

FULL PAPER

The assessment of iron deficiency biomarkers in both anemic and non-anemic dialysis patients: A systematic review and meta-analysis

Negar Shahkaramia^a | Maryam Nazari^b | Maryam Milanifard^c | Raheleh Tavakolimoghadam^d | Alireza Bahmani^{e,*}

^aMaster of Operating Room Technology Faculty, Member of Fasa University of Medical Sciences, Fasa, Iran

^bDepartment of Operating Room, Student Research Committee, Mazandaran University of Medical Sciences, Sari, Iran

^cResearcher in Anesthesia, Pain, Molecular and Cell Biology Research Center, Faculty of Medicine, Department of Anatomy, Iran University of Medical Sciences, Tehran, Iran

^dAnatomical and Clinical Pathologist at the Bahar Pathobiology Laboratory, Tehran, Iran

^eDepartment of Emergency Medicine, Ali Ibn Abitaleb Hospital, Zahedan university of Medical Sciences, Zahedan, Iran

All available articles on dialysis patients worldwide with or without iron deficiency were reviewed. Articles published 1 January 2015 to 30 June 2021 were reviewed in Literature databases (PubMed, Scopus, Web of Science, and EBSCO). 163 articles were found in the initial search, the full text of the article was reviewed, and finally, 144 studies were selected. Most existing studies have found that serum ferritin and Hb concentrations are appropriate for defining ID. Several studies have reported transferrin saturation (TSAT) as a complementary diagnostic test. Therapeutic goals in studies selected to treat iron deficiency were to increase the hemoglobin concentration to 10-12 g / dL. Some articles have reported that TSAT varies from 20% to 50% in dialysis patients. The mean value of iron deficiency biomarkers in anemic and non-anemic dialysis patients was assessed. Clinicians should notice hemoglobin and serum ferritin, especially in dialysis patients, and these two biomarkers can be a mark for iron deficiency diagnosis in dialysis patients. In contrast, the mean level of transferrin saturation in dialysis patients is normal.

***Corresponding Author:**

Alireza Bahmani

Email: drbahmani@yahoo.com

Tel.: N/A

KEYWORDS

Iron deficiency biomarkers, anemic dialysis, patients.

Introduction

One of the most common eating disorders in recent decades worldwide is ID, which affects more than 30% of the world's population with anemia, and as a result, is the leading cause of ID disease [1]. Nutrient deficiencies are more prevalent; women with more extended menstrual periods are more likely to have ID. One of the most common complications of ID cancer is reported in 42.6% in various tumors [2]. ID is common in 45% of people with inflammatory bowel disease, 25 to 85% in the chronic kidney [3] and dialysis patients, and 43 to 100% in patients with chronic heart failure [4,5]. Functional Identification Occurs when iron fails to escape from the

macrophages trapped by them, leading to kinetic imbalances. Iron demand increases but iron supply are low. Thus, in functional function, iron is excreted in the reticuloendothelial system. Hepatocytes in these conditions have a deficient concentration of iron [6]. ID can strongly predict mortality in heart and kidney disease [7]. As a result, the diagnosis and treatment of iron deficiency in the clinic are essential. According to the World Health Organization, iron deficiency is directly related to serum ferritin concentration thresholds, which vary by sex, age, and infectious background [1].

However, this explanation is not adequate in many clinical conditions, including inflammation and CKD(e.g., dialysis patients),

in which ferritin concentrations get increased and thus are incorrectly normal when associated with ID [7]. However, in clinical settings, these explanations are practically different. Although the ferritin concentration increases in dialysis patients, it is not enough. Although it is associated with ID, it is naturally incorrect. In previous studies, recommendations for drug ID have been reported differently in each indication; Consensus on results is essential and should be considered appropriately. The present study investigated the relationship between iron deficiency and biomarker symptoms in dialysis patients.

Biomarkers of iron deficiency

Laboratory biomarkers of iron deficiency such as hemoglobin content in reticulocytes (CHr), percentage of hypochromic red blood cells (%HYPO), erythrocyte zinc protoporphyrin (ZPP), soluble transferrin receptor (sTfR), hepcidin, and superconducting quantum interference devices (SQUID), with classical laboratory biomarkers, such as bone marrow iron stores, serum iron, transferrin saturation (TSAT), iron-binding capacity, and serum ferritin are the most critical biomarkers in dialysis patients [8].

