Coll. Antropol. **33** (2009) 2: 559–566
Original scientific paper

Hypoalbuminemia and Complication Incidence in Hemodialysed Uremic Patients

Gordan Galić[1], Monika Tomić[1], Krešimir Galešić[1], Ante Kvesić[1], Martina Šoljić[1], Vladimir Mozetić[2], Zdravka Lončar[1], Anton Maričić[2] and Željko Martinović[3]

[1] Clinical Hospital »Mostar«, Mostar, Bosnia and Herzegovina
[2] Universityl Hospital Center »Rijeka«, Rijeka, Croatia
[3] Franciscan Hospital »dr. fra Mato Nikolić«, Nova Bila, Bosnia and Herzegovina

A B S T R A C T

Discussions whether hypoalbuminemia is just a marker for the malnutrition-inflammation syndrome as well as for the increased morbidity and mortality of those patients or is it an etiological factor, are becoming more and more intense. In this research of the relation between hypoalbuminemia and the complications that threaten the vascular access with special reference to infection, and consequently to the life of the patients treated with chronic haemodialysis, we have chosen 120 patients with terminal renal insufficiency (ESRD) treated at the Clinical Hospital Mostar by chronic haemodialysis. The chosen patients for this study were observed throughout a time period of 18 months. Only the patients who, at the moment of starting the research did not exhibit either a local or a systemic infection, as well as no signs of any other complication that might have endangered the vascular access and consequently the life of the patient, were selected. From the 120 (100.0%) patients, 86.8% of them had a serum albumin level below 40,0 g/L. By analysing the research results of the clinical material, it has been established that in patients with serum albumin level below 40.0 g/L, the infection incidence was significantly higher than in those patients with the albumin level above 40.0 g/L (χ^2 = 7.215 P = 0.0077). The complication incidence is significantly higher (χ^2 = 9.92 P = 0.0022) among the patients with serum albumin level below 40.0 g/L, than in those patients with higher serum level. Among the patients with a serum albumin level lower than 40,0 g/L, the sepses incidence was significantly higher (χ^2 = 4.77 P 0.03), than among those patients with a serum albumin level above this value. However, the difference in incidence of local infection of the vascular access between the group of patients with a serum albumin level below 40.0 g/L and those patients with albumin level above this value is not significant (χ^2 = 0.65 P = 0.69). The total infection incidence in the 120 observed patients was 3.8 episodes per 100 patient months, and within the parameters mentioned by other authors.

Key words: *chronic renal failure, haemodialysis, hypoalbuminemia, complications*

Introduction

The incidence of mortality in patients undergoing the treatment of chronic haemodialysis is many times higher than in the rest of population[1–7]. It has been long noticed that the morbidity and mortality incidence of dialysed patients with pronounced hypoalbuminemia is higher than in those patients with normal serum albumin concentration[8–10]. Recently, the relation between hypoalbuminemia and the morbidity and mortality of the patients treated by chronic haemodialysis has become the subject of more intense research[8,10–14]. The research is twofold: 1. establishing the cause of hypoalbuminemia and 2. determining the possible role of hypoalbuminemia in the morbidity and mortality. Regarding the latter research point raised, whether hypoalbuminemia in those patients is just a serological marker of inflammation and malnutrition as well as the mortality marker, or whether it is in a cause-effect relation with the higher morbidity and mortality rate[12,15–17], becomes the subject matter.

About 50% of dialysed patients have a serum albumin concentration lower than 4.0 g/L[8]. There are two efficient factors that in these patients directly influence the decrease of the serum albumin concentration in the affected organism, especially in uremic patients: inflam-

Received for publication March 15, 2009

mation and malnutrition[18,19,20]. These two factors act in synergy in such a way that the inflammation intensifies the effect of the malnutrition (the malnutrition-inflammation syndrome)[8,10].

In these patients, hypoalbuminemia is in a positive correlation with the morbidity and mortality[1,12,21] and they are, according to the reported findings of Lowrie et al.[9] and Kaysen et al.[22], in 20 times greater death risk than those patients with a normal serum albumin concentration. Khan and Catto[23] claim that dialysed patients with serum albumin concentration between 30.1 and 35.0 g/L, are exposed to 5 times higher death risk than those patients in which the concentration is between 40.1 i 45.0 g/L. Powe and associates[10] have found that dialysed patients with an albumin concentration below 3.5 mg/dl, are at a 66% greater risk of septicemia than those patients with serum albumin higher levels.

