Hematological Disorders in the Single-Cell Era
eISSN: 3120-3833
https://hdsce.cultechpub.com/index.php/hdsce

Assessment of Iron Profile in Men with Benign Prostatic Hyperplasia in Owerri, Nigeria

Nduka Favour Amarachi[1,*], Aloy-Amadi Oluchi Chinwe[2], Akujobi Augustine Ugochukwu[3], Akogu Okechukwu[4]

[1,2]Department of Medical Laboratory Science, Faculty of Health Sciences, Imo State University, Owerri, Nigeria

[3,4]Department of Optometry, Faculty of Health Sciences, Imo State University, Owerri, Nigeria

[*]Corresponding author

Abstract

Benign prostatic hyperplasia (BPH) is a chronic, non-malignant enlargement of the prostate gland that affects a large proportion of aging men globally. Although primarily regarded as a urological condition, evidence suggests that systemic metabolic changes, including disturbances in iron metabolism, may play a contributory role in its pathophysiology. This study aimed to assess the iron profile in men with BPH in Owerri, Nigeria, by evaluating serum levels of ferritin, iron, total iron-binding capacity (TIBC), and transferrin. A total of 60 men aged 40 years and above were enrolled, comprising 30 clinically diagnosed BPH patients and 30 age-matched healthy controls.Six milliliters of venous blood samples were collected, and laboratory assays were performed using standard immunoturbidimetric and colorimetric methods. Data analysis was conducted with SPSS version 21.0, employing Student's t-test for group comparisons and Pearson correlation for assessing inter-parameter relationships. Results revealed significantly elevated serum ferritin levels in BPH patients (185.60 ± 19.20) ng/mL compared with controls (144.07 ± 23.13)ng/mL ($p = <0.0001$). Conversely, serum iron, TIBC, and transferrin levels were significantly lower in BPH patients relative to controls ($p = <0.0001$ and $p= 0.022$). Age-based subgroup analysis within the BPH group showed no significant differences in iron profile parameters($p= 0.565$, $p=0.964$, $p= 0.354$ and $p= 0.653$). Correlation analysis further revealed no significant positive associations of ferritin with serum iron, TIBC, or transferrin in BPH patients. These findings suggest that BPH is associated with elevated serum ferritin and reduced circulating iron indices, which may reflect the role of chronic inflammation and altered iron homeostasis in BPH pathogenesis. The study underscores the need for further exploration of iron metabolism as a potential biomarker and therapeutic target in men with BPH.

Keywords

Benign Prostatic Hyperplasia, Ferritin, Iron Metabolism, Total Iron-Binding Capacity, Transferrin, Inflammation, Owerri

1. Introduction

Benign prostatic hyperplasia (BPH) is one of the most common urological conditions affecting men above the age of 40 years, and its prevalence rises steadily with advancing age. Histologically, it is defined as the nonmalignant proliferation of epithelial and stromal cells in the transition zone of the prostate gland, which surrounds the proximal urethra. The consequence of this growth is often obstruction of urinary outflow, resulting in lower urinary tract symptoms (LUTS) such as urinary frequency, nocturia, urgency, weak stream, and incomplete bladder emptying. Although not life-threatening, BPH significantly reduces quality of life, contributes to sexual dysfunction, and imposes substantial healthcare costs worldwide.

Epidemiological studies indicate that nearly 50% of men aged 50 years and above demonstrate histological features of BPH, and the prevalence approaches 90% in men over 80 years. In Nigeria, hospital-based prevalence studies have reported rates between 25% and 60%, with variations attributed to health-seeking behaviors, diagnostic tools, and sociocultural perceptions of urinary symptoms.

The pathogenesis of BPH is multifactorial, involving endocrine changes, stromal–epithelial signaling, growth factors, and chronic inflammation. Testosterone and dihydrotestosterone (DHT) play key roles in prostatic growth, while age-related changes in estrogen/androgen ratios further promote hyperplasia. Inflammatory mediators and oxidative stress have been implicated in disease progression.

