

Prevalence of Severe Anemia (Hb \leq 5 g/dl) in Non-Dialyzed Chronic Renal Failure Patients in the Nephrology and Hemodialysis Department of Point G University Hospital

Seydou Sy^{1,2*}, Magara Samaké^{2,3}, Aboubacar Sidiki Fofana³, Awa Diallo¹, Moctar Coulibaly⁴, Djibril Sy⁵, Atabième Kodio¹, Saharé Fongoro^{1,2}, Mahamane Kalil Maïga^{1,2}

¹Nephrology Department of Point G University Hospital, Bamako, Mali

²Faculty of Medicine of Bamako, Bamako, Mali

³Nephrology Unit of the Fousseyni Daou Hospital, Kayes, Mali

⁴Nephrology Unit of Mali Gavardo Hospital, Bamako, Mali

⁵Department of Internal Medicine, Point G University Hospital, Bamako, Mali

Email: *seydousy2002@yahoo.fr

How to cite this paper: Sy, S., Samaké, M., Fofana, A.S., Diallo, A., Coulibaly, M., Sy, D., Kodio, A., Fongoro, S. and Maïga, M.K. (2021) Prevalence of Severe Anemia (Hb \leq 5 g/dl) in Non-Dialyzed Chronic Renal Failure Patients in the Nephrology and Hemodialysis Department of Point G University Hospital. *Open Journal of Nephrology*, 11, 252-264.

<https://doi.org/10.4236/ojneph.2021.112020>

Received: April 1, 2021

Accepted: May 28, 2021

Published: May 31, 2021

Copyright © 2021 by author(s) and Scientific Research Publishing Inc.

This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).

<http://creativecommons.org/licenses/by/4.0/>



Open Access

Abstract

Introduction: Chronic renal failure (CRF) is defined as glomerular filtration rate (GFR) less than 60 ml/min/1.73m² for at least three (3) months. Anemia is one of its most common complications. Anemia increases the risk factor for cardiovascular mortality by 18% per gram of hemoglobin loss. **Objectives:** To determine the prevalence and characteristics of this severe anemia, to determine the indications for transfusion, the complications related to this anemia, the evolution and the prognosis of these patients. **Materials and Methods:** This was a descriptive study with retrospective data collection over 18 months (January 1, 2017 to June 30, 2018) that included hospitalized CRF patients. Were included, non-dialyzed chronic renal failure patients with Hb \leq 5 g/dl hospitalized during the said period. Not included were chronic renal failure patients with an Hb level \geq 5 g/dl, those followed up and/or hospitalized outside the study period. **Results:** Among 1176 patients, 26 had severe anemia (Hb level \leq 5 g/dl) on CRF, a prevalence of 2.21%. The mean age was 40 years \pm 32.62 with extremes of 15 and 67 years. Seventeen women and 9 men. The etiology of chronic renal failure (CRF) was hypertensive vascular nephropathy in 50% of cases. CRF was end-stage in 18 patients (69.2%). The mean hemoglobin level was 4.10 g/dl \pm 0.64 with extremes of 2 and 5 g/dl. The anemia was microcytic hypochromic in 50% and aregenerative (96.2%). The main symptoms were asthenia in 20 cases (76.9%), dizziness in 20 cases (76.9%), exertional dyspnea in 19 cases (73.1%). Signs of cardiac decompensation

sation (n = 12) were jugular turgor 10 cases (38.5%), hepato-jugular reflux 06 cases (23.1%), mitral insufficiency murmur 06 cases (23.1%). The main complication was left ventricular hypertrophy 17 cases (77.3%). There was no correlation between anemia and sex (p = 0.291), age (p = 0.778), malaria (p = 0.158), etiology of CRF (p = 0.26). The evolution after treatment of anemia was favorable in 19 patients (73.1%), unfavorable in 02 patients (7.7%) and 05 deaths (19.2%). The deaths were of cardiovascular cause: left ventricular insufficiency 04 cases, stroke 01 case. **Conclusion:** Anemia is frequent in patients with chronic renal failure and remains an important risk factor for cardiovascular disease and poor general condition.

