INTERNATIONAL JOURNAL OF BIOASSAYS
ISSN: 2278-778X
CODEN: IJBNHY
ORIGINAL RESEARCH ARTICLE
OPEN ACCESS

# Effect of nutrition on prognosis of Egyptian cirrhotic patients

**Engy Yousry Elsayed[1], Inas Elkhedr Mohamed[1*] and Iman Mahmoud Ezzat[2]**
[1]Department of Gastroenterology, Ain-Shams University, Cairo, Egypt
[2]Department of Internal Medicine, Alfayoum University, Alfayoum, Egypt

**Received for publication:** September 16, 2015; **Accepted**: October 24, 2015

**Abstract:** Malnutrition is a common complication of end-stage liver disease, by far the easiest methods to differentiate compensated and uncompensated liver cirrhosis are the Child–Pugh and MELD score. These methods have omitted under nutrition from their scoring. Present study is aimed to study the effect of malnutrition among Egyptian cirrhotic patients on the prognosis and mortality. 300 Egyptian cirrhotic patients, laboratory and nutritional assessment were done according to RFH-SGA, measurement of BMI, TST, and MAMC. Each patient was assigned a Child and MELD score and all patients were followed up for six months. 49 patients (16.3%) died throughout the follow up. 57(19%) were severely malnourished. 134 (44.66%) and109 (36.33%) were well nourished and moderately malnourished, respectively. 63.4% of non-survivors were malnourished (p=0.001). A higher MELD score in non-survivors (29.8) than that of survivors (21.7) (P=0.007). Increase the TST (triceps skinfold thickness) in survivors (10.1mm i.e. less than 50$^{th}$ percentile) than in non-survivors (5.9mm i.e. less than 5$^{th}$ percentile) (P=0.003). The MAMC (midarm muscle circumference) was 24.5cm (i.e. 10$^{th}$ percentile) and 19.4cm (i.e. less than 5$^{th}$ percentile) in survivors and non-survivors, respectively (P=0.002). The nutritional status of cirrhotic patients is an important tool, together with MELD and Child score, to predict the prognosis of such patients and the severity of malnutrition is associated with higher mortality, early referral of cirrhotic patients with bad nutritional status for transplantation is recommended.

**Key words**: Child score; Liver cirrhosis; MELD; Nutritional Status.

## INTRODUCTION

Cirrhosis is the result of the progression of many forms of necroinflammatory liver diseases leading to fibrosis, vascular remodeling, portal hypertension development along with its complications, and ultimately liver failure [1]. As a result of the complex pathophysiological processes associated with cirrhosis, it results in significant morbidity such as gastrointestinal bleeding from portal hypertension, and eventual mortality in many patients [2]. The prognosis of patients with advanced cirrhosis is grim, with a 5-year survival rate of <10% [3]. In addition to the associated morbidity highlighted above, protein-energy malnutrition (PEM) has often been observed in patients with liver cirrhosis [4,5]. Causes for malnutrition in liver cirrhosis are known to include a reduction in oral intake (for various causes), increased protein catabolism and insufficient synthesis, and malabsorption/maldigestion associated with portal hypertension [4,6,7]. Although a consequence of the disease, malnutrition alone can lead to further morbidity in patients with liver cirrhosis. Increased rates of septic complications, poorer quality of life, and a reduced life span have all been observed in cirrhotics with poorer nutrition status compared to those without[8,9]. An accurate diagnosis of patients helps appropriate selection of treatment program. The model of end-stage liver disease (MELD) score was originally determined to predict survival in cirrhotic patients undergoing surgery. It is now used to assign priority for liver transplantation [10]. MELD score is a useful tool to assess prognosis in critically ill cirrhotic patients [11]. The Child-Turcotte-Pugh (CP) classification is by far the most widely applied and reported system as it is easy to use as a bedside test [12]. Child-Pugh, and MELD scores constitute the best tools to predict mortality in patients with cirrhosis; however, one of their main limitations is the lack of assessing the nutritional and functional status. Currently, numerous methods are available to evaluate the nutrition status of the cirrhotic patient [13].

Present work is mainly focused to study the effect of malnutrition among Egyptian cirrhotic patients on the prognosis and mortality.

## MATERIALS AND METHODS

This prospective study was conducted at Ain Shams university hospital between January 2014 and January 2015. A series of consecutive 300 hospitalized, and outpatient clinic patients, with chronic liver disease were recruited for the study. All patients were followed up over a period of six months. The primary endpoint of this study was mortality. The following patients were excluded from the study: Patients with intrinsic renal disease, patients with any end organ failure, patients with hepatocellular carcinoma. Informed consent was obtained in all patients prior to participation in accordance with the recommendations of the institutional ethics committee. All participants had a systematic work up on admission, all data were collected prospectively, and the analysis performed on this data retrospectively. The patients were classified into two groups as the following: 251 survivor and 49 non-survivor. They were subjected to full history taking, clinical examination (hepatomegaly, signs of liver cell failure, ascites,…etc), Laboratory investigation (AST, ALT, ALP, Gamma GT, Albumin, Total protein, Prothrombin time, α-fetoprotien, CBC), Abdominal ultrasonography (cirrhotic changes, ascites, focal lesions…. etc).