Recently, systemic metabolic factors such as dyslipidemia, insulin resistance, and altered iron homeostasis have been suggested as contributors to BPH development. Iron, while essential for oxygen transport, DNA synthesis, and

mitochondrial function, can generate reactive oxygen species (ROS) when in excess, leading to oxidative stress and tissue damage.

Iron metabolism is commonly assessed through ferritin, serum iron, TIBC, and transferrin measurements. Ferritin reflects body iron stores, while serum iron represents circulating iron bound to transferrin. TIBC estimates the capacity of plasma to transport iron, indirectly indicating transferrin levels.

Importantly, ferritin is also an acute-phase reactant, elevated during inflammatory conditions. Since BPH is associated with chronic prostatic inflammation, ferritin levels may be disproportionately raised. Conversely, hypoferremia, reduced transferrin, and reduced TIBC are characteristic of chronic inflammatory states due to iron sequestration by cytokines.

Despite global advances, there is a paucity of data on the relationship between BPH and iron metabolism in sub-Saharan Africa. This study therefore assessed iron indices in men with BPH attending the Federal Teaching Hospital, Owerri.

2. Materials and Methods

2.1 Study Area

This research was carried out at the Federal Teaching Hospital, Owerri, Imo State, Nigeria, a major tertiary healthcare institution that serves as a referral center for southeastern Nigeria. The hospital is equipped with modern diagnostic and laboratory facilities and provides specialized care in various medical disciplines, including urology, internal medicine, and laboratory medicine. The study was conducted within the Department of Chemical Pathology in collaboration with the Urology Unit of the hospital. The geographical location of Owerri is characterized by a tropical climate with distinct wet and dry seasons, which may influence certain biological and environmental variables relevant to clinical research [1].

2.2 Study Design

This research was carried out at the Federal Teaching Hospital, Owerri, Imo State, Nigeria, a major tertiary healthcare institution that serves as a referral center for southeastern Nigeria. The hospital is equipped with modern diagnostic and laboratory facilities and provides specialized care in various medical disciplines, including urology, internal medicine, and laboratory medicine. The study was conducted within the Department of Chemical Pathology in collaboration with the Urology Unit of the hospital. The geographical location of Owerri is characterized by a tropical climate with distinct wet and dry seasons, which may influence certain biological and environmental variables relevant to clinical research [2].

2.3 Study Population

This was a descriptive cross-sectional study designed to assess variations in serum ferritin, serum iron, total iron-binding capacity (TIBC), and transferrin concentrations among patients diagnosed with benign prostatic hyperplasia (BPH) and healthy controls. The study was conducted over a two-month period, from September to October 2024. All eligible participants who met the inclusion criteria and provided written informed consent were consecutively recruited prior to sample collection. The design allowed for comparison between the study group and control group at a single point in time, thereby minimizing temporal variability in laboratory and clinical parameters [3].

2.4 Selection Criteria

Inclusion criteria: Participants eligible for inclusion were men aged 40 years and above. The study group consisted of those clinically diagnosed with BPH, while the control group included apparently healthy individuals with no evidence of prostatic disease or systemic illness [4].

Exclusion criteria: Participants were excluded if they had any known systemic or chronic illnesses such as diabetes mellitus, chronic kidney disease, or malignancies. Additionally, individuals with infectious diseases such as HIV, hepatitis B or C, or syphilis were excluded to prevent confounding effects on serum biochemical parameters. Subjects who declined to give informed consent or were unwilling to comply with study procedures were also excluded [5].

2.5 Sample Collection

For each participant, six (6) milliliters of venous blood was aseptically collected from the antecubital vein using sterile disposable syringes and allowed to clot at room temperature. The samples were then centrifuged at 3000 revolutions per minute (rpm) for five (5) minutes to obtain clear serum. The separated serum was carefully aspirated into clean, labeled plain bottles and stored at –20°C until analysis to maintain biochemical stability. All samples were processed within 24 hours of collection to ensure accuracy and reliability of laboratory results [6].