Keywords

Severe Anemia, Chronic Renal Failure, Blood Transfusion, Mali

1. Introduction

Chronic renal failure is defined as glomerular filtration rate (GFR) below 60 ml/min/1.73m² for at least three (3) months [1].

Anemia is one of the most frequent complications of uremic patients, affecting their daily life and exposing them to the risk of repeated transfusions [2] [3]. It is known to be a major independent factor of cardiovascular morbidity and mortality in uremic patients [2] [3]. Thus, the destruction of almost all functional glomeruli caused by CRF is proportional to the severity of anemia [3]. Anemia increases the risk factor for cardiovascular mortality by 18% per gram of hemoglobin loss [2] [3]. In a 2007 study in the same department, 95.5% of patients had a hemoglobin level below 8 g/dl [4].

The objectives of this work were to determine the prevalence and characteristics of this severe anemia; to determine the indications for transfusion, the complications related to this anemia, the evolution and the prognosis of these patients.

2. Materials and Methods

This was a retrospective data collection descriptive study. It took place from January 1, 2017, to June 30, 2018 (18 months). The study included patients with CRF hospitalized in the nephrology and hemodialysis department of Point G University Hospital.

Were included, non-dialyzed chronic renal failure patients with a hemoglobin level less than or equal to 5 g/dl hospitalized in our department during the said period. Were not included, chronic renal failure patients with a hemoglobin level \geq 5 g/dl, chronic renal failure patients followed and/or hospitalized in the service outside the study period.

The data were collected through a pre-established individual survey form and the exploitation of the patients' hospitalization records.

The following variables were studied:

Sociodemographic variables: age, gender, occupation, ethnicity, education level, marital status (single, married, divorced, widowed), residence;

- Socioeconomic variables: Three levels were defined: low socioeconomic level, medium socioeconomic level, high socioeconomic level. The high level included government and private sector executives as well as economic operators and army officers; the medium level included government and private sector employees with secondary education and army non-commissioned officers; and the low level included farmers, workers and casual employees in the cities. This classification was made on the basis of income and housewives or homemakers were equated with their spouses.

Clinical variables: functional signs of intolerance of severe anemia (chest pain, dizziness, headache, dyspnea, palpitation, asthenia, impact on physical activity); heart murmur(s) on auscultation;

Biological variables: Complete blood count (CBC); marital status (transferrin saturation coefficient, ferritin, serum iron); calcium, phosphorus, 24-hour proteinuria, folate; B12 vitamins; hemolysis workup (schizocytes, haptoglobin, lactate dehydrogenase (LDH), direct coombs test).

- A renal ultrasound to specify the size and echostructure of the kidneys.

Therapeutic variables: Blood transfusion, iron supplementation (oral or parenteral), vitamin B12 and folate supplementation.

Operational Definitions:

Prevalence is defined as the number of diseases or illnesses present at a given time in a population whether the diagnosis was made in the past or recently.

Severe anemia is defined in this study by a hemoglobin level ≤ 5 g/dl.

Normocytic anemia: anemia associated with a mean blood volume (MBV = 80 fl).

Macrocytic anemia: anemia associated with a MBV ≥ 100 fl.

Microcytic anemia: anemia associated with a MBV less than 80 fl.

Normochromic anemia: anemia associated with a mean corpuscular hemoglobin content (MCHC) (32 - 36.0) g/dl and/or a mean corpuscular hemoglobin level (MCHL) (28 - 32) pg.

Hypochromic anemia (anemia associated with a MCHC < 32 g/dl and/or a MCHL < 28 pg).

Regenerative anemia (anemia associated with a reticulocyte count above 120,000 cells/mm³).

Aregenerative anemia (anemia associated with a reticulocyte count of less than or equal to 120,000 cells/mm³).

Absolute martial deficiency (anemia associated with a ferritin level < 100 μ g/l and a saturation coefficient $< 20\%$).