Hypoalbuminemia in patients treated with chronic haemodialysis is in correlation with morbidity and mortality, but also with the inflammation markers such as proteins of the acute inflammation phase (CRP)[24,25,26] and cytokines[27], thus sometimes regarding albumin as a negative protein of the inflammation acute phase[1,11,28]. In the majority of dialysed patients death is caused by cardiovascular disorders[1,7,29]. Inflammation on dialysed patients has two effects: hypoalbuminemia and damaging of the endothels' vascular structures. In the creation of atheromatous vascular changes, co-morbidity, such as, diabetes, hyperlipidemia and hypertension[9,16], have their effects, with additional participation of oxidative stress and endothelial dysfunction of the cardiovascular system initiated by inflammation[30]. Stenvinkel et al.[28] consider that inflammation and malnutrition are frequently used as synonyms for hypoalbuminemia[22,31], which work in synergy in the creation of the conditions for atherosclerosis, which would then establish the conditions for increased morbidity and mortality of this population of patients.

Put forward, as considered by Don and Kaysen[12], points to the fact that the hypoalbuminemia problem in the patients treated with chronic haemodialysis is more complex than it is comprehended and is frequently related to malnutrition in such a way that sometimes malnutrition and hypoalbuminemia are sometimes regarded as synonyms[22,31]. The fundamental question that remains to be answered is whether hypoalbuminemia in dialysed patients is just a serological marker of inflammation and malnutrition (*malnutrition – inflammation complex syndrome*) as well as strong marker (*predictor*) of mortality, or whether is it in a cause-effect relation with increased morbidity and mortality[8,21]. Throughout our research we wanted to give a contribution to the responses to the questions risen above.

Patients and Methods

The patients with terminal renal insufficiency have been treated for 40 years in the Hospital Dialysis Centre of the Clinical Hospital Mostar by chronic haemodialysis. In the last decade, the vascular approach in the Clinical Hospital Mostar has been established on the majority of patients by using the arteriovenous fistula, primarily on the upper extremities in the wrist area (Bresci-Cimino fistulae), and if possible, on the non-dominant arm. The vascular approach by tetrafluorethylene graft is rarely established, and the vascular access on the lower extremities is established as an exception, that is, when all other possibilities of using upper extremities have been exhausted.

For the purpose of establishing a relation between hypoalbuminemia and the complication incidence that endangers vascular access, especially infection, and then the life of the dialysed person, we have chosen 120 patients treated through the programme of chronic dialysis to monitor, who had AV-fistulae established on their forearm. The patients were chosen randomly, and only those who did not exhibit any signs of local infection of the arteriovenous access or septicemia at the moment of starting the research, as well as no signs of any other complication which could endanger the vascular access, and consequently the life of the patients. The patients have been observed throughout the time period of 18 months and apart from the blood tests, they were also tested for the albumin levels in serum at 3 month intervals.

Descriptive statistics has been used in the basic evaluation of the numerical data in order to determine the mean values of the standard deviation and the span. The categorical data is shown by the frequency of appearance, as well as its proportion, that is, its percentage. In the statistical analysis we have used the χ^2 test. The statistical significance considered for the level of importance is $P \leq 0.05$, that is with a 95% of relevance threshold. For the analysis of the collected data we have used the application program Statistics, version 8.0 whereas the graphic presentation has been made in Excel.

Results

The distribution of patients according to age and sex is shown in table 1, whereas table 2 illustrates the pathological conditions that were the cause of the terminal re-

TABLE 1
DISTRIBUTION OF PATIENTS ACCORDING TO AGE AND SEX

Age	Male	Female	Total
11–20	–	1	1
21–30	3	–	3
31–40	8	6	14
41–50	10	7	17
51–60	23	13	36
61–70	10	7	17
71–80	15	13	28
81–90	–	3	3
91–100	1	–	1
Total	70	50	120

TABLE 2
PATHOLOGICAL CONDITIONS WHICH CAUSED TERMINAL KIDNEY FAILURE (ESRD)

Cause of renal isufficiency	Total of patients
Hypertensive nephrosclerosis	35
Interstitial nephritis	27
Glomerulonephritis	21
Diabetic nephropathy	12
Chronic pyelonephritis	11
Polycystic kidney	6
Obstructive uropathy	5
Unknown	3
Total	120

nal insufficiency (ESRD). On table 2 it can be seen that the most frequent cause of kidney failure was hypertensive nephrosclerosis, then interstitial nephritis, followed by glomerulonephritis, whereas other pathological conditions were considerably less represented. Table 3 illustrates comorbidity. Some of those comorbidity conditions, such as diabetic nephropathy, as it could be concluded, contribute as a self-working agent to increased morbidity and mortality of dialysed patients.