2.6 Laboratory Analysis

All biochemical analyses were performed in the Chemical Pathology Laboratory of the Federal Teaching Hospital, Owerri, using standardized and validated protocols.

• **Serum Ferritin:** Ferritin concentration was determined using a latex-enhanced immunoturbidimetric method as described by Tietz (2015). The assay is based on the principle of antigen-antibody reaction leading to increased turbidity, which is measured spectrophotometrically.

• **Serum Iron and Total Iron-Binding Capacity (TIBC):** These were determined using the chromazurol colorimetric method, which measures the intensity of color produced when chromazurol reacts with iron ions under acidic conditions (Tietz, 2016).

• **Serum Transferrin:** Transferrin levels were measured by turbidimetric immunoassay according to the method outlined by Doumas (2017).

All reagents were prepared according to manufacturers' instructions, and quality control sera were analyzed alongside samples to ensure accuracy and reproducibility of results.

2.7 Statistical Analysis

Data obtained were entered and analyzed using the Statistical Package for the Social Sciences (SPSS) software, version 21.0 (IBM Corporation, Chicago, USA). Descriptive statistics such as means, standard deviations, and ranges were computed for continuous variables. The independent samples t-test was used to compare mean differences between BPH patients and controls. Pearson's correlation coefficient was applied to evaluate the relationships among biochemical parameters such as ferritin, iron, TIBC, and transferrin. All tests were performed at a 95% confidence level, and a p-value less than 0.05 was considered statistically significant [7].

2.8 Ethical Approval

Ethical approval for this study was obtained from the Research and Ethics Committee of the Federal Teaching Hospital, Owerri (approval number: FTHO/REC/2024/09/22). The study adhered strictly to the ethical principles outlined in the Declaration of Helsinki (2013) for biomedical research involving human subjects. Informed consent was obtained in writing from each participant after the study objectives, procedures, potential risks, and benefits had been clearly explained. All collected data were treated with confidentiality, and participants were assigned identification codes to ensure anonymity throughout the study [8].

3. Results

Table 1. Mean Values of Ferritin, Iron, TIBC and Transferrin in BPH Patients versus Controls (Mean ± SD)

Parameter	BPH (n=30)	Control (n=30)	t-value	p-value
	185.60 ± 19.20	144.07 ± 23.13	7.57	<0.0001*
Ferritin (ng/mL) Iron (µg/dL) TIBC (µg/dL)	70.03 ± 3.95	87.43 ± 3.22	–18.69	<0.0001*
Transferrin (mg/dL)	303.50 ± 11.79	334.57 ± 25.38	–6.08	<0.0001*
	268.00 ± 26.13	280.77 ± 14.13	–2.35	<0.022*

Key:

TIBC - Total Iron Binding Capacity

* - Significant p value

Table 1 shows the mean values of ferritin, iron, TIBC and transferrin in BPH patients compared to control subjects. The mean value of ferritin was significantly higher in BPH patients (185.60 ± 19.20 ng/mL) compared to controls (144.07 ± 23.13 ng/mL) (t = 7.57, p = <0.0001), while that of iron, TIBC and transferrin were significantly lower in BPH patients (70.03 ± 3.95) µg/dL, (303.50 ± 11.79) µg/dL, (268.00 ± 26.13) mg/dL, compared to controls (87.43 ± 3.22) µg/dL, (334.57 ± 25.38) µg/dL, (280.77± 14.13) mg/dL, receptively (t = –18.69, p = <0.0001; t = –6.08, p = <0.0001; t = –2.35, p =0.022).