Functional martial deficiency (anemia associated with a saturation coefficient $< 20\%$, serum iron < 50 μ g/dl and ferritinemia > 1000 ng/ml).

Mechanical hemolytic anemia (anemia associated with red blood cell lysis characterized by the presence of schizocytes in the blood).

Immunologic hemolytic anemia (anemia associated with red blood cell lysis characterized by a positive direct coombs test).

Chronic renal failure (Glomerular filtration rate less than 60 ml/min/1.73m² for at least 3 months).

- Mild chronic renal insufficiency (glomerular filtration rate between 60 and 89 ml/min/1.73m²).
- Moderate chronic renal insufficiency (glomerular filtration rate between 30 and 59 ml/min/1.73m²).
- Severe chronic renal insufficiency (glomerular filtration rate between 15 and 29 ml/min/1.73m²).
- Chronic end-stage renal disease (glomerular filtration rate less than 15 ml/min/1.73m²).
- 24-hour proteinuria (amount of protein excreted in 24-hour urine), it can be:
 - Minimal (proteinuria less than 1 g/24h).
 - Moderate (proteinuria between 1 and 3 g/24h).
 - Massive (proteinuria greater than 3 g/24h).
- Therapeutic failure: It is a situation in which a patient finds himself in which the medical treatment has no or no more therapeutic effect.
- Socio-economic level: refers to the economy and society and their mutual relations.
- Evolution: it is considered favorable if the hemoglobin level increases after treatment or unfavorable if the hemoglobin level is stable or decreased after treatment of the anemia.

To ensure confidentiality of the results, the anonymity of the medical records was strictly respected.

Data were entered on Epi info software and analyzed on SPSS 20 French version. The X2 test was used to compare our proportions with a probability threshold $p \leq 0.05$ as a significant value.

3. Results

Among 1176 patients, 26 had severe anemia (Hb level ≤ 5 g/dl) on CRF, a prevalence of 2.21%. The mean age was 40 years ± 32.62 with extremes of 15 and 67 years. Seventeen women and 9 men. Every second woman was a housewife. Twenty-one patients (80.5%) had a history of hypertension (see **Table 1**).

The mean hemoglobin level was 4.10 g/dl ± 0.64 with extremes of 2 and 5 g/dl. Hypochromic microcytic anemia was observed in every second patient (see **Table 2**). The symptomatology of the anemia was, in decreasing order of frequency, asthenia 20 cases (76.9%), dizziness 20 cases (76.9%), exertional dyspnea 19 cases (73.1%), headache 18 cases (69.2%), anorexia 15 cases (57.7%). The clinical signs are summarized in **Table 3**. The signs of intolerance of anemia (see **Table 4**) are almost the same as those of anemia. Twelve patients (46.15% of cases) presented with cardiac decompensation. The signs of cardiac decompensation (n = 12) were jugular turgor 10 cases (38.5%), hepato-jugular reflux 06 cases (23.1%),

mitral insufficiency murmur 06 cases (23.1%) (cf. **Table 5**). Complications of anemia were left ventricular hypertrophy 17 cases (77.3), high blood pressure (HBP) 8 cases (38.4%), left ventricular failure 4 cases (18.2%) and stroke 01 case (4.5%) (see **Table 6**).

Table 1. Distribution of patients according to history.

Background	Workforce	Proportion in % of total
High blood pressure	21	80.5
Gastro-duodenal ulcer	5	22.7
Bilharzia	4	19
Dysuria	4	19
Pollakiuria	4	19
Burning of the bladder	3	14.3
Hematuria	2	10.5
Lumbar pain	2	10.5
Malaria access	2	10.5
Surgical history	2	10.5
Diabetes	1	3.8

The majority of patients were hypertensive.

Table 2. Distribution of patients by type of anemia on CBC.

Type of anemia	Workforce	Proportion in % of total
Hypochromic microcytic anemia	13	50
Hypochromic normocytic anemia	7	27
Normocytic normochromic anemia	6	23

Hypochromic microcytic anemia was observed in one out of two patients.

