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Research Article

Thyroid Hormonal, Lipidic and Inflammatory Profiles in Patients with Hepatic Encephalopathy

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Abstract

Introduction and Objectives. The number of the patients with liver cirrhosis is globally increasing, but the complex mechanisms of the evolution of this disease is not yet completely discovered. The aim of this study is to analyse the thyroid hormone profile in patients with advanced liver cirrhosis and those suffering from hepatic encephalopathy, concomitantly with lipid metabolism disturbance and inflammatory status. The practical purpose of this work is to prevent the onset of hepatic encephalopathy and a better management of the disease in general and encephalopathy in particular, in the foreground.

Methods and materials. We study the onset of the encephalopathy and the evolution of it, analysing the seric level of thyroid hormones, inflammatory test and lipidic profiles of the patients with liver cirrhosis. The group under study included 419 cases of patients with liver cirrhosis admitted over the course of a year in Constanta County Hospital, of which a number of 135 presented hepatic encephalopathy.

Results. Between 01.03.2022 and 31.03.2023, 419 patients with liver cirrhosis were admitted: 130 women (31.03%) and 289 men (68.97%). A total of 135 patients had hepatic encephalopathy with thyroid TSH (p=0.004), fT4 (p=0.022), T3 (p=0.100), inflammatory ERS (p=0.014), fibrinogen (p=0.121), and lipid cholesterol (p=0.357) profiles. Of 33 patients who died during hospitalization, 26 suffered from encephalopathy.

Conclusions. The links between the occurrence of hepatic encephalopathy and the functioning of other systems and organs present a challenge to the clinician. The present study shows that thyroid, lipid and inflammatory biohumoral changes in patients with hepatic encephalopathy may be indicators for an unfavourable prognosis. Attention to these correlations are helpful in monitoring the progression of liver cirrhosis, the occurrence of encephalopathy, and decreasing mortality.

Keywords: Liver Failure; Evolution of Cirrhosis; Endocrine Profile; Hepatic Disease

Introduction

Liver cirrhosis tends to become a public health problem, the number of cases is increasing both globally and at European level. The Global Burden of Disease (GBD) Study (2017) indicates that the estimated number of persons diagnosed with compensated liver cirrhosis was of 112 million worldwide, with a 10% growth trend until 2019 at the European level [1] The final stage of chronic liver diseases is represented by the replacement of the normal liver tissue by unhealthy, scar tissue. The loss of liver function has serious repercussions, being a worrying cause of mortality and morbidity. According to WHO, cirrhosis was associated with 2.4% of total deaths globally [2].Hepatic encephalopathy represents one of the serious complications of liver cirrhosis and includes all neuropsychological clinical manifestations (signs and symptoms), in the absence of neurological diseases, caused by the impairment of the detoxification function of the liver [3].The management of chronic liver diseases is difficult to perform due to the complexity of liver functions and the metabolic correlations in which they play a decisive role. The evolution of liver cirrhosis is chronic and has potentially fatal irreversible repercussions. The absence of evolution and prognosis markers makes the monitoring and treatment of this complication difficult to keep under control. Given the fact that there is no balance between cell destruction and regeneration and there is no clearly established link between the various biological constants and the disease development makes the evolution of patients in their final stage of chronic liver disease totally unpredictable [4]. The prevention of such complications and finding some correlations between the evolution of the disease and bioumoral changes are important research themes [5].The main objective was to evaluate the thyroid, inflammatory and lipid profile in patients with liver cirrhosis and the secondarily the evaluation of the thyroid, inflammatory and lipid profile in patients with hepatic encephalopathy.

Material and Method

The group under study included 419 cases of patients with liver cirrhosis admitted between 01.03.2022 and 31.03.2023 in Constanta County Hospital, of which a number of 135 presented hepatic encephalopathy.

General features of the group

Initially, the group was made of 455 patients, evaluated to be included in the study. Following the application of the inclusion and exclusion criteria, 36 patients who did not meet the inclusion criteria (4 patients) or presented exclusion criteria (32 patients) were taken out. Among the 32 with exclusion criteria, 21 were previously diagnosed with autoimmune thyroiditis, 18 of them at the same time with hepatitis C virus infection.

Inclusion criteria

All patients aged 18 years old, can read and write, they have given their informed consent, who have been diagnosed with liver cirrhosis following a clinical examination, paraclinical-biological and imaging tests, classified into severity categories and treated accordingly, appearing in the records of an internist or gastroenterologist, or newly diagnosed during hospital admission.