Table 4 shows the original places of infection development, and then septicemia, as well as the number of patients with infection. The number of patients in which the causative agent of infection was isolated, that is, the type of microorganism, is shown in the right column of the aforementioned table. Hence, it can be seen that in some patients with infection the causative agent has not been isolated. As it is evident from the presented data, the most frequently present is the respiratory infection in the patients, the infection of the vascular access area follows, that is, local infection of the vascular access, which then, alongside other things mentioned in the table, represented the original point of sepses.

TABLE 3
THE INCIDENCE OF CONCOMITANT DISEASES

Comorbidity	Number of patients	%
Cardiovascular disease	51	42.5
Diabetes	12	10.0
Carcinoma	5	4.2
Others	2	1.7

The results of requested blood tests indicative to the observation and treatment of patients treated with chronic haemodialysis, among which are albumins, ferritin as well as calcium in serum, are illustrated in table 5. The highest and the lowest levels, as well as the mean value of the results with standard deviation of all parameters are presented, and they were established in all patients included in the research.

The values of serum albumin level and the distribution of patients according to the relation between those findings and the infection incidence are illustrated in table 6. Among those 48 (100.0%) with an infection, 47 (97.2%) of them had hypoalbuminemia, and only 1 (2.1%) had serum albumin concentration above 40.0 g/L. The difference between those two values is 95.1%. From those 72 (100.0%) patients, 57 (79.2%) had a serum albumin level below 40.0 g/L, and 15 (20.8%) above this level. The difference between these two values is 58. 4%.

The research results of the ratio of patients with an albumin level below 40.0 g/L and those above the aforementioned value among the patients with an infection are 95.1. This ratio among the patients without an infection is 58.4. These two results illustrate the relation between hypoalbuminemia and an infection in patients treated with chronic haemodialysis. The difference between the aforementioned two values, that is, 95.1 and 58.4 is 36.7, and shows the relation between hypoalbuminemia and the infection incidence in the patients. It is

TABLE 4
THE ILLUSTRATION OF THE PRIMARY SPOT OF INFECTION AND THE KINDS OF ISOLATED MICROORGANISMS IN HYPOALBUMINEMIC PATIENTS WITH INFECTION

Primary spot of infection	Number of patients	Isolated microorganisms	Number of patients with microorganism	%
Vascular access	15	*Staphylococcus epidermidis*	4	(26.6)
		Staphylococcus aureus	6	(40.0)
Infection of respiratory system		*Streptococcus pneumoniae*	1	(6.2)
	16	*Klebsiella pneumoniae*	2	(12.4)
Infectio of urinary system		*Escherichia coli*	3	(3 3.3)
	9	*Enterococcus species*	2	(22.2)
Infectio of skin and soft tissues		*Streptococcus pypgenes*	1	(25.0)
	4	*Streptococcus aureus*	2	(50.0)
		Staphylococcus epidermidis	1	(25.0)
Endocarditis	2	*Staphylococcus epidermidis*	2	(100.0)
Others	2	–	–	–

TABLE 5
RESULTS OF BLOOD TESTS (THE LOWEST, THE HIGHEST AND MIDDLE VALUES AS WELL AS STANDARD DEVIATION) OF VARIABLES

Variable	Minimum	Maximum	Arithmetic mean	Standard deviation
Serum proteins g/L	30.6	85.1	63.46	6.93
Serum albumins g/L	20.2	50.1	36.6	3.8
C-reactive proteins mg/L	0.1	191.8	12.31	21.05
Serum feritin μg/L	4.43	111.9	169.05	206.58
Hemoglobin g/dL	59	160	102.61	17.83
Urea prior to dialysis mmol/L	5.2	44	21.3	5.16
Serum calcium mmol/L	5.24	13.12	8.94	0.84

TABLE 6
DISTRIBUTION OF PATIENTS ACCORDING TO THE RELATION BETWEEN THE ALBUMIN LEVEL IN THE SERUM AND INFECTION INCIDENCE

Albumin level in serum (g/L)	Number of patients			
	with infection	without infection	Total	%
< 30.0	5	–	5	4.17
30.1–35.0	27	2	29	24.17
35.1–40.0	15	55	70	58.33
40.1–45.0	1	15	16	13.33
Total	48 (40.0%)	72 (60.0%)	120	100.0

The results of examinees comparison according to the relation of the albumin level in serum and incidence of infection

evident that there is a significant difference considering the frequency of infection in patients with the albumin level in serum < 40 and those > 40 (χ^2 = 7.215 P = 0.0077).