Table 3. Distribution of patients by anemia symptomatology.

Symptomatology	Workforce	Proportion in % of total
Asthenia	20	76.9
Vertigo	20	76.9
Dyspnea of effort	19	73.1
Headache	18	69.2
Anorexia	15	57.7
Insomnia	7	26.9
Fatigability	5	19.2
Chest pain	3	11.5
Palpitation	3	11.5

Vertigo, asthenia, exertional dyspnea, and headache were the main signs.

Table 4. Distribution of patients according to signs of anemia intolerance.

Signs of intolerance	Workforce	Proportion in % of total
Asthenia	20	76
Vertigo	20	76
Dyspnea of effort	18	72
Headache	18	72
Tachycardia	10	40
Chest pain	3	12
Orthopnea	2	8

Signs of anemia intolerance were asthenia, dizziness, exertional dyspnea, and headache.

Table 5. Distribution of patients according to signs of cardiac decompensation (n = 12).

Signs of decompensation	Workforce	Proportion in % of total
Jugular turgidity	10	38.5
Hepato-jugular reflux	6	23.1
Mitral insufficiency murmur	6	23.1
Orthopnea	2	7.6
Hepatomegaly	1	3.8

Jugular turgor was the most common sign of decompensation.

Table 6. Distribution of patients by complications of anemia.

Complications of anemia	Workforce	Proportion in % of total
Left ventricular hypertrophy	17	77.3
High blood pressure	8	36.4
Left ventricular failure	4	18.2
Stroke	1	4.5

Ventricular hypertrophy was the most common complication.

The etiology of CRF was hypertensive vascular nephropathy in 50% of cases (see **Table 7**). End-stage CRF was diagnosed in 18 patients (69.2%). Anemia was aregenerative in almost all cases (96.2%). Proteinuria was minimal in 15 cases (57.7%), moderate in 08 cases (30.8%) and massive in 03 cases (11.5%). Renal ultrasound showed kidneys that were reduced in size in 22 cases (84.6%), hyper-echoic in 18 cases (69.2%) and poorly differentiated in 26 cases (100%). Hypocalcemia and hyperphosphatemia were found in 24 patients (92.3%) and 23 patients (88.5%) respectively. Martial assessment performed in 11 patients was normal in 08 patients (73%). Absolute martial deficiency was noted in 02 cases (18%) and functional martial deficiency in 01 cases (09%). Thick blood cell count was positive in 02 cases (10.5%). The presence of schizocytes (n = 14) was found in 01 patient (13%).

There was no correlation between anemia and gender (p = 0.291), age (p = 0.778), malaria (p = 0.158), etiology of CRF (p = 0.26).

Patients were treated with blood transfusion 25 cases (96.2%), injectable iron

18 cases (69.2%) and erythropoietin 3 cases (11.5%) (see **Table 8**).

The clinical course after treatment of the anemia was favorable (Hb > 5 g/dl) in 19 patients (73.1%), unfavorable (stable Hb level) in 02 patients (7.7%) and 05 deaths (19.2%) (see **Table 9**). The deaths were of cardiovascular cause: left ventricular failure 04 cases, stroke 01 case.

4. Discussion

This was a retrospective descriptive study, conducted in the nephrology and hemodialysis department of Point G University Hospital from January 1, 2017 to June 30, 2018. The population of chronic renal failure patients hospitalized during this period was 1176 of which 26 had severe anemia (hb level \leq 5 g/dl), that is, a prevalence of 2.21% of cases.

4.1. Sociodemographic Data

Our patients were young in contrast to Europe and the USA where CRF affects elderly subjects [5]. The average age of our patients was 40 years with extremes of 15 and 67 years. Patients aged 50 years and over were the most numerous (38.5%). The female sex predominated in our study (65.4%), with a sex ratio of 0.53, contrary to studies carried out in Mali and elsewhere [6] [7] [8] [9].

Table 7. Distribution of patients by etiology of chronic renal failure.