Methods search strategy

This systematic review study used Web of Science, Google Scholar, PubMed, Scopus, and EBSCO databases to search for articles. PRISMA checklist was used to search for the studies.

Only Observational and interventional investigations focusing on ID in dialysis patients (Adults) were included. Other type of study design, a sample size of fewer than 10 patients, studies limited to iron deficiency patients with other renal conditions than dialysis were excluded. Selected article filters were performed from 1 January 2015 to 27 November 2021. The text of the article is available in English.

Results

First, 163 articles were identified in the initial search. After removing duplicates, entry criteria for the titles were applied to the remaining 163 articles, and a summary of the remaining articles was reviewed. In the following, the full text of 46 articles were reviewed (Table 1). Finally, nineteen study Studies after removing duplicate articles were selected [9-27].

TABLE 1 the demographic variables and iron deficiency biomarkers of confirmed cases

First author	Publication date	country	mean Age	Mean dialysis duration	Hemoglobin level	TSAT	Ferritin level	sex
Ahmed et al	2020	United Arab Emirates	59.38±13.55	1-3 years 40 (35.7%) 210±7.1	10.18±1.8	31.27±15.19	904.5±721.3	Males/female s 63 (56.25%)/49 (43.75%)
Ala,et al	2018	Iraq	49.8 (±12.3)	3 minutes 36.6 ± 19.3 months	8.9±2.2	26.2±13.6		Male/female 38/32 (54.3/45.7)
Bathla et al	2016	India	54.3±15.1		8.6±1.7			
Cichota et al	2015	Brazil	Control=58.1 ± 14.0 HD patients=56.7 ± 12.7		Control=237.3 ± 51.4 HD patients=178 ± 40.5	Control=205 (139 - 352) HD patient=1248 (892 - 1598) <	Male Control=65.0 HD patients=37.5	

				with iron deficiency (n = 16)= 8.60 ± 1.07 / without iron deficiency (n = 38)= 8.00 ± 1.35 / without anemia (n = 18)= 11.36 ± 1.16	with iron deficiency (n = 16)= 15.48 ± 4.09 / without iron deficiency (n = 38)= 51.90 ± 22.20 / without anemia (n = 18)= 57.46 ± 29.25	with iron deficiency (n = 16)= 484.375 ± 717.82 / without iron deficiency (n = 38)= 1362.53 ± 702.77 / without anemia (n = 18)= 1397.92 ± 623.51	Men with iron deficiency (n = 16)= 15 without iron deficiency (n = 38)= 16 without anemia (n = 18)= 12
Dalimu nthe et al	2016	Indonesia		with iron deficiency (n = 16)= 49.25 ± 11.58 / without iron deficiency (n = 38)= 45.18 ± 12.97 / without anemia (n = 18)= 43.11 ± 17.67			
Deira et al	2016	Spain			10.2 ± 1.7	24.2 ± 8.6	433 ± 385
Deng et al	2017	China	63.90±6.47	35.34±1.298(month)	107±9.55	15.35±3.35	Female 20 (62.5%)
Heidari et al	2014	Iran	hs-CRP C5 mg/l (n = 42)= 49.8 ± 15.8 hs-CRP \5 mg/dl (n = 31)= 50.7 ± 17.2	24 (3-280)	hs-CRP C5 mg/l (n = 42)= 10.3 ± 1.39 hs-CRP \5 mg/dl (n = 31)= 12.5 ± 1.5 (12.7)	hs-CRP C5 mg/l (n = 42)= 549 ± 429 (431) hs-CRP \5 mg/dl (n = 31)= 507 ± 438 (384)	Male/female 50 ± 16.9
Karaboyas et al	2018	USA	62.4 ± 15.1		11.2 ± 1.2	31.3 ± 12.7	Male 56
Kim et al	2021	Korea	65.84 ± 11.91	70.98 (24-130)	10.80 ± 0.51	28.81 ± 6.44	228.18 (49.14-1233.45) Male 39 (51.3)/Female 37 (48.7)
Kurzawa et al	2016	Poland			Hemodialyzed=9.74 ± 1.64 Control=1 5.58± 0.83	Hemodialyzed=26.16 ± 19.66 Control=33.3 8 ± 10.22	Hemodialyzed=179.47 ± 227.46 Control=100.79 ± 114.30
Liu et al	2016	Taiwan	69.09 ± 14.81	8.59 ± 6.26	11.1 ± 1.1	0.3 ± 0.2	432.9 ± 279.3 Male 24 (44%)
Magalhães	2017		0.033		8.8 (7.7: 10.1)		336 (193; 616) Male 0.154
Marzocco et al	2018	Germany	69.9 ± 11.2	45 ± 19	6 weeks12.2 ± 1.5	6 weeks39 ± 18	6 weeks 63 ± 80 (male/female) 11/9
Kristina Petrulienė et al	2017	Lithuania	63.19 ± 15.60	682.2 ± 79.66	98.8±9.8		283.1 139.8 Males 51.2
Plastina et al	2019	Brazil	Functional iron deficiency (n = 65)= 55.0 (51.0 - 59.1)	Functional iron deficiency (n = 65)=	Functional iron deficiency (n = 65)= 11.09	Functional iron deficiency (n = 65)=	Functional iron deficiency (n = 65)= 1010.0 Functional iron deficiency (n = 65)= males/female