Table 7 shows the type and complications incidence that can endanger the vascular access and consequently the life of a dialysed patient. Throughout the time period of 18 months of observation 120 (100.0%) patients, 59 (49.16%) of them had complications. Of those 59 (100.0%) patients with complications, 58 (98.3%) of them, as it can be seen from table 7, had serum albumin level below 40.0 g/L, that is, they had hypoalbuminemia.

The results of the comparison relation between the albumin level and the complication incidence are presented in table 7. It is evident that there is a significant difference regarding the complication frequency in patients with the serum albumin level < 40 and those > 40 (χ^2 = 9.92 P = 0.0022).

The patients have been divided into two groups in a way that one group consisted of those patients with sepses, and those patients without a single sepses infection attack were put in the other group (table 8). In the majority of patients with sepses, as it is evident from the table, infection was displayed with one episode, and in only 8 patients two episodes. Table 8 also shows that only 1 (2.5%) out of 40 (100.0%) patients with sepses had a serum albumin level above 40.0 g/L, whereas 39 (97.5%) patients had a serum albumin level below the aforementioned value, hence, they had hypoalbuminia. The difference between these two values in percentages is 95.0%, which evidently indicates a high association between hypoalbuminemia and bacteremia. Moreover, out of 80 (100.0%) patients without infection, 65 (81.25%) patients had a serum albumin level below 40.0 g/L, and 15 (18.75%) of them above this value. The difference between these two percentage values is 62.5. In the group of patients with sepses, the difference in percentages between those

TABLE 7
THE RELATION BETWEEN ALBUMIN LEVEL IN SERUM AND COMPLICATION INCIDENCE

Albumin level in serum (g/L)	Infection	Thrombosis	»Steal« syndrome	Monomelic neuropathy	Pseudo-aneurysm	Hemorrhage serom	Total
< 30.0	5	–	–	–	–	2	7
30.1–35.0	27	4	–	–	–	–	31
35.1–40.0	15	4	–	–	1	–	20
40.1–45.0	1	–	–	–	–	–	1
Total	48 (81.36%)	8 (13.56%)	0	0	1 (1.69%)	2 (3.39%)	59 (100.0%)

TABLE 8
THE RELATION BETWEEN THE ALBUMIN LEVEL IN SERUM AND THE INCIDENCE OF SEPSIS – THE NUMBER OF INFECTIONS EPISODES

Albumin level in serum (g/L)	Nuber of patients		Patients with sepses	
	with sepses	without sepses	infection episodes	
			1	2
< 30.0	5	–	5	1
30.1–35.0	18	11	18	4
35.1–40.0	16	54	16	3
40.1–45.0	1	15	1	–
Total	40	80	40	8

TABLE 9
THE RELATION BETWEEN THE ALBUMIN LEVEL IN SERUM AND THE INCIDENCE OF THE VASCULAR ACCESS INFECTION – INFECTION EPISODES

Albumin level in serum (g/L)	Number of patients		Patients with infection of vascularaccess	
	with the infection of vascular access	without the infection of vascular access	infection episodes	
			1	2
< 30.0	1	4	1	–
30.1–35.0	8	21	8	3
35.1–40.0	5	65	5	–
40.1–45.0	1	15	1	–
Total	15	105	15	3

whose level of serum albumins was below 40.0 g/L and those with the level above that value is 95.0, and in the group of patients without sepses, that same difference is 62.5. If those two values, that is, 95.0 and 62.5 are put in relation, the difference is 32.5 and it is statistically significant ($\chi^2 = 4.77$ P = 0.03), and represents in fact the index of association of hypoalbuminemia and sepses within the observed group of patients.

Table 9 also illustrates the relation between the level of serum albumins and the incidence of localised infection of the vascular access. Out of the 15 (100.0%) patients with vascular access infection, 14 (93.34%) of them had a level of serum albumins below 40.0 g/L, and only 1 (6.7%) above that value. The difference between those two values in percentages is 86,6. On the other hand, out of the 105 (100.0%) patients without vascular access infection, as it is shown in table 9.90 (85.71%) of them had a serum concentration of albumins below 40.0 g/L, and 15 (14.28%) above this value. The difference in percentages is 71.43. If we compare the percentages, the difference between the hypoalbuminemia incidence in the patients with an infection of the vascular access, and is 86,6, and those without infection is 71.43, the difference between these two values is 15.17, and it is statistically insignificant ($\chi^2 = 0.165$ P = 0.69).