Table 2. Mean Values of Ferritin, Iron, TIBC and Transferrin in BPH Patients Based on Age (Mean ± SD)

Parameter	40–59 yrs (n=15)	≥60 yrs (n=15)	t-value	p-value
Ferritin (ng/mL)	187.67 ± 24.85	183.53 ± 11.70	0.58	0.565
Iron (µg/dL)	70.07 ± 5.04	70.00 ± 2.65	0.05	0.964
TIBC (µg/dL)	305.53 ± 12.49	301.47 ± 11.10	0.94	0.354
Transferrin (mg/dL)	270.20 ± 32.68	265.80 ± 18.33	0.46	0.653

Key:

TIBC - Total Iron Binding Capacity

Table 2 shows the comparison of mean values of ferritin, iron, TIBC and transferrin between BPH patients aged 40–59 years and those aged ≥60 years.

There were no significant differences in the mean values of ferritin, iron, TIBC and transferrin between BPH patients aged 40–59 years (187.67 ± 24.85) ng/mL, (70.07 ± 5.04) µg/dL, (305.53 ± 12.49) µg/dL, (270.20 ±

32.68) mg/dL, respectively and those aged ≥60 years (183.53 ± 11.70) ng/mL, (70.00 ± 2.65) µg/dL, (301.47 ± 11.10) µg/dL, (265.80 ± 18.33) mg/dL, respectively (t = 0.58, p = 0.565; t = 0.05, p = 0.964; t = 0.94, p = 0.354; t = 0.46, p = 0.653), respectively.

Table 3. Correlation of Ferritin withIron, TIBC, Transferrin in Patients with BPH

Dependent Variable	n	r	p-value
Iron	30	0.15	0.441
TIBC	30	0.001	0.997
Transferrin	30	0.03	0.871

Key:

TIBC – Total Iron Binding Capacity

r – Pearson correlation coefficient

Table 3 shows the Pearson correlation of ferritin with iron, TIBC and transferrin in patients with BPH.

There was a non- significant positive correlation of ferritin with iron (r = 0.15, p = 0.441), TIBC (r= 0.001, p = 0.997) and transferrin (r = 0.03, p = 0.871) in patients with BPH.

4. Discussion

The present study demonstrates that Nigerian men diagnosed with benign prostatic hyperplasia (BPH) exhibit significantly elevated serum ferritin levels accompanied by reduced concentrations of serum iron, total iron-binding capacity (TIBC), and transferrin. These findings collectively indicate a disturbance in iron metabolism among BPH patients, likely mediated by underlying chronic inflammation and altered cytokine activity associated with the pathophysiology of the disease [9].

4.1 Ferritin Elevation and Inflammatory Mechanisms

Ferritin serves as both an intracellular iron storage protein and an acute-phase reactant. Its elevation in BPH patients observed in this study may therefore not only reflect altered iron homeostasis but also the presence of subclinical or chronic prostatic inflammation. Previous research has established that inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) can stimulate hepatic synthesis of ferritin, leading to elevated serum concentrations during inflammatory states.[18,19] The hyperferritinemia observed in this study can thus be interpreted as a biochemical manifestation of chronic inflammatory activity within the prostate tissue. Furthermore, ferritin itself may have a role in modulating immune response by sequestering iron, thereby limiting microbial growth and reducing oxidative stress—an adaptive mechanism that may, paradoxically, lead to functional iron deficiency in the circulation [10].

4.2 Reduced Serum Iron and TIBC

The observed decrease in serum iron and TIBC among BPH subjects mirrors the pattern commonly associated with anemia of chronic disease (ACD). In such conditions, inflammatory mediators stimulate the hepatic production of hepcidin, a peptide hormone that regulates systemic iron balance. Hepcidin acts by binding to and degrading ferroportin—the primary iron exporter present on enterocytes, macrophages, and hepatocytes—thereby reducing iron efflux into the bloodstream.[20,21] The resulting hypoferremia, despite adequate or even elevated total body iron stores, reflects a defense mechanism designed to deprive pathogens of iron but also contributes to reduced erythropoietic availability of iron. The concurrent decrease in transferrin and TIBC observed in this study further supports this mechanism, as transferrin synthesis is typically suppressed by inflammatory cytokines. Thus, the biochemical pattern noted in BPH patients—high ferritin, low serum iron, and low TIBC—corresponds well to inflammation-induced alterations in iron metabolism rather than true iron deficiency anemia [11].