The diagnosis of liver cirrhosis was clinically confirmed by the presence of signs and symptoms of liver failure and of portal hypertension, but also paraclinical – based on biological tests and imaging methods. Child Pugh and Meld scores were calculated so that each case was placed in a category in terms of evolution and severity.

Exclusion criteria

Eighteen-years-old patients, who refused participation, who, prior to the diagnosis of cirrhosis, had a thyroid condition under or without treatment, were not included in the study, patients who were on medication that could have influenced the metabolism of thyroid hormones were excluded from the study group, such as the contrast agent with iodine and amiodarone, which exacerbates the conversion of T4 to Triiodothyronine T3, but also of other classes of drugs such as glucocorticoids and dopamine, which decrease the secretion of TSH with the subsequent decrease of T3.

The biological samples under analysis - description, method of collection, analysis, and normal values. We have determined the value of the thyroid-stimulating hormone (TSH, thyrotropin), which presents a stimulating action on the formation and secretion of thyroid hormones, as well as a proliferative action [6]. The pituitary release of TSH is the central mechanism for regulating the biological action of thyroid hormones and represents a highly specific and sensitive parameter for the control of the thyroid function [7].

The TSH sampling is done on fasting and no blood is collected for determining TSH after a recent thyroid biopsy, nor after a surgical procedure on the thyroid. There is a diurnal variation of the TSH level, registering the maximum level at 23:00 [8]. Collected sample – venous blood. Sampling container - vacutainer without anticoagulant, with/without separating gel. Necessary processing after sampling - the serum is separated by centrifugation; the fresh serum is processed; if this is not possible, the serum shall be kept at 2-8 ˚C or at -20 ˚C. Sample volume – at least 0.5 mL serum. The determination method is immunochemistry with electrochemiluminescence detection (ECLIA). Considering the age of the patients in the study group, we considered as normal values the range between 0.27 and 4.2 microIU/ml and we excluded any analytical and drug interferences [8].

Thyroxine (T4) is a thyroid hormone with effects on general metabolism, but it also represents a physiological component of the thyroid gland regulatory circuit. Most of the circulating thyroxine is connected to transport proteins (TBG, prealbumin and albumin) [7]. The rest of the hormone circulates freely in the form of fT4 (free thyroxine), biologically active. The fT4 determination has the advantage of being independent of the concentration and binding properties of the proteins that transport thyroxine, thus faithfully correlating with the patient`s clinical status. Sample collecting is done on fasting, from venous blood. Sample collection container - vacutainer without anticoagulant, with/without separating gel.

Necessary processing after collection - the serum is separated by centrifugation; the fresh serum is processed; if this is not possible, it shall be stored at 2-8°C or -20°C. Sample volume – at least 0.5 mL serum. Method – immunochemical with detection by electrochemiluminescence (ECLIA). **The reference values are between 12.0-22.0 pmol/L. We excluded any possible interferences with some elements of the kit, but also with the drugs the patient was taking and that could have influenced the resulting value** [8]**.**

Triiodothyronine is mainly responsible for the actions of thyroid hormones at the level of various target organs. Most part of T3 hormone is produced outside the thyroid gland, particularly in the liver, by enzymatic deiodination in 5′ position of T4. For this reason, the serum concentration of T3 reflects more the functional state of the peripheral tissues, than the secretory performance of the thyroid gland. Reducing the conversion of T4 to T3 generates a decrease in the serum concentration of T3 [9]. Just like T4, more than 99% of the amount of T3 is connected to the transporter proteins, but with a 10 times lower affinity. **Sampling and determination method are like fT4 determination. It is considered as the reference range 1.3-3.1 nmol/L** [10]**.** Erythrocyte Sedimentation Rate (ESR) represents the rate at which red blood cells from an anticoagulated blood sample sediment in one hour. The faster the red blood cells sediment, the higher is the ESR, thus being an indicator of acute phase response. An increase in ESR occurs at least 24 hours after the initiation of the inflammatory response, and after the end of the acute phase response it declines with a half-life of 96-144 hours [11]. Compared to CRP and serum amyloid A, ESR is also increased in situations where there is an increase in the concentration of immunoglobulins, immune complexes and other proteins.

Patient preparation – fasting/after eating; a lipid meal can cause plasma alterations. Collected sample – venous blood. The sample collection container is represented by a vacutainer with buffered sodium citrate of 3.8% or a vacutainer with EDTA K3 (for the capillary microphotometric method), and the amount collected - as much as the vacuum allows.