Discussion

It has generally been accepted that malnutrition and inflammation, used separately and in synergy, are in a positive correlation with the morbidity and mortality of dialysed patients[31–33]. Kaysen et al.[21] and Mailloux[33] et al. are of the opinion that malnutrition, and in a consistent manner, hypoalbuminemia, would not be considered responsible on their own for increased mortality. Waldman et al.[34] as well as Don and Kaysen[12] mention the example of organisms with analbuminemia that lead a normal life in spite of their condition. In addition, it has been stated that the serum albumin level starts to decline much earlier than the organism reaches the state of terminal renal insufficiency, that is, as early as in the pre-terminal kidney failure phase[35]. Correspondingly, in the malnutrition state of an otherwise healthy organism, the serum albumin level does not decrease significantly for a longer period, yet this basically happens only when in a terminal phase of severe starvation[36].

Our research of dialysed patients went in the direction of establishing the relation between the low serum albumin level and the complication incidence, with special emphasis made on the infection without special emphasis into the research of the hypoalbuminemia oscillation influence, as it has been monitored by Kalantar-Zadeh et al.[8]. In earlier research we have also observed those oscillations of serum albumin concentration. In the majority of monitored patients treated with chronic haemodialysis we did not succeed significantly to influence the stabilisation of the serum albumin level by a programmed dietetic nutritional regime, although Powe et al.[10], based on their findings, suggest that a systematically programmed nutrition could influence the improve-

ment of hypoalbuminemia. 86% of our patiens had an albumin concentration below 40.0 g/L (table 6). Based on experience, this serum albumin concentration was set as a limit as the boundary of determining hypoalbuminemia, and in such respect out views almost concurred with those of Kalantar-Zadeha and al.[8], whereas Powe and al.[10], Held and al.[37] as well as Bergström and al.[38], set a somewhat lower hypoalbuminemia level while researching mortality risk factors.

The hypoalbuminemia incidence difference in percentages among patients with an infection and those without infection as viewed from table 6, is 36.7% and it is statistically significant. This finding regarding the patients treated with chronic haemodialysis could hardy remain being interpreted simply as malnutrition and inflammation markers and morbidity and mortality predictors. Established in a very close relation to hypoalbuminemia observation on the one hand and inflammation on the other hand imposes a further intensive expert-scientific research in the direction of determining the causality of such a relation and parameters. It is to be expected, especially through an intensive investigation of the ways by which hypoalbuminemia significantly influences the increase of infection incidence, which is accompanied by a really high morbidity and mortality rate of dialysed patients.

In the group of 120 (100.0%) examinees included in this study, threatening complications were displayed in 59 (49.2%) of them (table 7), although in an earlier research it was found that the incidence of threatening complications in our patients was ranging at somewhat lower values[39]. Out of those 59 (100.0%) patients with complications, 48 (81.35%) of them exhibited an infection as a complication. Out of these 48 (100.0%) patients with an infection, 47 (97.2%) had a serum albumin level below 40.0 g/L as a complication, and only 1 (2.1%) above this value. This parameter indicates a high association of hypoalbuminemia and the complication occurrence, especially infection.

Table 8 shows patients divided into two groups: one group is comprised of the patients who had a sepses episode and the other group the ones without sepses. Separated in such a way, that is, divided according to the criteria of septic infection, they are also divided according to the serum albumin concentration level. The aforementioned table illustrates that 39 (97.5%) out of 40 (100.0%) patients with sepses had a serum albumin level below 40,0 g/L, that is, they had hypoalbuminemia, and only 1 (2.5%) of them above this level. The difference in percentages is 95.0, and it expressed the hypoalbuminemia ratio in the patients with sepses. Out of 80 (100.0%) of the patients without sepses, 65 (81.25%) of them had serum albumin level below 40.0 g/L and 15 (18.75%) above this value. The difference between these two values is 62.5 and it expresses the hypoalbuminemia ratio in patients without sepses. The calculated hypoalbuminemia ratio in examinees with sepses, as evident from table 8, is 32.5 and it is statistically significant (P = 0.03), and represents a relative index of association between hypoalbuminemia and infection. This finding indicates more a cause-effect relation of hypoalbuminemia and infection than it points that hypoalbuminemia could be only an inflammation and malnutrition *marker*, as well as a possible mortality predictor.