Sanai et al	2017	China	66±13, 86/39	10±10	10.4±1.3	28±14	76±127	Male/ female 66±13, 86/39
Targut et al	2021	Turkey	MIS Score < 8 (Quartile 1 and 2; n = 68)= 36 (IQR:43) (Mean R:67.26 R:67.71) MIS Score ≥ 8 (Quartile 3; n = 90)= 69.50 (IQR:16) (Mean R:88.41) R:88.74)	MIS Score < 8 (Quartile 1 and 2; n = 68)= 36 (IQR:43) (Mean R:67.26 R:67.71) MIS Score ≥ 8 (Quartile 3; n = 90)= 69.50 (IQR:16) (Mean R:88.41) R:88.74)	MIS Score < 8 (Quartile 1 and 2; n = 68)= 36 (IQR:43) (Mean R:67.26 R:67.71) MIS Score ≥ 8 (Quartile 3; n = 90)= 69.50 (IQR:16) (Mean R:88.41) R:88.74)	MIS Score < 8 (Quartile 1 and 2; n = 68)= 36 (IQR:43) (Mean R:67.26 R:67.71) MIS Score ≥ 8 (Quartile 3; n = 90)= 69.50 (IQR:16) (Mean R:88.41) R:88.74)	MIS Score < 8 (Quartile 1 and 2; n = 68)= 36 (IQR:43) (Mean R:67.26 R:67.71) MIS Score ≥ 8 (Quartile 3; n = 90)= 69.50 (IQR:16) (Mean R:88.41) R:88.74)	Female MIS Score < 8 (Quartile 1 and 2; n = 68)= 23 (14.6%) MIS Score ≥ 8 (Quartile 3; n = 90)= 37 (23.4%)
Zhou et al	2021	China	55.29 ± 15.82	16.00 (6.00, 64.00)	Baseline=36. 93 ±9.61 Post- therapy=31. 76 (24.06, 39.73)		Male/Female 18 (58.06%)/13 (41.94%)	

One investigation outlined dialysis as >30% decrease in GFR. However, if specified in other studies, dialysis was defined by serum creatinine cut-off. In cichota *et al.* investigation, which defined dialysis

according to decline in GFR, the mean GFR in dialysis patients was 6.7±3.2 ml/min while the control group was 83.8±17.4. Figure 1 summarizes the mean age of dialysis patients.

Study	ES	[95% Conf. Interval]	% Weight	
Ahmed	59.380	53.639	65.121	11.81
Ali	49.800	38.134	61.466	7.65
Bethla	54.400	46.540	62.260	10.25
Belo	71.000	62.128	79.872	9.52
Cichota	56.700	39.129	74.271	4.81
Dalimunteh	45.840	35.252	56.428	8.33
Deng	63.900	41.760	86.040	3.46
Magalheas	50.000	45.241	54.759	12.49
Marzocco	69.900	39.266	100.534	2.04
Petruliene	63.190	53.774	72.606	9.13
Sanai	66.000	54.430	77.570	7.71
Turgut	66.250	55.920	76.580	8.50
Zhou	55.290	36.133	74.447	4.28
D+L pooled ES	58.452	53.721	63.183	100.00

Heterogeneity chi-squared = 32.84 (d.f. = 12) p = 0.001
 I-squared (variation in ES attributable to heterogeneity) = 63.5%
 Estimate of between-study variance Tau-squared = 40.7460