Etiologies	Workforce	Proportion in % of total
N.vascular	13	50
N.indeterminate	6	23.1
N.glomerular	5	19.2
N.tubulo-interstitial	1	3.8
Hereditary N.	1	3.8

Hypertensive nephropathy accounted for 50% of the etiologies of CKD.

Table 8. Distribution of patients by treatment received.

Treatment received	Workforce	Proportion in % of total
Blood transfusion	25	96.2
Iron	18	69.2
Erythropoietin	3	11.5

Blood transfusion was the main treatment.

Table 9. Distribution of patients by clinical course at discharge.

Evolution at discharge	Workforce	Proportion in % of total
Favorable	19	73.1
unfavorable	2	7.7
Death	5	19.2
Total	26	100

The evolution was favorable in the majority of cases. Causes of death: stroke 01 cases, left ventricular failure 04 cases.

4.2. Socio-Economic Data

According to occupation, housewives were in the majority (57.7%), followed by farmers (11.5%), pupils (7.6%), students (7.6%), civil servants and workers (3.8%). This result confirms a trend reported by previous studies in the department [7] [9].

4.3. Patient History

High blood pressure was the main antecedent of the patients, representing 80.5% of our population. This CRF-HBP association has been reported in other studies [10] [11].

4.4. Aetiological Aspects

The etiologies encountered in order of frequency were: vascular nephropathy, particularly hypertensive nephropathy (50%), chronic glomerular nephropathy (19.2%), indeterminate nephropathy (23.1%), tubulointerstitial nephropathy (3.8%) and hereditary nephropathy (3.8%). Three studies conducted in the same department in 2010, 2011 and 2016 found the predominance of vascular nephropathy [6] [7] [12]. On the other hand, a study carried out in Côte d'Ivoire concerning 800 cases of CRF identified in the internal medicine department of the University Hospital Center of Treichville in 1990 revealed a predominance of chronic glomerulonephritis (CGN) at 49.1% followed by chronic vascular nephropathy (CVN) at 25.3% [13]. Of 118 patients with vascular nephropathy syndrome, 78.8% were of hypertensive origin [13]. Chronic vascular nephropathy is twice as common in the United States among the black population, as in Canada and France [5]. Diabetic nephropathy is five times more frequent in Japan and four times more frequent in the United States (among the black population) than in France [8]. This difference can be explained by the importance of hypertensive disease in black subjects, late discovery and non-compliance with treatment [8].

4.5. Paraclinical Data

The anemia was microcytic hypochromic in 50% of cases, normocytic hypochromic in 27% of cases, and normocytic normochromic in 23% of cases. It was aregenerative in 96.2% of cases. These results are similar to those of Diallo and Maiga who reported a predominance of microcytic hypochromic anemia in 93.3% and 68.75% of cases respectively [7] [12]. In Cameroon, a study carried out in 1994 on the haematological profile of 47 patients with chronic renal failure, found normocytic normochromic anaemia in 80% of cases and microcytic anaemia in 20% [14]. The normocytic normochromic anemia of CRF is secondary to a deficit in EPO synthesis [2]. The predominance of hypochromic microcytic anemia in our study may be due to other causes such as deficiencies (iron, folate, vitamin C), aluminum intoxication, inflammatory syndrome [14] [15].

The combination of hypocalcemia and hyperphosphatemia is common in end-

stage renal disease [16] [17]. The majority of our patients had hypocalcemia and hyperphosphatemia in 92.3% and 88.5% of cases, respectively.

Among the twenty-six patients in whom 24-hour proteinuria was performed, 57.7% had minimal proteinuria. These results are similar to those of Eyram and Maiga who reported a predominance of minimal proteinuria, 73.06% and 63.3% of patients respectively [6] [7]. This minimal proteinuria may be explained by the high frequency of vascular nephropathy in our study.