Among the patients with an infection of the vascular access (Table 9), out of the 15 (100.0%) of them, 14 (93.3%) had a serum albumin level below 40.0, and only 1(6.7%) had higher than this value. The difference between these two values regarding percentages, that is, ranging between 93.3% and 6.7%, is 86.6 and it expresses the hypoalbuminemia ratio in patients with an infection of the vascular access. On the other hand, out of 105 (100.0%) patients without vascular access infection, as indicated in table 9, 90 (85.71%) of them had a serum albumin level below 40.0 g/L, and 15 (14.28%) above this level. The difference between these two values expressed in percentages is 71.43, and it expresses the hypoalbuminemia ratio in patients without a vascular access infection. If put in a relative comparison, the percentage difference expressed between the hypoalbuminemia incidence in patients with vascular access infection, which is 86.6%, and those in patients without infection, which is 71.43%, is statistically insignificant as the difference between these two values is 15.17 (P = 0.69). The association index between hypoalbuminemia and infection can also be seen in the research result of this relation, as well as in the relationship between sepses and hypoalbuminemia (table 8). However, we are of the opinion that the basis of this result is too a small specimen or the time interval monitored too short. The research result of this monitored relation also indicated the increased rate of infection and hypoalbuminemia association.

The total infection incidence of the 120 observed patients regarding hypoalbuminemia displayed the usual 3.8 episodes on 100 patients months. This finding fits the research results of other authors in which the infection incidence ranges from 0.7 to 7.0 episodes per 100 patients months. In such respect, Kessler et al.[40] had 0.7, Hoen et al.[41] 0.9 and Tokars et al.[42] 3.5 episodes of infection per 100 patients months.

At the end of the programmed research of clinical causatives encompassed in this study, the following results can be illustrated:

1. 86.6% of patients had a serum albumin concentration below 40.0 g/L,
2. out of 120 (100.0%) patients, 59 (49.16) of them had at least one possible

   complication throughout the observation period,
3. out of 59 (100.0%) patients with complications, 48 (81.35%) of them had an

   Infection,
4. out of 48 (100.0%) patients with an infection, 47 (97.2%) of them had a serum albumin level below 40.0 g/L, that is hypoalbuminemia, and out of 72 (100.0%) without infection, 57 (79.2%) of them had serum albumin level below the mentioned value. The difference in ratios between hypoalbuminemia and infection in

patients with infection and those patients without infection is 36,7 and it is statistically significant (χ^2 =7.215 P =0.0077),

5. from the 48 (100.0%) patients with an infection, 40 (83.3%) exhibited sepses

Symptoms,

6. from the 40 (100.0%) patients with sepses, 39 (97.5%) of them had a serum albumin level below 40.0 g/L, and out of 80 (100.0%) of those patients without sepses, 65 of them (81.25%). The difference in ratios of the relation between hypoalbuminemia and infections in patients with sepses and those patients without is 32.5 and it is statistically significant (χ^2 = 4.77 P = 0.03),
7. 93.3%of the patients with a vascular access infection exhibited a serum albumin level below 40.0 g/L, and in the group of those patients without local infection 85.71% of them. The difference in ratios between hypoalbuminemia and a local infection of the vascular access in patients with an infection and those without an infection is 15.7 and it is not statistically significant (χ^2 = 0.165 P = 0.69),
8. total infection incidence in all 120 patients is 3.8 episodes per 100 patients months.

Conclusion

Established and illustrated values clearly prove a high association of hypoalbuminemia and infection, which demand further comprehensive complex research. This research should find an answer to the continuously repeated question, both in the profession and by science, whether hypoalbuminemia is just a marker of relation between malnutrition and infection and possibly a predictor of morbidity and mortality, or is it in a cause-effect relation with the development of the infection in patients treated with haemodialysis.