Methods for determining the sedimentation rate of red blood cells: the manual Westergren method: the tube is placed in a vertical position in a millimetre graduated support and read the sedimentation level of red blood cells in mm after 1 hour; in some assays, the result is also read after an interval of 2 hours, but it does not provide any additional information; the automatic ESR reading method (using an infrared ray system); the capillary microphotometric method: it measures the aggregation capacity of erythrocytes (the first stage of sedimentation) in the presence of agglomerants, at 37 °C.

Reference values

Men: Women: <50 years <15 mm/h <50 years <20 mm/h 50-85 years <20 mm/h 50-85 years < 30 mm/h >85 years <30 mm/h >85 years <42 mm/h

Fibrinogen is a particularly important parameter in the evaluation of patients with chronic liver disease. It is the substrate of action both for thrombin, the last enzyme in the coagulation cascade, as well as for plasmin, the enzyme of the fibrinolytic system. In addition, fibrinogen belongs to the group of acute phase proteins (increased values appear 24-48 hours after the occurrence of the event) [12].Patient preparation – on fasting. The collected sample is represented by venous blood. Sample collection container - vacutainer with Na citrate 0.105M (sodium citrate - blood ratio = 1/9). The pressure created by the tourniquet cuff must be between the value of systolic pressure and the diastolic pressure and shall not exceed 1 minute. If the venous puncture has failed, a new attempt on the same vein can only be made after 10 minutes. The collected quantity - as much as the vacuum allows it; to prevent partial coagulation of the sample, the correct mixing of the blood with the anticoagulant shall be ensured, by inverting the tube. Necessary processing after sample collection - the sample will be centrifuged for 15 minutes at 2500g. Sample stability - the sample is stable for 4 hours at room temperature, and the separated plasma for several months at -20 °C. The coagulometric method (Clauss): in the presence of an excess of thrombin, the coagulation time of a citrated plasma, diluted (1/10), poor in platelets is inversely proportional to the concentration of fibrinogen. The test determines the functional level of fibrinogen (activity). Normal values are considered in the range between 200 and 400 mg/dl [13]. The increase of fibrinogen consumption: in CID and in hyper-fibrinolysis reactions from metastatic cancers, acute promyelocytic leukaemia, obstetric complications. The consumption of fibrinogen can start extremely quickly; therefore, its dosage must be carried out at short time intervals. When CID overlaps the acute phase response, fibrinogen may be falsely elevated [14]. The decrease in fibrinogen synthesis: in severe liver diseases accompanied by a decrease in liver parenchyma (liver cirrhosis, mushroom poisoning), in diseases accompanied by abnormal liver irrigation (right-side heart failure). Thrombolytic therapy: the decrease in fibrinogen concentration

depends on the dose and the type of medication administered: streptokinase and urokinase determine a pronounced decrease in fibrinogen (values < 10mg/dL); the tissue plasminogen activator (t-PA) and prourokinase determine a moderate decrease in fibrinogen. Moderate alcohol consumption lowers fibrinogen levels [15]. The increase of fibrinogen synthesis in the acute phase response due to infections, inflammations, tumours, traumas, burns. In the case of extensive cellular destruction (e.g. surgical procedures, myocardial infarction, radiotherapy) fibrinogen returns to normal after the acute phase response, unlike active chronic inflammatory processes from rheumatic diseases and collagen diseases, in which its level remains elevated for a long period of time. As a compensatory response to protein loss (particularly of albumin) in patients with nephrotic syndrome, multiple myeloma, hepatic disease, cirrhosis, oestrogens treatment, compensated intravascular coagulation [16]. Determining the cholesterol level evaluates the lipid status and metabolic disorders, the risk of atherosclerosis, coronary stenosis, and myocardial infarction [5]. The preparation of the patient shall comply with several essential conditions for an accurate result. Thus, the patient shall have to follow an unchanged diet for 3 weeks before the sample collection, he/she must have a stable body weight and not eat 12-14 hours before the sampling, and he shall not have consumed alcohol for 72 hours prior to sampling. It should also be taken into consideration that after 20 minutes of lying down, the cholesterol level is 10-15% lower than when standing, and after 20 minutes of sitting, the cholesterol level is 6% lower than when standing, and the blood pressure extended over 2 minutes of the tourniquet, shall increase the value of cholesterol by 2-5%. Sample collected - venous blood, sample collection container - vacutainer without anticoagulant, with/without separating gel. Necessary processing after sampling - the serum is separated by centrifugation; it is processed on the same day or stored at 4 ℃ or -20 ℃5, sample volume – at least 0.5 mL serum. Sample stability – the separated serum is stable for 5-7 days at 2-8 ℃; 3 months at -20 ℃; several years at -70 ℃. Method – spectrophotometric (enzymatic–colorimetric) [17].