Test of ES=0 : z= 24.21 p = 0.000

FIGURE 1 The mean age of dialysis patients

It should be considered that the majority of included investigations did not report the evidence in iron deficiency and non-iron deficiency dialysis patients. Figure 2 reveals the mean Hemoglobin among 10859 dialysis patients. Figure 3 demonstrated the mean transferrin saturation in 1260 dialysis patients. Figure 4 reports the mean serum ferritin in 1538 dialysis patients. These

statistics demonstrate that the mean hemoglobin in dialysis patients is below the normal range (10.37 g/dl). Transferrin saturation is in the normal range (25.34%). Serum ferritin is above the normal (413.63 ng/mL) in dialysis patients, and these statistics should be considered for iron deficiency management in dialysis patients.

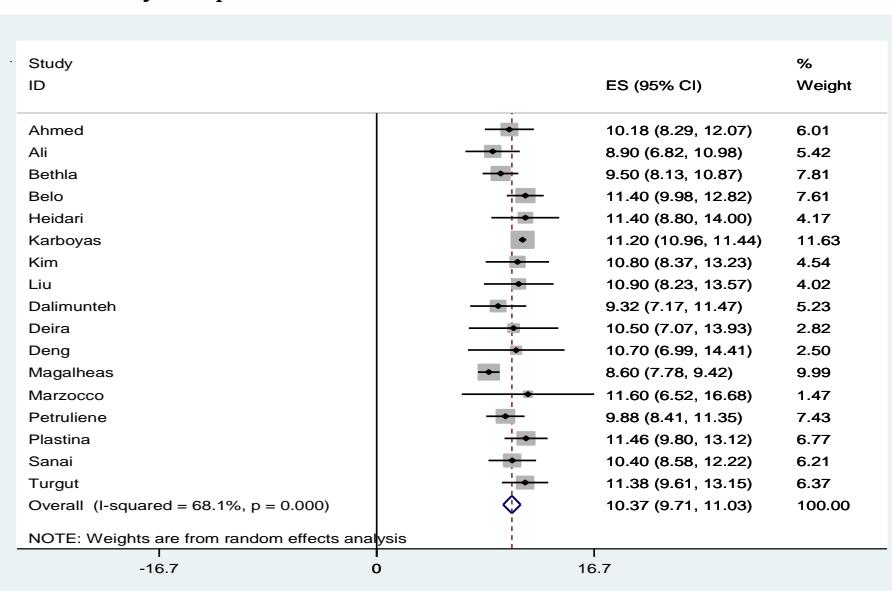
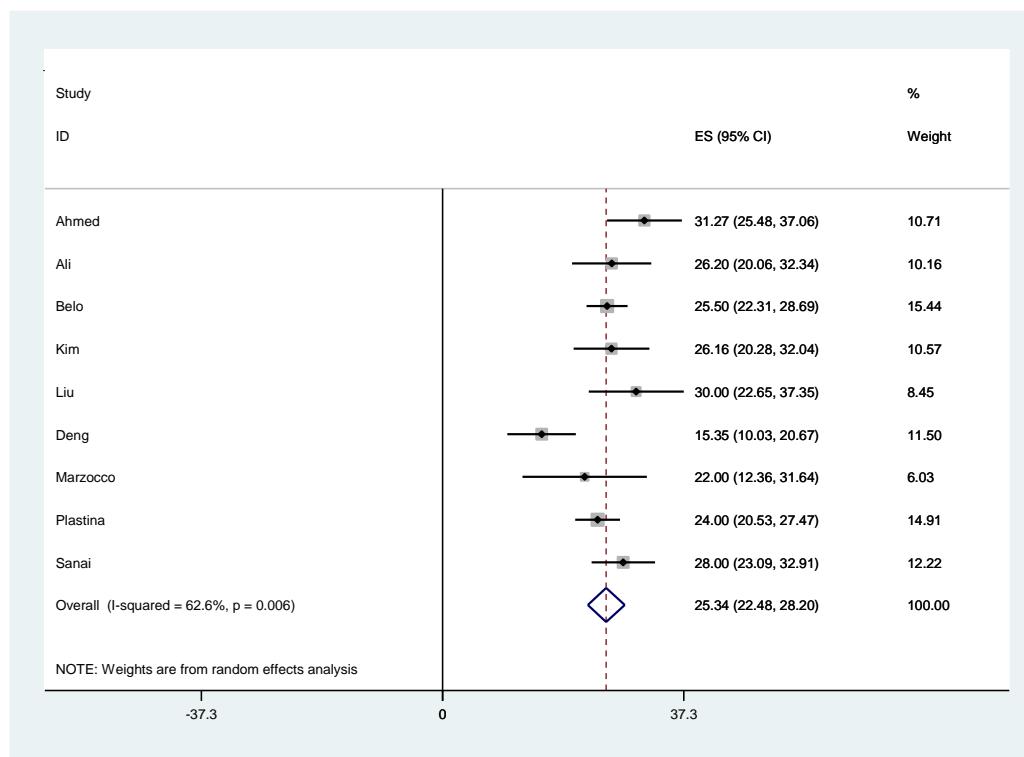
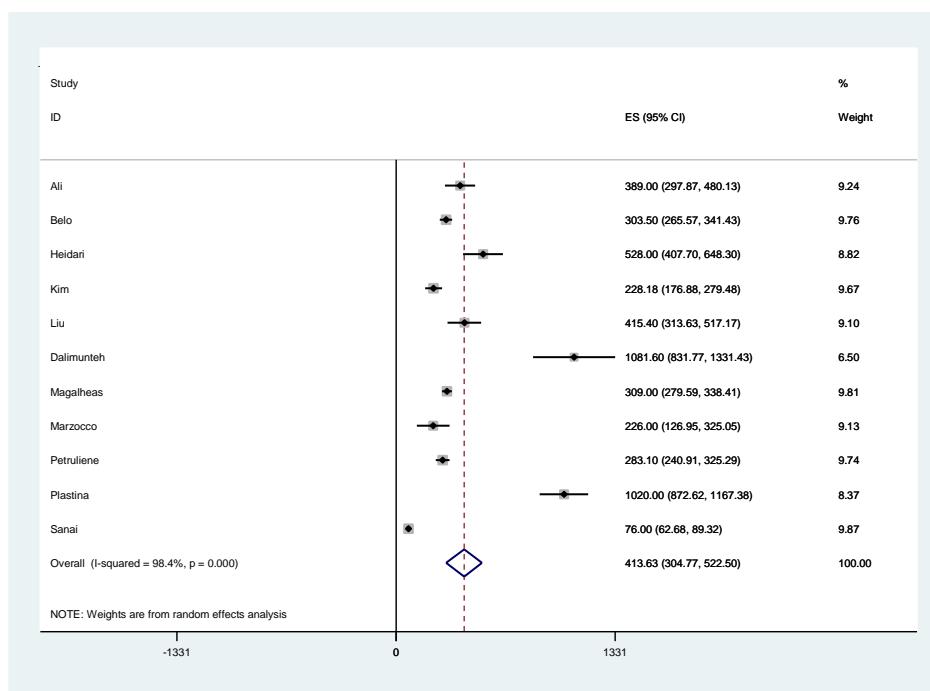
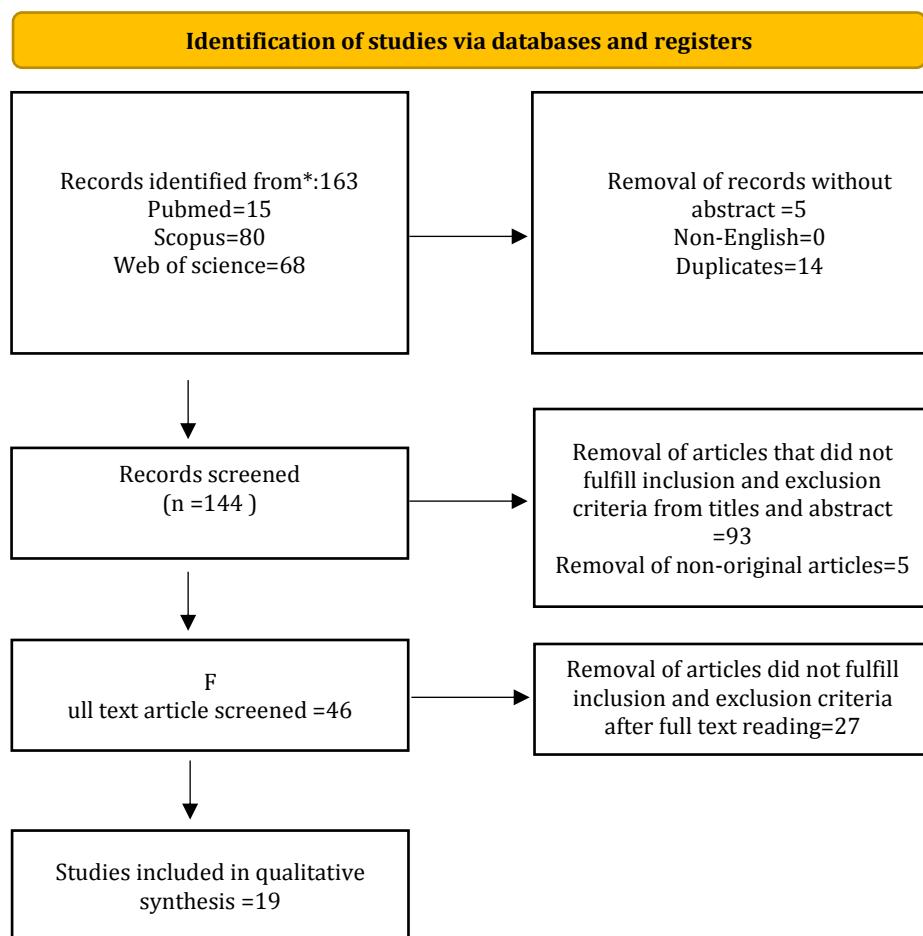


