0.0
	Absent	100	100
Laboratory parameters			
Haemoglobin (g/dL), Mean (SD)		8.4 (1.6)	
Serum creatinine (mg/dL), Mean (SD)		5.8 (3.2)	
Blood urea (mg/dL), Mean (SD)		96.3 (44.7)	
Serum ferritin (ng/dL), Mean (SD)		82.0 (67.8)	
Serum iron (µg/dL), Mean (SD)		31.3 (13.4)	
Total iron binding capacity (µg/dL), Mean (SD)		509.9 (87.1)	
Transferrin saturation (%),Mean (SD)		17.5 (8.2)	
C-reactive protein (mg/L), Mean (SD)		38.5 (28.2)	
Transferrin saturation (%)	Less than 20	85	85.0
	≥20	15	15.0
Type of iron deficiency	Functional	50	58.8
	Absolute	35	41.2
SD, Standard deviation			

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Table 2: Association between demographic, clinical and laboratory parameters and type of iron deficiency

		Iron deficiency		P value
		Functional N = 50	Absolute N = 35	
		n (%)	n (%)	
Age (in years), Mean (SD)		55.3 (11.4)	54.8 (12.5)	0.845
Age (in years)	≤30	1 (2.0)	2 (5.7)	0.658
	31 to 60	33 (66.0)	22 (62.9)	
	>60	16 (32.0)	11 (31.4)	
Gender	Male	30 (60.0)	21 (60.0)	1.000
	Female	20 (40.0)	14 (40.0)	
Diabetes	Present	43 (86.0)	29 (82.9)	0.692
	Absent	7 (14.0)	6 (17.1)	
Hypertension	Present	30 (60.0)	21 (60.0)	1.000
	Absent	20 (40.0)	14 (40.0)	
Haemoglobin (g/dL), Mean (SD)		8.3 (1.6)	8.2 (1.7)	0.759
Serum creatinine (mg/dL), Mean (SD)		5.7 (3.0)	6.6 (3.4)	0.174
Blood urea (mg/dL), Mean (SD)		97.7 (43.8)	100.3 (38.7)	0.778
Serum ferritin (ng/dL), Mean (SD)		144.4 (30.5)	20.7 (22.0)	<0.001*
Serum iron (µg/dL), Mean (SD)		32.7 (12.9)	29.0 (14.9)	0.222
Total iron binding capacity (µg/dL), Mean (SD)		514.9 (93.1)	525.4 (80.3)	0.592
Transferrin saturation (%),Mean (SD)		14.6 (4.3)	14.6 (3.3)	0.955
C-reactive protein (mg/L), Mean (SD)		50.8 (30.7)	34.6 (22.4)	0.010*
*Statistically significant at p<0.05 SD, Standard deviation				

Table 3: Correlation between CRP levels and other laboratory parameters

	Pearson's correlation coefficient (rp)	P value
Haemoglobin (g/dL)	-0.341	0.001*
Serum creatinine (mg/dL)	0.189	0.060
Blood urea (mg/dL)	0.034	0.739
Serum ferritin (ng/dL)	0.288	0.004*

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Serum iron (µg/dL)	-0.095	0.346
Total iron binding capacity (µg/dL)	0.061	0.547
Transferrin saturation (%)	-0.183	0.069
*Statistically significant at p<0.05		

Table 4: ROC analysis showing area under the curve of CRP levels to predict functional iron deficiency

	AUC (95% CI)	Cut off	Sensitivity (%)	Specificity (%)	P value
Functional iron deficiency	0.663 (0.549 to 0.778)	>41.0	60.0	65.7	0.011*
*Statistically significant at p<0.05 AUC, Area under the curve; CI, Confidence interval					

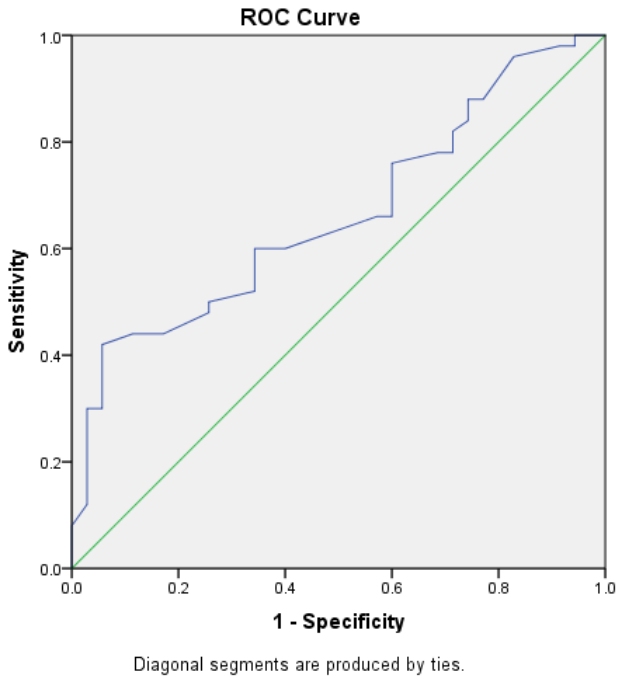

Figure 1: ROC analysis showing area under the curve of CRP levels to predict functional iron deficiency