It should be mentioned that there are intra-individual variations (4-10%), seasonal variations: values higher by up to 8% in winter than in summer, and the diet rich in cholesterol as well as pregnancy shall determine increases in cholesterol. In terms of drugs interferences, there is a whole series of drugs that can change the results of cholesterol both in the sense of its increase and decrease [18]. Among these drugs, we mention diuretics and beta blockers, frequently used in the treatment of cirrhosis and which determine higher cholesterol values compared to the real value [19].

Determining the level of triglycerides shall require the observance of special measures before collecting the blood sample - the patient must follow an unchanged diet for 3 weeks before the sample collection, he/she shall have a stable body weight, not eat 12-16 hours before the collection, abstinence from alcohol is recommended for 72 hours before sampling. Collected sample – venous blood. Sample collection container - vacutainer without anticoagulant with/without separating gel.

Reasons for sample rejection - intensely haemolysed specimen. Necessary processing after sampling - the serum is separated within a maximum of 1 hour after sampling; it shall be processed on the same day or stored at 4 ℃ or -20 ℃. Sample volume – at least 0.5 mL serum. Sample stability – the separated serum is stable for 5-7 days at 2-8 ℃; 3 months at -20 ℃; several years at -70 ℃. Method – spectrophotometric (enzymatic colorimetric) [20].

The intra-individual variation is of 12-40% (minimum values in the morning and maximum values around lunch). Transient increases may occur after a rich lunch and alcohol ingestion. Increased values can be encountered in pregnancy, obesity, lack of physical activity, smoking. Intense effort (transient decrease), diet modification (within three weeks` time), weight loss can determine low values [21].

Changes in lipid profile could be also related to changes in fibrinogen levels, which acts as both an inflammatory marker and clotting factor. Thus, in the patient with uncompensated liver cirrhosis, the results of blood samples should be seen in correlation and not interpreted individually [22].

An optimal level of triglycerides is considered a value lower than 150 mg/dl. Results between 150 and 199 mg/dl are considered borderline high, while between 200 and 499 mg/dl are high values, and over 500 mg/dl we talk about very high values [23]. Descriptive statistics are presented according to variable characteristics. Post-hoc analysis was performed on significant variables to establish the significant relationships between the groups. Statistical tests were determined by a normality check using the Shapiro-Wilks test, with the Dunn Kruskal-Wallis post-hoc test used for non-parametric numerical variables. A chi-square test was used for the categorical variables. For the calculations of coefficients, multiple multinomial models were fitted: a basic, an inclusive, and a disjointed-down model.

A P value of <.05 was considered statistically significant. Tests were not paired. Statistical analyses were performed using R statistical software version 4.0.2 (The R Foundation for Statistical Computing), using RStudio 1.2.5033.

Results

The group under analysis included 419 participants. Distribution in terms of sex: 130 women (31.03%) and 289 men (68.97%). Age distribution showed a median value of 66 years. The results obtained, 30-40 years - 7 cases (1.67 %), 40-50 years - 30 patients (7.16 %), 50-60 years - 109 patients (26.01%), over 60

years old 273 cases (65.16 %), show an increased frequency of cases in the 50-60 age group and over 60 years old.

About the presence of hepatic encephalopathy (HE) in cirrhotic patients included in the study, we noted that 135 of the patients with cirrhosis had an episode of hepatic encephalopathy at presentation or during hospital admission. In the age group 30-40 years - 2 patients with HE (1.48% of all patients with HE), 40-50 years - 12 cases of HE (8.88%), 50-60 years -61 cases with HE (45.18%), age group over 60 years, - 60 patients with HE (44.44%).

Distribution according to the duration of the disease - we noted that the phenomena of hepatic encephalopathy appear at least 2 years after the diagnosis of cirrhosis - in all analysed cases, with several 97 patients (71.85%) who presented neurological disorders with different degrees, and after 3 years from the diagnosis, 128 patients presented decompensation of liver function accompanied by neurological disorders (94.85%).