FIGURE 2 The mean Hb IN DIALYSIS PATIENTS (g/dl)

**FIGURE 3** The mean transferrin saturation(%)**FIGURE 4** The mean serum ferritin in dialysis patients

**FIGURE 5** Schematic of research

Discussion

ID is one of the significant risk factors for death and disability worldwide. Approximately 2 billion people are affected by this issue [28-30]. The majority of articles recommend measuring the concentration of hemoglobin and ferritin to diagnose ID [31-33]. Serum ferritin concentrations have been shown to increase irreversible diabetes, chronic alcoholism, macrophage activation syndrome, liver or muscle cytolysis, hyperthyroidism, and some metabolic syndromes [34-36]. According to the above, one of the reasons for the low ferritin level in serum concentration can be ID. The approved level for serum ferritin depends on gender and age [37]. One review in 1988 recommended cut-offs proposed by WHO International Standards: ferritin can rule out ID anemia in

inflammatory and non-inflammatory patients at cut-offs of 70 and 40 mg/L, respectively [38]. In this systematic review, ranges of ferritin defining ID varied across studies from 12 to 800 mg/L for absolute and functional ID. In fact, in studies, the concentration of ferritin 100 mg/L should be further investigated to better define ID. In fact, at the cellular level, the cell is unable to make ferritin without iron. It has been reported that TSAT can be considered a complementary diagnostic method for iron deficiency [39]. TSAT reduction is one of the biomarkers of ID in the early stages, in both absolute and functional ID. Its primary advantage is that the normal range in this biomarker is narrower (between 20% and 40% in adults).

Conclusion

According to our results, the TSAT level for ID diagnosis ranged from 15% to 25%. TSAT alone is not able to differentiate between absolute ID from functional ID. sTfR is a part of the membrane receptor independent of inflammation and can be considered a useful marker in patients with ID. Increased sTfR concentration is directly related to functional ID. Only one article mentioned sTfR tests. As a result, we did not use this biomarker in our meta-analyze. This systematic and meta-analyze review conducted serum ferritin concentration, hemoglobin, and TSAT to calculate the mean value through the 11021 confirmed dialysis patients. In this review, we worked on the meta-analysis of the mean value of iron deficiency biomarkers in anemic and non-anemic dialysis patients. We found that the hemoglobin and serum ferritin should be noticed by clinicians, especially in dialysis patients, and these two biomarkers can be a land mark for iron deficiency diagnosis in dialysis patients. In contrast, the mean level of transferrin saturation in dialysis patients is normal.

Acknowledgements

We would like to thank all the people who helped in preparing and compiling the article and collecting the available data.