4.3 Influence of Age and Disease Specificity

Interestingly, the alterations in iron-related parameters were independent of patient age in this study population. This observation suggests that the metabolic changes are primarily associated with the disease process itself, rather than age-related physiological decline.[22] Although aging is known to influence iron turnover and ferritin accumulation, the consistency of differences between BPH patients and age-matched controls indicates that chronic prostatic inflammation plays a more dominant role. These findings are consistent with previous studies reporting that inflammatory biomarkers, rather than chronological age, are stronger predictors of altered iron homeostasis in chronic non-communicable diseases [12].

4.4 Clinical Implications

The disturbance in iron metabolism identified in this study carries several potential clinical implications. Firstly, serum ferritin, iron, and transferrin indices could serve as adjunctive markers for assessing the inflammatory status and progression of BPH. Elevated ferritin, in particular, may reflect ongoing inflammatory activity even in the absence of overt infection or systemic illness. Secondly, recognizing altered iron profiles in BPH patients may help clinicians

differentiate between anemia of chronic disease and true iron deficiency anemia, thereby guiding appropriate therapeutic interventions.[23] Moreover, targeted anti-inflammatory or iron-modulating therapies could be explored to mitigate the metabolic consequences of chronic inflammation in BPH. Monitoring these biomarkers might also be beneficial in evaluating treatment response, particularly in patients receiving medical management such as alpha-blockers or 5-alpha-reductase inhibitors, which may indirectly influence inflammatory pathways [13].

4.5 Comparison with Other Studies

The findings of this research align with previous studies reporting elevated ferritin levels in various chronic inflammatory and metabolic disorders, including cardiovascular diseases, metabolic syndrome, and prostate disorders.[24] Elevated ferritin has also been described in patients with chronic kidney disease, rheumatoid arthritis, and other chronic inflammatory states, where it serves as both an indicator of inflammation and a predictor of disease severity.[25] These consistencies reinforce the hypothesis that elevated ferritin in BPH reflects systemic inflammatory activation rather than isolated prostate pathology. However, few studies have specifically explored this association in African populations, making the current study particularly relevant in the context of regional genetic, nutritional, and environmental differences that may influence iron metabolism [14].

4.6 Summary and Future Perspectives

In summary, this study highlights a distinct alteration in iron metabolism among Nigerian men with BPH, characterized by elevated ferritin and decreased serum iron, TIBC, and transferrin. These biochemical changes are likely mediated by inflammation-driven hepcidin activation and cytokine regulation. Future research with larger sample sizes and inclusion of additional biomarkers—such as C-reactive protein (CRP), hepcidin, and IL-6—could provide a more comprehensive understanding of the interplay between inflammation and iron metabolism in BPH. Longitudinal studies may also help determine whether these biochemical markers can predict disease progression or response to therapy, thereby offering new insights into the metabolic dimension of benign prostatic hyperplasia [15].

5. Conclusion

The findings of this study provide valuable insight into the biochemical and pathophysiological alterations associated with benign prostatic hyperplasia (BPH) among men in Owerri, Nigeria. The observation of significantly elevated serum ferritin levels, accompanied by reduced serum iron, total iron-binding capacity (TIBC), and transferrin concentrations, strongly suggests a disturbance in iron metabolism that is closely linked to chronic inflammatory processes inherent in BPH [16].

The elevation of ferritin, a well-known acute-phase reactant, may reflect the presence of persistent low-grade inflammation within the prostate tissue, possibly mediated by cytokines such as interleukin-6 and tumor necrosis factor-alpha. Conversely, the reduction in circulating iron and TIBC mirrors a pattern commonly seen in anemia of chronic disease, where iron is sequestered within the reticuloendothelial system under the regulatory influence of hepcidin [17]. Together, these findings reinforce the concept that inflammatory pathways play a significant role not only in the development and progression of prostatic hyperplasia but also in systemic metabolic alterations affecting iron balance [18].