Child Pugh				
	A	B	C	P
Total	78	206	135	
TSH (mean (SD)	2.71(1.55)	2.62(1.85)	2.71(1.83)	0.883
T3 (mean (SD)	1.85 (0.75)	1.91 (0.74)	1.93 (0.77)	0.719
fT4 (mean (SD)	14.16(2.66)	14.19(2.79)	14.04(2.33)	0.875
Cholesterol (mean (SD)	297.21(64.18)	288.88(55.94)	296.92(65.42)	0.39
Fibrinogen (mean (SD)	318.69(105.74)	306.79 (104.24)	323.04(102.39)	0.339
ERS (mean (SD)	33.11(6.94)	32.39 (6.96)	34.12(8.70)	0.118

Table 1: Changes in thyroid hormones, inflammatory samples and lipid profile depending on the severity of cirrhosis.

The reporting of the thyroid, lipid and inflammatory profile to the Child Pugh classification did not lead to significant data. We noticed that there are no statistically significant differences in the thyroid hormone profile depending on the Child stage. Cholesterol values were increased in a similar proportion in all cirrhotic patients, regardless of belonging to a particular Child Pugh class. Inflammatory tests did not register changes in correlation with the Child Pugh classification (Table 1). Distribution according to the number of deaths during hospitalization. - 33 patients from the total number of cases of liver cirrhosis included in the study group died during hospitalization, and of these, 26 suffered from encephalopathy at the time of the cardio-respiratory arrest. Our study confirmed the fact that the risk of death increases as the disease progresses (a higher risk with the entry into the next Child Pugh classification category). It should be noted that among the 33 patients who died, 30 had presented phenomena of hepatic encephalopathy at admission or during their last hospitalization.

Encephalopathy grade						
	0	1	2	3	4	p
Total	284	28	74	21	12	
Cholesterol (mean (SD)	292.75 (60.44)	286.64 (52.49)	288.43 (58.52)	304.24 (67.45)	323	0.357
ERS (mean (SD)	32.54 (6.76)	33.00 (9.26)	33.35 (8.85)	38.19 (10.20)	35.58 (5.12)	0.014*
Fibrinogen (mean (SD)	322.87 (106.05)	294.64 (84.17)	303.78 (99.13)	281.81 (109.10)	276.92 (99.22)	0.121
TSH (mean (SD)	2.55(1.70)	2.15 (1.27)	2.92 (1.82)	3.90 (2.73)	2.92 (1.71)	0.004*

T3 (mean (SD)	1.87 (0.74)	2.01 (0.57)	1.88 (0.55)	2.32 (1.45)	1.87 (0.60)	0.1
fT4 (mean (SD)	14.06 (2.63)	15.55 (3.15)	14.20 (2.56)	13.11 (1.76)	14.13 (1.80)	0.022*

Table 2: Changes in thyroid hormones, inflammatory samples and lipid profile depending on the degree of encephalopathy.

The study followed thyroid samples, lipid metabolism and inflammatory status depending on the degree of hepatic encephalopathy. The results showed that there is a link between the progression of encephalopathy and the level of TSh and fT4. Statistically significant values were also obtained in the case of the sedimentation rate of erythrocytes and fibrinogen depending on the degree of encephalopathy. The same cannot be said about the T3 value, as no values were recorded to prove a real connection **(Table 2).**

Discussions

This study analyses the thyroid hormone, lipid, and inflammatory profile in patients with liver cirrhosis, particularly in those with hepatic encephalopathy.

Unlike the current trend, of a decrease in the number of cases of viral liver cirrhosis given the emergence of revolutionary antiviral treatments, particularly regarding hepatitis C, the distribution of cases was relatively uniform, with the predominance of toxic ethanol aetiology. Patients with autoimmune aetiology were not included in the study, presenting various degrees of thyroid impairment. We note that the male gender predominates, with a median age of 68.

The biochemical parameters included in the Child Pugh classification are definitely associated with liver dysfunction and are not markly influenced by distant factors. The decline on this scale is mainly caused by the damage to the organic functions and the evolution of liver failure.

The most frequent abnormality of the thyroid function associated with liver cirrhosis was represented by the decrease in T3 level. Either associated or not with increased TSH values, this drop was encountered in 17% of cases. The decrease of T3 value below the values of 1.3 nmol/l, in the absence of a thyroid disease, is called low T3 syndrome or euthyroid disease. The possible explanation could be that, since the liver is the main region for T4 to T3 conversion, a decrease in T3 conversion indicates that the severity of the liver disease directly affects the thyroid hormone deiodination process, rather than having an indirect systemic impact.