The diagnosis of chronic renal failure is made in the face of diminished kidney size (<10 cm) on ultrasound associated with anemia, hypocalcemia, and hyperphosphatemia [15] [17] [18]. Renal ultrasound remains one of the key elements in the diagnosis of chronic renal failure. The kidneys were decreased in size, poorly differentiated and hyperechoic in 84.6%, 100%, 69.2% of cases respectively. Diallo in a study performed in the same department in 2010 reported a frequency of 90%, 100% and 66.7% in respectively for decreased size, poor differentiation and hyperechogenicity of the kidneys [12].

4.6. Consequences of Anemia in Chronic Renal Failure

The signs of intolerance of the anemia in order of frequency were physical asthenia (76%), dizziness (76%), exertional dyspnea (72%), headache (72%), tachycardia (40%), chest pain (12%) and orthopnea (8%). Signs of cardiac decompensation of this anemia were jugular turgor (83.3%), hepato-jugular reflux (50%), mitral insufficiency murmur (50%), hepatomegaly (8.3%).

In terms of chronic complications of anemia, left ventricular hypertrophy (LVH) was the most common, representing 77.5% of cases.

Two studies performed in the same department in 2009 and 2016 reported LVH in 52.1% and 58% respectively [7] [17]. Thus, the more severe the anemia is chronically, the more cardiac involvement is in the foreground. Some studies have indeed shown that the existence of LVH is a determinant of morbidity and mortality during chronic kidney disease (CKD) [19].

Among the factors explaining the occurrence of LVH, in addition to the role of hypertension, the primary role of anemia must be emphasized. Indeed, the role of anemia in cardiac damage is well known when it comes to the appearance or aggravation of angina pectoris, the existence of tachycardia or exertional dyspnea, more insidious as a factor of LVH [20].

4.7. Martial Assessment and Iron Supplementation

Iron deficiency is present in 25% - 37% of chronic renal failure patients [3]. In this study, martial deficiency was present in 27.3% of our patients, of which 18.2% had absolute martial deficiency and 9.1% had functional martial deficiency.

This functional martial deficiency could be explained by increased markers of chronic inflammation secondary to infections, atherosclerosis, vascular or cardiac ischemic lesions, neurodegenerative diseases, dialysis membrane that block

the release of iron by macrophages of the reticuloendothelial system. The result is hypochromic hyposidermic anemia with a collapsed saturation coefficient while ferritinemia is elevated [21]. In our study, martial therapy was initiated in 69.2% of our patients. This result is similar to that of the DiaNE study where 61% of the patients received martial supplementation [21], in contrast to Maiga D. and Maiga S.Y where 27.1% and 40% of the patients received iron supplementation [7] [17]. Among our 18 patients who received iron, 35.3% were treated orally (80 mg ferrous sulfate per day) and 64.7% parenterally (100mg ferrous sulfate per week) with the aim of correcting the anemia of patients with absolute iron deficiency and to have higher iron stores before initiating erythropoietin (EPO) therapy. Due to the acceleration of erythropoiesis, Kidney disease: Improving Global Outcomes (KDIGO) 2012 as well as other societies such as Kidney Disease Outcomes Quality Initiative (KDOQI), National Institute of Health and Clinical Excellence (NICE), and the former European Renal Best Practice (ERBP) 2009 recommend a ferritinemia $\geq 500 \mu\text{g}$ and a saturation coefficient $\geq 30\%$ [22].

4.8. EPO Treatment

Patients who received recombinant human erythropoietin were 11.5%. This result was similar to that of Maiga who found 11.6% [7]. In contrast, in Diallo's 2001 study, only one patient received erythropoietin [23]. In the past, EPO was not readily available in Mali and was very expensive for patients, which made its use problematic.