From a clinical standpoint, evaluating iron-related biomarkers in patients with BPH may serve as a useful adjunct in disease assessment and monitoring. Routine measurement of serum ferritin, iron, TIBC, and transferrin could help clinicians identify underlying inflammatory activity, differentiate between iron deficiency and inflammation-induced alterations, and potentially guide personalized therapeutic approaches. Furthermore, these markers may serve as prognostic indicators for disease progression or response to medical therapy [19].

In conclusion, this study highlights the importance of considering iron metabolism and inflammation as interconnected components in the pathophysiology of BPH. Future research incorporating larger sample sizes and additional inflammatory markers—such as C-reactive protein, hepcidin, and interleukin-6—would further clarify the mechanisms involved and strengthen the evidence base for incorporating iron profile assessments into the clinical management of BPH. Ultimately, a better understanding of these interactions could open new avenues for diagnostic refinement and targeted therapeutic interventions aimed at improving patient outcomes [20].

6. Limitations

6.1 Small Sample Size and Single-Center Design

The study involved a relatively small number of participants (60 men in total) and was conducted at a single tertiary healthcare institution—the Federal Teaching Hospital, Owerri. Although this provided a controlled clinical environment and ensured consistency in diagnostic and laboratory procedures, the limited sample size may restrict the generalizability of the findings to the wider Nigerian population or other demographic groups [21]. Larger multicenter studies involving participants from different geographical and ethnic backgrounds would provide a more representative assessment and strengthen the external validity of the results.

6.2 Did not Assess Cytokines (Il-6, Tnf-α) or Hepcidin

This study did not include measurements of key inflammatory and regulatory biomarkers such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and hepcidin. These markers play central roles in mediating inflammation-induced alterations in iron metabolism and could have provided stronger mechanistic insights into the observed biochemical changes [22]. The absence of these parameters limits the ability to directly link the alterations in ferritin, serum iron, and transferrin to specific inflammatory pathways. Future studies incorporating these biomarkers would offer a more comprehensive understanding of the molecular mechanisms underlying iron dysregulation in BPH [23].

6.3 Nutritional and Dietary Iron Intake not Evaluated

Dietary and nutritional factors, which significantly influence iron status and ferritin levels, were not assessed in this study. Variations in dietary iron intake, vitamin C consumption, and the presence of substances that inhibit or enhance iron absorption (such as phytates or tannins) could have contributed to the differences observed in serum iron and related indices. Failure to account for these factors introduces potential confounding effects that may influence the interpretation of the biochemical results. Therefore, future research should incorporate detailed dietary assessments or nutritional screening to better control for these variables [24].

6.4 Cross-Sectional Design Cannot Infer Causality

The cross-sectional nature of the study limits its ability to establish causality between BPH and the observed alterations in iron metabolism. The design allows only for the identification of associations at a single point in time and does not capture changes in iron or inflammatory markers over the course of disease progression or treatment. Longitudinal or interventional studies would be better suited to clarify the temporal relationships and causal pathways linking prostatic inflammation, cytokine activation, and iron dysregulation [25].

While these limitations do not invalidate the study's findings, they highlight areas where further research is warranted. Future investigations incorporating larger populations, multicenter collaborations, comprehensive inflammatory profiling, and dietary assessments will help to refine the understanding of iron metabolism alterations in BPH and enhance the translational relevance of these findings.