Modified Child-Pugh classification and MELD score at the time of admission or assessment as outpatient.

**Child classification:** Each patient was assigned a score and a grade reflecting the severity of liver affection according to the numerical system of Child Turcotte Pugh (CTP) [14]. Class A (score 5-6) class B (score 7-9) and class C (score more than 9) as shown in (Table 1).

***Corresponding Author:**
**Inas Elkhedr Mohamed**
Department of Gastroenterology,
Ain-Shams university,
Cairo, Egypt.

**Table 1:** Child-Turotte-Pugh Classification

| Parameter | Points assigned | | |
|---|---|---|---|
| | 1 | 2 | 3 |
| Ascites | Absent | slight | moderate |
| Bilirubin mg/d | < /=2 | 2-3 | >3 |
| Albumin | >3. 5 | 2. 8-3. 5 | <2. 8 |
| Prothrombin time INR | 1-3 | 4-6 | >6 |
| | <1. 7 | 1. 7-2. 3 | >2. 3 |
| Enceohalopathy | None | Grade 1-2 | Grade 3-4 |

MELD score was calculated according to original formula proposed by the mayo clinic group: meld score= {9. 57xlog creatinine (mg/dl) +3. 78x log bilirubin (mg/dl) +11. 2 x log INR+6. 4. we used on-line available worksheet to compute MELD scores (http://www. mayoclinic. org/gi-rst/mayomode15. html).

### *Nutritional Assessment*

The standard SGA (subjective global assessment) "Fig. 1" comprised a nutritionist evaluation of height, weight (current, before illness, and weight range in the previous 6 months), nutritional history (appetite, intake, gastrointestinal symptoms), physical appearance (subjective assessment of fat loss, muscle wasting, edema and ascites). Detailed nutritional assessment collected by single person according to specific questionnaire provided by dietitian, specialized in liver disease. A modified body mass index (BMI) has been proposed for patients with cirrhosis, and to provide an index for underweight in patients with ascites. 1 A BMI < 18. 5 kg/m2 is usually considered underweight, but in patients with cirrhosis a BMI < 20 kg/m2 was associated with increased mortality. A BMI < 23 kg/m2 may indicate underweight in patients with mild ascites, while BMI < 25 kg/m2 may be underweight for patients with severe or tense ascites [15]. Further anthropometric measurements included the following: midarm circumference (MAC), triceps skinfold thickness (TST), midarm muscle circumference (MAMC). MAC was measured to the nearest centimeter with a measuring tape at the right arm. TST, an established measure of fat stores, was measured to the nearest millimeter at the right arm using Harpenden skinfold caliper (Baty Ltd, British Indicators) in a standard manner [16]. Mid-arm muscle circumference (MAMC), an established measure of muscle protein mass, was calculated from MAC and TST using a standard formula: MAMC = MAC - (3. 1415×TSF) [16]. TST and MAMC recorded according to Frisancho percentile tables [17].

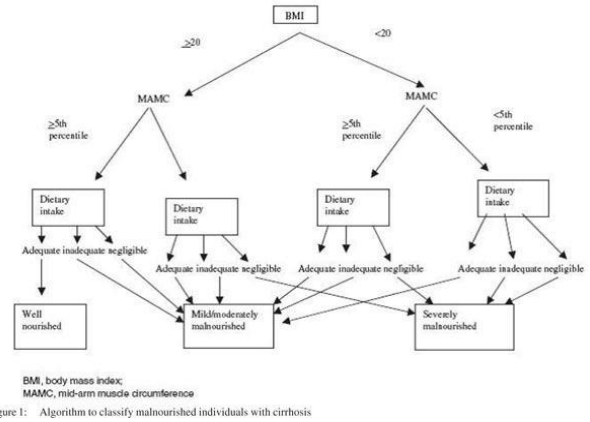


**Figure 1:** subjective global assessment

Measurements of the upper arm circumference and triceps skinfold the middle of the upper arm is measured using a measuring tape (right arm).

- Determine the middle between the shoulder top and the point of the elbow and mark this point.
- At this point you measure the upperarm circumference, the measuring tape gently around the arm, do not pull.
- Measure the triceps skinfold at the back of the arm at the same height as the upperarm circumference, using a skinfold caliper.
- Take the skinfold in a vertical position between thump and pointing finger.
- Ask the patient to stretch his arm and strain his muscles, so the muscles are no part of in the skinfold.
- Ask the patient to relax his arm and let it hang down along the body.
- Place the caliper on the skinfold.
- Let go the handgrip and wait two seconds before reading the value.
- Repeat this measurement three times.
- Calculate the mean of the three measurements.