REFERENCES

1. FOLEY RN, PARFREY PS, HARNETT JD, KENT GM, MURRAY DC, BARRE PE, J Am Soc Nephrol, 7 (1996) 728. — 2. KEANE WE, COLLINS AJ, Am J Kidney Dis, 24 (1994) 1010. — 3. FELDMAN HI, KORBIN S, WASSERSTEIN A, J Am Soc Nephrol, 7 (1996) 523. — 4. SHLIPAK MG, FRIED LF, CUSHMAN M, JAMA, 293 (2005) 1737. — 5. BLOEMBERGEN BE, PORT FK, Adv Renal Replac Ther, 3 (1996) 201. — 6. TOKARS JI, MILLER ER, STEIN G, Am J Infect Control, 30 (2002) 288. — 7. USRDS 2005 Annual Data Report, Am J Kidney Dis, 45 suppl 1 (2005) S1-S280. — 8. KALANTAR-ZADEH K, KILPATRICK RD, KIWAE N, MCALLISTER CJ, ALCORN H, KOPPLE JD, GREENLAND S, Nephrol Dial Transplant, 20 (2005) 1880. — 9. LOWRIE EG, LEW NL, HUANG WH, Kidney Int, 42 suppl 38 (1992) S22-S31. — 10. POWE NR, JAAR B, FURTH SL, HERMANN J, BRIGGS W, Kidney Int 55 (1999) 1081. — 11. KAYSEN GA, CHERTOW GM, ADHIKARLA R, YOUNG B, BRONCO C, LEVIN NW, Kidney Int, 60 (2001) 333. — 12. DON BR, KAYSEN GA, Semin Dial, 17 (2004) 432. — 13. GULATI S, SAHU KM, AVULA S, SHARMA RK, AYYAGIRI A, PANDEY CM, Renal failure, 25 (2003) 967. — 14. SCHOMIG M, EISENHARDT A, RITZ E, Blood Purification, 18 (2000) 327. — 15. HUANG TL, LEE CT, Psychiatry Research, 152 (2007) 277. — 16. OWEN WF, LEW NL, LIU Y, LOWRIE EG, LAZARUS M, NEJ Med, 329 (1993) 1001. — 17. SULE SD, FADROWSKI JJ, FIVUSH BA, GORMAN G, FURTH SL, Pediatr Nephrol, 22 (2007) 2041. — 18. KAPLAN AA, HALLEY SE, LAPKIN RA, GRAEBER CW, Kidney Int, 47 (1995) 573. — 19. KAYSEN GA, RATHORE V, SHEARER GC, DEPNER TA, Kidney Int, 48 (1995) 510. — 20. KAYSEN GA, GREENE T, DAUGIRDAS JT, KIMMEL PL, SCHULMAN GW, TOTO RD, LEVIN NW, YANG G, HEMO Study Group, Am J Kidney Dis, 42 (2003) 1200. — 21. KAYSEN GA, DUBIN A, MULLER HG, MITCH W, ROSALES LM, LEVIN NW, HEMO Study Group, Kidney Int, 61 (2002) 2240. — 22. KAYSEN GA, DUBIN JA, MULLER HG, ROSALES LM, LEVIN NW, Hemo Study Group, Kidney Int, 58 (2000) 346. — 23. KHAN IH, CATTO GRD, Kidney Int, 43 suppl 41 (1993) S143-S148. — 24. ZIMMERMANN J, HERRLINGER S, PRUY A, METZGER T, WANNER C, Kidney Int, 55 (1999) 648. — 25. BOLOGA RM, VELINE DM, PARKER TS, CHEIGH JS, SERUR D, STENZEL KH, RUBIN AL, Am J Kidney Dis, 32 (1998) 107. — 26. YEUN JY, LEVINE RA, MANTADILOK V, KAYSEN GA, Am J Kidney Dis, 35 (2000) 469. — 27. STEINVINKEL P, HEIMBURGER O, PAULTRE F, DICZFALUSY U, WANG T, BERGLUND L, JOGEDTRANDT T, Kidney Int, 55 (1999) 1899. — 28. MA KW, GREENE RI, RAIJ L, Am J Kidney Dis, 19 (1992) 505. — 29. HALLIWELL B, Hemostasis, 23 (1993) 118. — 30. HAKIM RM, LEVIN N, Am J Kidney Dis, 21 (1993) 125. — 31. CHURCHILL DN, TAYLOR DW, COOK RJ, Am J Kidney Dis, 19 (1992) 214. — 32. JAAR BG, HERMANN JA, FURTH SL, BRIGGS W, POWE NR, Am J Kidney Dis, 35 (2000) 282. — 33. MAILLOUX LU, BELLUCCI AG, WILKES BM, Am J Kidney Dis, 18 (1991) 326. — 34. WALDMANN TA, GORDON RS, ROSSE W, Am J Med, 37 (1984) 960. — 35. KOPPLE JD, GREENE T, CHUMLEA WC, HOLLINGER D, MARONI BJ, MERRILL D, SCHERCH LK, SCHULMAN G, WANG SR, ZIMMER GS, Kidney Int, 57 (2000) 1688. — 36. RIGAUD D, HASSID J, MEULEMANS A, POUPARD AT, BOULIER A, Am J Clin Nutr, 72 (2000) 355. — 37. HELD P, WOLFE RA, GAYLIN DS, PORT F, LEVIN NW, TURENNE MN, Am J Kidney Dis, 23 (1994) 692. — 38. BERGSTROM J, LINDHOLM D, Am J Kidney Dis, 32 (1998) 834. — 39. GALIĆ G, KVESIĆ A, TOMIĆ M, REBAC Z, BREKALO Z, MARTINOVIĆ V, VUČKOV Š, Coll Antropol, 32 (2008 109. — 40. KESSLER M, HOEN B, MAYEUX D, HESTIN D, FONTENAILLE C, Nephron, 64 (1993) 95. — 41. HOEN B, PAUL-DAUPHIN A, HESTIN D, KESSLER M, J Am Soc Nephrol, 9 (1998) 869. — 42. TOKARS JI, LIGHT P, ANDERSON J, MILLER ER, PARRISH J, ARMISTEAD M, JARVIS WR, GEHR T, Am J Kidney Dis, 37 (2001) 1232. — 43. HELD P, WOLFE RA, GAYLIN DS, PORT F, LEVIN NW, TURENNE MN, Am J Kidney Des, 23 (1994) 781. — 44. BERGSTRÖM J, LINDHOLM M, Am J Kidney Dis, 32 (1998) 834.