References

[1] McNeal JE. Normal histology of the prostate. Am J Surg Pathol. 1988;12(8):619-633.
[2] Abrams P, Cardozo L, Fall M, et al. The standardisation of terminology in lower urinary tract function. Neurourol Urodyn. 2002;21(2):167-178.
[3] Foster HE, Barry MJ, Dahm P, et al. Benign prostatic hyperplasia: AUA guideline. J Urol. 2018;200(3):612-619.
[4] Berry SJ, Coffey DS, Walsh PC, Ewing LL. The development of human benign prostatic hyperplasia with age. J Urol. 1984;132(3):474-479.
[5] Eke N, Sapira MK. Prostate cancer in Port Harcourt, Nigeria: features and outcome. Niger J Surg Sci. 2002;12(1):7-12.
[6] Ajayi EA, Ajayi AO, Adegun PT. Lower urinary tract symptoms in Nigerian men: prevalence and association with prostate enlargement. Niger Med J. 2013;54(6):400-405.
[7] De Nunzio C, Kramer G, Marberger M, et al. The controversial relationship between benign prostatic hyperplasia and prostate cancer: the role of inflammation. Eur Urol. 2011;60(1):106-117.
[8] Marks LS, Partin AW, Epstein JI, et al. Effects of testosterone replacement therapy on prostate tissue in men with late-onset hypogonadism: a randomized controlled trial. JAMA. 2006;296(19):2351-2361.
[9] Robert G, Descazeaud A, Nicolaïew N, et al. Inflammation in benign prostatic hyperplasia: a 282 patients' immunohistochemical analysis. Prostate. 2009;69(16):1774-1780.
[10] Gacci M, Sebastianelli A, Salvi M, et al. Metabolic syndrome and benign prostatic enlargement: a systematic review and meta-analysis. BJU Int. 2015;115(1):24-31.
[11] Valko M, Rhodes CJ, Moncol J, Izakovic M, Mazur M. Free radicals, metals and antioxidants in oxidative stress-induced cancer. Chem Biol Interact. 2006;160(1):1-40.
[12] Oustamanolakis P, Koutroubakis IE, Messaritakis I, Niniraki M, Kouroumalis EA. Assessment of serum ferritin and iron indices in patients with inflammatory bowel disease. Eur J Gastroenterol Hepatol. 2011;23(2):135-139.
[13] Kell DB, Pretorius E. Serum ferritin is an important inflammatory disease marker, as it is mainly a leakage product from damaged cells. Metallomics. 2014;6(4):748-773.
[14] Weiss G, Goodnough LT. Anemia of chronic disease. N Engl J Med. 2005;352(10):1011-1023.
[15] Dati F, Schumann G, Thomas L, et al. Consensus of a group of professional societies and diagnostic companies on guidelines for interim reference ranges for ferritin assays. Clin Chem Lab Med. 1996;34(6):517-520.
[16] Rimon E, Kagansky N, Kagansky M, et al. Are we giving too much iron? Low-dose iron therapy is effective in octogenarians. Am J Med. 2005;118(10):1142-1147.
[17] Young DS. Effects of drugs on clinical laboratory tests. Ann Clin Biochem. 2000;37(6):758-759.
[18] Kell DB. Iron behaving badly: inappropriate iron chelation as a major contributor to chronic inflammation. BMC Med Genomics. 2009;2:2.
[19] Barton JC, Acton RT. Hepcidin, iron, and infection. Curr Opin Hematol. 2009;16(1):47-52.
[20] Nemeth E, Ganz T. Regulation of iron metabolism by hepcidin. Annu Rev Nutr. 2006;26:323-342.
[21] Nicolas G, Chauvet C, Viatte L, et al. The gene encoding the iron regulatory peptide hepcidin is regulated by anemia, hypoxia, and inflammation. J Clin Invest. 2002;110(7):1037-1044.
[22] Fleming DJ, Jacques PF, Dallal GE, Tucker KL, Wilson PW, Wood RJ. Dietary determinants of iron stores in a free-living elderly population: The Framingham Heart Study. Am J Clin Nutr. 1998;67(4):722-733.

[23] Maggio M, Lauretani F, Basaria S, et al. The relationship between testosterone and molecular markers of inflammation in older men. J Endocrinol Invest. 2005;28(11 Suppl Proceedings):116-119.
[24] Ghio AJ, Ford ES, Kennedy TP, Hoidal JR. The association between serum ferritin and mortality in men with benign prostatic hyperplasia. Urology. 2001;57(1):127-132.
[25] Ganz T, Nemeth E. Iron sequestration and anemia of inflammation. Semin Hematol. 2009;46(4):387-393.