4.9. Blood Transfusion

In our study, 96.2% of the patients were transfused in the presence of signs of intolerance and the previously mentioned signs of cardiac decompensation. Maiga [7] reports transfusion in 100% of patients with severe anemia associated with signs of intolerance and cardiac decompensation. Nineteen point two percent (19.2%) of the patients died and 7.7% had a stable hemoglobin level despite blood transfusion. This high mortality rate could be secondary to certain complications of transfusion such as post-transfusion malaria and especially hyperkalemia. Transfusions should be reserved for patients with a hemoglobin level of less than 10 g/dl associated with signs of intolerance, cardiac decompensation, diabetic patients, patients with coronary artery disease, as well as subjects whose age is greater than or equal to 65 years. It is desirable that patients be transfused with phenotyped blood as they are candidates for transplantation; otherwise, blood collected less than one week earlier should be used to avoid certain post-transfusion complications such as the appearance of irregular antibodies in the blood and the occurrence of hyperkalemia. During the study period complications such as endotoxin shock, allergic events, hyperhemolysis due to erythrocyte incompatibility were rare because we proceeded to systematically inject methylprednisolone after each transfusion. Citrate overload accidents with hypocalcemia, viral infec-

tions to know Hepatitis B Virus (HBV), Hepatitis C Virus (HCV), and Human Immunodeficiency Virus (HIV) were not found. Diallo [23] found that 12.5% of patients had acute complications such as pruritus (allergy).

4.10. Evolution after Treatment

The evolution was favorable in 73.1% of our patients and unfavorable in 26.9% of patients. This result is similar to that of Maiga who found a favorable evolution in 64% of cases and an unfavorable evolution in 18% of cases [7].

However, our study suffered from some shortcomings that contributed to limit the size of our sample, namely the retrospective nature of the study constituting a bias due to the difficulty in exploiting the medical records, the low socio-economic level of the patients, the high cost of the examinations, the inadequacy of the technical platform, the very fragile health of the patients who consulted at an already advanced stage of CRF and died early before any treatment and the return of the patients to their homes against medical advice.

5. Conclusions

Anemia is common in patients with chronic renal failure and remains an important risk factor for cardiovascular disease and poor general health.

The treatment of choice for renal anemia is the use of erythropoiesis stimulating agents, after correction of any iron, folic acid, and vitamin B12 deficiencies, and even the absence of occult digestive bleeding. Blood transfusion should be reserved for emergency situations of hemodynamic intolerance.

Acknowledgements

We thank all the staff of Point G University Hospital, the hospital of Kayes and the Mali-Gavardo hospital of Sébénicoro.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

- [1] Ponte, B., Martin, P.Y., Pechère, A., Burnier, M. and Guessous, I. (2010) Chronic Renal Failure: Attitudes and Screening Practices in the Absence of Randomized Studies. *Revue Médicale Suisse*, **6**, 1400-1404.
- [2] Bernard, J., Lévy, J.P., Varet, B., *et al.* (1998) *Abrégés d'hématologie*. 9th Edition, Masson, Paris.
- [3] French Health Products Safety Agency (2005) Treatment of Anaemia in Chronic Renal Failure: Arguments. French Health Products Safety Agency, 60 p. www.afssaps.sante.fr
- [4] Abgrall, J.F., Arurot, L., Berthrier, A.M., *et al.* (1995) *Hématologie*. Technique et Documentation, Cachan, 437 p.
- [5] Dysney, A.P.S. (1995) Demography and Survival of Patients Receiving Treatment