G. Galić

Akademika Ivana Zovko 14, 88000 Mostar, Bosnia and Herzegovina

ODNOS HIPOALBUMINEMIJE I INCIDENCIJE KOMPLIKACIJA U BOLESNIKA NA PROGRAMU KRONIČNE HEMODIJALIZE LIJEČENIH U KLINIČKOJ BOLNICI MOSTAR

S A Ž E T A K

Hipoalbuminemiju prati povišeni morbiditet i mortalitet bolesnika liječenih kroničnom hemodijalizom. Sve se intenzivnije vode rasprave o tome da li je hipoalbuminemija samo marker malnutricija-inflamacija sindroma te predskazivač povećanog morbiditeta i mortaliteta ili je etiološki čimbenik. Isto se tako sve više istražuje sinergističko djelovanje malnutricije i inflamacije na morbiditet i mortalitet ove poulacije bolesnika. No, ni danas nije nađeno adekvatno objašnjenje tih fenomena. U ispitivanje odnosa hipoalbuminemije i komplikacija koje ugrožavaju vaskularni pristup s posebnim osvrtom na infekciju, a onda i na život liječenih kroničnom hemodijalizom, uzeli smo u obradu 120 bolesnika s terminalnom renalnom insuficijencijom (ESRD) liječenih u Kliničkoj bolnici Mostar kroničnom hemodijalizom. Bolesnici uzeti u obradu u ovoj studiji promatrani su kroz 18 mjeseci. Uzeti su samo oni bolesnici koji u trenutku započinjanja ispitivanja nisu imali ni lokalnu niti sistemsku infekciju, a niti znakove neke od komplikacija koje mogu ugroziti vaskularni pristup, a onda i život bolesnika. Od 120 (100,0%) promatranih ispitanika njih je 86,8% imalo serumsku razinu albumina ispod 40,0 g/L. Analizom rezultata ispitivanja u obradu uzetog kliničkog materijala utvrđeno je da je u ispitanika sa serumskom razinom albumina ispod 40,0g/L incidencija infekcije značajno veća nego u onih s razinom albumina iznad 40,0 g/L. (χ^2 = 7,215 P = 0,0077). Incidencija komplikacija značajno je veća (χ^2 = 9,92 P = 0,0022) među ispitanicima s razinom serumskih albumina ispod 40,0 g/L, nego među onima s višom serumskom razinom. Među ispitanicima s razinom albumina u serumu nižom od 40,0 g/L incidencija sepse je značajno veća (χ^2 = 4,77 P = 0,03), nego među onima sa serumskom razinom albumina iznad te vrijednosti. Međutim razlika u incidenciji lokalne infekcije vaskularnog pristupa između grupe ispitanika sa serumskom razinom albumina ispod 40,0 g/L i onih s razinom albumina iznad te vrijednosti nema značajne razlike (χ^2 = 0,65 P = 0,69). Rezultat ispitivanja ovog parametra, koji ne prati rezultate ispitivanja gore navedenih parametara, može se shvatiti uvjetno kao ishod praćenja nedovoljvo velikog broja ispitanika. Sveukupna incidencija infekcije u 120 promatranih bolesnika iznosi 3,8 epizoda na 100 bolesnikovih mjeseci što se nalazi unutar referiranih vrijednosti drugih autora.