The decrease in the level of T3 is associated with an unfavourable evolution of patients suffering from hepatic encephalopathy and an increased mortality rate. We noted that more severe decreases in T3 level are associated with more serious forms of encephalopathy (grade 3 and 4 of encephalopathy at T3 values below 1 nmol/l).

Furthermore, in the context of T3 decrease, the increased level of TSH can indirectly help to identify patients with a poor prognosis.

The attempt to administer hormone substitution does not seem to have any effect on the evolution of the disease. There is a tendency to normalize T3 level in the treatment of the underlying disease, particularly in patients whose evolution is favourable. About the relationship between thyroid function and the aetiology of liver disease, it is found that ethanol aetiology is most often correlated with T3 and TSH changes, and only to a small extent with T4 variations.

In addition, lipid metabolism is a point of interest - it is well-known that the liver plays a crucial role in the synthesis, secretion, catabolism, and storage of lipids and lipoproteins. Therefore, serum concentrations of lipids and lipoproteins in liver diseases could be altered [24].

The results of this study do not provide information if alcoholic and non-alcoholic liver cirrhosis equally affect lipid metabolism, nor if hepatic encephalopathy or thyroid status can be correlated with the level of dyslipidaemia. It is known that the major effects of high alcohol consumption on lipid metabolism are the excessive synthesis of triglycerides, hypertriglyceridemia and hypercholesterolemia, the defective esterification of plasma cholesterol, and the decrease in cholesterol levels [25], but we have insufficient data to clearly state a hypothesis in this regard. The results of the studies carried out until the present time are controversial. The reason for the discrepancy in the results could be the different etiology of the liver injury, but also drug interference. Another reason for the insufficiently significant results could be represented by the conditions that must be observed by the patients before the sample collection, impossible to achieve in most cases. The patients included in the study group were hospitalized after exclusively appearing in the Emergency Unit, and the samples for the lipid profile were collected in most cases on the first day of hospital admission [26].

Furthermore, inflammation plays an essential role in the production and evolution of liver disease [27]. Hepatic inflammation

is a trigger of liver disease and is the main factor affecting liver tissue, thus triggering the progression from liver disease to severe fibrogenesis to cirrhosis and hepatocellular carcinoma [28]. The production of pro-inflammatory cytokines (IL-1α, IL-1β, tumour necrosis factor-alpha, and IL-6) proves the role played by inflammation in the production of steatosis, fibrosis, production, and decompensation of liver cirrhosis [29,30].

We know that the increased level of ammonia causes an inflammatory response [13]. The results confirm the link between the erythrocyte sedimentation rate, as a marker of inflammation, and the degree of encephalopathy, as an expression of increased ammonium levels with central repercussions.

Our data confirms this and can represent a significant indicator in following the evolution of these patients while there is also a link between the occurrence of low T3 syndrome in patients with hepatic encephalopathy and the presence of inflammation in the body.

Conclusions

The persistence of low T3 syndrome can represent an indicator of unfavourable evolution of the decompensation episode of chronic liver disease. This fact proves to be useful in monitoring patients suffering from hepatic encephalopathy associating with serious cases, with evolution towards severe forms and exitus.

The presence of inflammation in the body seems to be an aggravating factor of the decompensation of chronic liver disease and to have a connection with the occurrence of hepatic encephalopathy, but also to thyroid changes (T3 decrease) in these patients. Considering that fibrinogen, used as a marker of inflammation, is produced by the liver, we cannot consider fibrinogen values as a benchmark in the monitoring and follow-up of patients with liver cirrhosis. Instead, the results obtained show a close relationship between the red blood cell sedimentation rate and the level of hepatic decompensation, but also with encephalopathy degree.

According to the data under analysis, the lipid profile does not yet provide enough information to be considered a landmark in the follow-up of patients with complicated liver cirrhosis or with encephalopathy.

Although, according to the available literature data, the most common change in thyroid hormones in cirrhotic patients is represented by the decrease in T3, in this study the closest correlation between the degree of encephalopathy and thyroid function is achieved through TSH and FT4 level. Therefore, in the monitoring and follow-up of the evolution of patients with hepatic encephalopathy, there is a new connection with important value.

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