- for Chronic Renal Failure in Australia and New Zealand Report on Dialysis and Renal Transplantation Treatment from the Australia and New Zealand Dialysis and Transplant Registry. *American Journal of Kidney Diseases*, **25**, 165-175.
[https://doi.org/10.1016/0272-6386\(95\)90641-X](https://doi.org/10.1016/0272-6386(95)90641-X)
- [6] Eyrarn, Y.A. (2011) Epidemiological and Clinical Profile of Renal Failure in the Nephrology and Hemodialysis Department of Point G University Hospital from 2008 to 2010 [Thesis]. University of Sciences, Techniques and Technologies of Bamako, Bamako.
- [7] Maiga, S.Y. (2016) Clinico-Biological, Evolutionary and Therapeutic Aspects of Anemia during chronic Renal Failure in Non-Dialysis Hospitalized Patients in the Nephrology and Hemodialysis Department of Point G University Hospital [Thesis]. University of Sciences, Techniques and Technologies of Bamako, Bamako.
- [8] Delgourt, C. and Papoz, L. (1996) Le diabète et ses complications dans la population française. Inserm, Paris.
- [9] Maiga, D. (2015) Autoimmune Hemolytic Anemia; Biological Diagnosis and Therapeutic Approaches in the Nephrology and Hemodialysis Department of the Point G University Hospital [Dissertation]. University of Sciences, Techniques and Technologies of Bamako, Bamako.
- [10] Dia, K. (1996) Chronic Renal Failure in the Hospital Environment of Dakar: An Epidemiological and Clinical Study [Thesis]. Cheikh Anta Diop University, Dakar.
- [11] Lengani, A. (1997) Epidemiology of Severe Chronic Renal Failure in Burkina. *Cahier Santé*, **7**, 379.
- [12] Diallo, B. (2010) Biological Diagnosis of Microcytic Anemia Due to Martial Deficiency in Non-Dialyzed Chronic Renal Failure: Interest of TMGH and CCMH in the Nephrology and Hemodialysis Department of the UCU of Point G [Thesis]. University of Sciences, Techniques and Technologies of Bamako, Bamako.
- [13] Diallo, A.D., Niamkey, E. and Yao, B. (1997) Chronic Renal Failure in Côte d'Ivoire: A Study of 800 Hospital Cases. Scientific Article/ann. Biol Clinique, 1997. Manuscript No. 1849, "Public Health", 3 p.
- [14] Youmbissi, T.J., Kenoe, P., Zekeng, L., Ngu, J.-C. and Kapture, N.L. (1994) Hematological Profile of a Group of Chronic Renal Failure Patients in Yaounde. *African Medicine*, **33**, 29-31.
- [15] Diarra, M. (2008) Indication of Blood Transfusion during Chronic Renal Failure in the Nephrology and Hemodialysis Department of the Point G University Hospital. [Thesis]. University of Sciences, Techniques and Technologies of Bamako, Bamako.
- [16] Sadou, M. (2005) Factors of Aggravation of Chronic Renal Failure: An Epidemiological and Clinical Study in the Nephrology and Hemodialysis Department of the Point G National Hospital [Thesis]. University of Sciences, Techniques and Technologies of Bamako, Bamako.
- [17] Maiga, D. (2009) Treatment of Anemia in Chronic Hemodialysis Patients in the Nephrology and Hemodialysis Department of Point G University Hospital [Thesis]. University of Sciences, Techniques and Technologies of Bamako, Bamako.
- [18] Ould, M. (2007) Water Intake during Chronic Renal Failure in the Nephrology and Hemodialysis Department of the Point G University Hospital [Thesis]. University of Sciences, Techniques and Technologies of Bamako, Bamako.
- [19] Kané, I. (2000) The Anemia of Chronic Renal Failure in the Nephrology and Hemodialysis Service of the National Hospital of Point G [Thesis]. University of Sciences, Techniques and Technologies of Bamako, Bamako.

- [20] Harnett, J.D., Kent, G.M., Foley, R.N. and Parfrey, P.S. (1995) Cardiac Function and Hematocrit Level. *American Journal of Kidney Diseases*, **25**, S3-S7.
[https://doi.org/10.1016/0272-6386\(95\)90673-8](https://doi.org/10.1016/0272-6386(95)90673-8)
- [21] Kessler, M., Landais, P., Canivet, E., Yver, L., Bataille, P., Brillet, G., et al. (2009) Is the Management of Anemia in Hemodialysis Patients in France Improving? Results of the DiaNE Study. *Nephrology & Therapeutics*, **5**, 114-121.
- [22] Rostoker, G., et al. (2014) Updates on the Management of Anemia and Martial Deficiency in the Dialysis Patient. *Nephrology & Therapeutics*, **10**, 221-227.
- [23] Diallo, M. (2001) Treatment of Anemia in Patients with Chronic Renal Failure at the Pre-Dialysis and Dialysis Stages in the Nephrology and Hemodialysis Department of the Point G National Hospital [Thesis]. University of Sciences, Techniques and Technologies of Bamako, Bamako.