Orcid:

Negar Shahkarami: <https://orcid.org/0000-0001-8530-895X>

Maryam Milani Fard: <https://orcid.org/0000-0002-0888-8847>

Alireza Bahmani: <https://orcid.org/0000-0003-2690-9784>

References

- [1] A. Susanabadi, S. Etemadi, M.S. Sadri, B. Mahmoodiyeh, H. Taleby, M. Milani Fard, *Ann. Rom. Soc. Cell Biol.*, **2021**, 25, 2875–2887. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [2] F.E. Sadr, Z. Abadi, N.E. Sadr, M. Milani Fard, *Ann. Rom. Soc. Cell Biol.*, **2021**, 25, 6839–6852. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [3] H. Jahandideh, A. Yarahmadi, S. Rajaeih, A. Ostvar Shirazi, M. Milani Fard, A. Yarahmadi, *JPRI*, **2019**, 31, 1-7. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [4] K. Ghajarzadeh, M. Milani Fard, H. Alizadeh Otaghvar, S.H.R. Faiz, A. Dabbagh, M. Mohseni, S.S. Kashani, A.M. Milani Fard, M.R. Alebouyeh, *Ann. Rom. Soc. Cell Biol.*, **2021**, 25, 2457–2465. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [5] K. Ghajarzadeh, M. Milani Fard, H. Alizadeh Otaghvar, S.H.R. Faiz, A. Dabbagh, M. Mohseni, S.S. Kashani, A.M. Milani Fard, M.R. Alebouyeh, *Ann. Rom. Soc. Cell Biol.*, **2021**, 25, 2449–2456. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [6] K. Ghajarzadeh, M. Milani Fard, M.R. Alebouyeh, H. Alizadeh Otaghvar, A. Dabbagh, M. Mohseni, S.S. Kashani, A.M. Milani Fard, S.H.R. Faiz, *Ann. Rom. Soc. Cell Biol.*, **2021**, 25, 2466-2484. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [7] R. Alimoradzadeh, H. Mirmiranpour, P. Hashemi, S. Pezeshki, S.S. Salehi, *J. Neurology Neurophys.*, **2019**, 10, 1000483. [\[Pdf\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [8] R. Alimoradzadeh, M. Mokhtare, S. Agah, *Iran. J. Age.*, **2017**, 12, 78-89. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [9] S. Etemadi, B. Mahmoodiyeh, S. Rajabi, A. Kamali, M. Milani Fard, *Ann. Rom. Soc. Cell Biol.*, **2021**, 25, 2417-2426. [\[Pdf\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [10] M. Mokhtare, R. Alimoradzadeh, S. Agah, H. Mirmiranpour, N. Khodabandehloo, *Middle East J Dig Dis.*, **2017**, 9, 228-234. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [11] R. Alimoradzadeh, M.A. Abbasi, F. Zabihi, H. Mirmiranpour, *Iran. J. Age.*, **2021**, 15, 524-533. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [12] M.B. Abhari, P.F. Afshar, R. Alimoradzadeh, H. Mirmiranpour, *Immunopathologia Persa*, **2019**, 6, e10. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [13] S.G.R. Mortazavi Moghaddam, G.R. Sharifzadeh, M.R. Rezvan, *Iran. J. Med. Sci.*,

- 2016**, **41**, 322-327. [[Google Scholar](#)], [[Publisher](#)]
 [14] G. Mortazavi Moghaddam, H. Akbari, A.R. Saadatjoo, *Iran J. Med. Sci.*, **2005**, *30*, 110-114. [[Google Scholar](#)], [[Publisher](#)]
 [15] G. Mortazavi Moghaddam, A.R. Saadatjoo, *Iran J. Med. Sci.*, **2014**, *39*, 418-423. [[Google Scholar](#)], [[Publisher](#)]
 [16] F. Zabihi, MA. Abbasi, R. Alimoradzadeh, *Ann. Rom. Soc. Cell Biol.*, **2019**, *1*, 2573-2579. [[Pdf](#)], [[Google Scholar](#)], [[Publisher](#)]
 [17] A. Firoozfar, M. Dousti, *Mag. Civ. Eng.*, **2019**, *90*, 119-129. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [18] A. Sharma, S. Tiwari, M.K. Deb, J.L. Marty, *Int. J. Antimicrob.*, **2020**, *56*, 106054. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [19] A. Zali, M.R. Sohrabi, A. Mahdavi, N. Khalili, M.S. Taheri, A. Maher, M. Sadoughi, A. Zarghi, S.A. Ziai, A.A. Shabestari, M. Bakhshayeshkaram, *Acad. Radiol.*, **2020**, *28*, 1654-1661. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [20] A.J. Rodriguez-Morales, J.A. Cardona-Ospina, E. Gutiérrez-Ocampo, R. Villamizar-Peña, Y. Holguin-Rivera, J.P. Escalera-Antezana, L.E. Alvarado-Arnez, D.K. Bonilla-Aldana, C. Franco-Paredes, A.F. Henao-Martinez, A. Paniz-Mondolfi, *Travel Med. Infect. Dis.*, **2020**, *34*, 101623. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [21] C.C. Lai, T.P. Shih, W.C. Ko, H.J. Tang, P.R. Hsueh, *Int. J. Antimicrob.*, **2020**, *55*, 105924. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [22] C.R. Jutzeler, L. Bourguignon, C.V. Weis, B. Tong, C. Wong, B. Rieck, H. Pargger, S. Tschudin-Sutter, A. Egli, K. Borgwardt, M. Walter, *Travel Med. Infect. Dis.*, **2020**, *37*, 101825. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [23] Z. Cheng, Y. Lu, Q. Cao, L. Qin, Z. Pan, F. Yan, W. Yang, *Am. J. Roentgenol.*, **2020**, *215*, 121-126. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [24] F. Fu, J. Lou, D. Xi, Y. Bai, G. Ma, B. Zhao, D. Liu, G. Bao, Z. Lei, M. Wang, *Eur. Radiol.*, **2020**, *30*, 5489-5498. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [25] F. Shi, J. Wang, J. Shi, Z. Wu, Q. Wang, Z. Tang, K. He, Y. Shi, D. Shen, *IEEE Rev. Biomed. Eng.*, **2020**, *14*, 4-15. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [26] B.K.K. Fields, N.L. Demirjian, H. Dadgar, A. Gholamrezaebehad, *Semin. Nucl. Med.*, **2020**, *51*, 312-320. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [27] J. Liu, H. Yu, S. Zhang, *Eur. J. Nucl. Med. Mol. Imaging.*, **2020**, *47*, 1638-1639. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [28] M. Dousti, A. Firoozfar, *J. Eng. Ind. Res.*, **2022**, *3*, 54-68. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [29] M. Mokhtare, R. Alimoradzadeh, S. Agah, H. Mirmiranpour, N. Khodabandehloo, *Middle East J. Dig. Dis.*, **2017**, *9*, 228-234. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [30] M.B. Abhari, P.F. Afshar, R. Alimoradzadeh, H. Mirmiranpour, *Immunopathologia Persa*, **2019**, *6*, e10. [[Google Scholar](#)], [[Publisher](#)]
 [31] S. Ghorbanizadeh, Y. Raziani, M. Amraei, M. Heydarian, *J. Pharm. Negative Results*, **2021**, *12*, 54-58. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [32] Y. Raziani, B.S. Othman, S. Raziani, **2021**, *Ann. Med. Surg.*, *69*, 102739. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [33] Y. Raziani, B.S. Othman, **2021**, *Veins and Lymphatics*, *10*, 5-10. [[Google Scholar](#)], [[Publisher](#)]
 [34] Y. Raziani, S. Raziani, **2021**, *Journal of Chemical Reviews*, *3*, 83-96. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [35] Z.L. Zhang, Y.L. Hou, D.T. Li, F.Z. Li, *Scand. J. Clin. Lab. Invest.*, **2020**, *80*, 441-447. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [36] S. Khosravani, F. Handjani, R. Alimohammadi, N. Saki, *Int. Sch. Res. Notices*, **2017**, *24*, 6053267. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
 [37] V. Poortahmasebi, M. Zandi, S. Soltani, S.M. Jazayeri, *Adv. J. Emerg. Med.*, **2020**, *4*, e57. [[Google Scholar](#)], [[Publisher](#)]

- [38] N. Shahidi, F. Mahdavi, M.K. Gol, *J. Educ. Health Promot.*, **2020**, 9, 153. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)
- [39] R. Eghdam-Zamiri, M. Khanbabayi Gol, *Iran. J. Obstet. Gynecol. Infertil.*, **2020**, 22, 15-21. [\[Crossref\]](#), [\[Google Scholar\]](#), [\[Publisher\]](#)

How to cite this article: Negar Shahkarami, Maryam Nazari, Maryam Milanifard, Raheleh Tavakolimoghadam, Alireza Bahmani*. The assessment of iron deficiency biomarkers in both anemic and non-anemic dialysis patients; A systematic review and metaanalysis. *Eurasian Chemical Communications*, 2022, 4(6), 463-472.

Link:
http://www.echemcom.com/article_147127.html