The triceps skinfold in mm is measured in the middle on the backside of the right upperarm [17,18]. If MAMC is less than 23. 5 cm, BMI is likely to be less than 20 kg/m² i. e. subject is likely to be underweight. If MAMC is more than 32. 0 cm, BMI is likely to be more than 30 kg/m² i. e. subject is likely to be obese [16]. McWhirter, 1994, demonstrated in his research that an upper arm muscle circumference below 15th percentile shows that we can speak about malnutrition. Below the 5th percentile it's severe malnutrition [19].

### *Statistical analysis*

Kaplan–Meier and Cox proportional hazards regression models used to prospectively identify factors associated with mortality. Statistical presentation and analysis of the present study was conducted, using the mean, standard error, Chi-square, Linear and Analysis of variance [ANOVA] tests by SPSS V17. $P < 0.05$ was considered significant and $P < 0.01$ was considered highly significant.

## RESULTS

300 consecutive patients with liver cirrhosis collected from Ain-Shams university hospital and followed up for six months, patients classified into: 251survivors, (age ranging from 20-65 with mean age 51years), the majority were males 63 females (21%) and 237 males (79%) and 49 non-survivor group (age ranging from 45-70 with mean age 61 years), again mostly were males eleven were females (22.4%) and 38 males (77.6%). Basic nutritional parameters measured on the patients are summarized in (Tables 2).

**Table 2:** Representing Nutritional Parameters Measured At Initial Assessment.

| Parameter | RFH-SGA | | | |
|---|---|---|---|---|
| | All patients | Well nourished | Mild/moderate malnourished | Severely malnourished |
| Number of patients | 300 | 134 | 109 | 57 |
| bilirubin | 3±0. 29 | 2. 9±0. 29 | 4±0. 35 | 4. 1±0. 41 |
| Albumin | 2. 51±0. 86 | 2. 93±0. 95 | 2. 4±0. 65 | 2. 2±0. 63 |
| T. protien | 6. 01±1. 3 | 6. 00±0. 9 | 6. 23±1. 1 | 5. 5±1. 3 |
| INR | 2±1. 9 | 1. 8±0. 17 | 2±0. 19 | 2. 2±0. 24 |
| PT | 19. 5±8. 3 | 15. 2±6. 1 | 18. 2±7. 5 | 23. 1±9. 2 |
| creatinine | 1. 1±8. 4 | 0. 94±0. 23 | 1. 09±0. 9 | 1. 24±1. 00 |
| CP score | | | | |
| A | 111 | 84 | 27 | 0 |
| B | 159 | 45 | 73 | 41 |
| C | 30 | 5 | 9 | 16 |
| MELD | 23. 46±9. 8 | 17. 7±5. 6 | 23. 9±7. 9 | 28. 8±9. 9 |
| BMI | 21. 05±3. 8 | 23. 3±4. | 19. 3±2. 7 | 19. 9±3. 1 |
| TST | 8. 1±5. 4 | 11. 3±6. 8 | 8. 1±5. 8 | 5. 0±3. 9 |
| MAMC | 21. 95±4. 9 | 22. 7±5. 3 | 21. 6±5. 1 | 19. 8±4. 8 |
| Diet intake: | | | | |
| Adequate | 164 | 107 | 42 | 15 |
| Inadequat | 93 | 18 | 59 | 16 |
| Negligible | 43 | 9 | 8 | 26 |

PT: Prothrombin time. BMI: body mass index (Kg/m²).
INR: International normalized ratio. CP score: Child Pugh score.
TST: triceps skinfold thickness (mm). MAMC: midarm muscle circumference (cm).

Overall, 134 (44.66%) of the patients were well nourished, 109(36. 33%) were moderately malnourished, and 57(19%) of patients were severely malnourished. Individuals who are severely malnourished had a higher CP and MELD scores than other nutritional groups ($p$=0. 004). The laboratory parameters, the liver functions and creatinine, were better in well-nourished and moderately malnourished than severely malnourished, a higher T. protein and albumin and less INR, PT and creatinine ($p$=0. 006). Inadequate dietary intake more among the severely malnourished (16 patient i. e. 28%). Anthropometric parameters were impaired in the severely malnourished individuals, TST and MAMC were 5mm ($P$=0. 001) and 19. 8 (P=0. 006) cm respectively, this means less fat stores and more muscle wasting. Although BMI was little higher in those group (19. 9 Kg/m²) than moderately malnourished (19. 3 Kg/m²), but this can be explained by the presence of ascites.

(Table 3) represented a comparison between survivors and non-survivors regarding nutritional parameters: The total protein was higher in survivor group ($P$=0. 006). Serum albumin was higher in survivor group (P=0. 24). Better anthropometric parameters in survivor group including BMI, TST and MAMC, $P$= 0. 003, $P$=0. 002, $P$=0. 001 respectively. A higher MELD and CP scores in the non-survivors ($P$=0. 007, 0. 00 respectively). BMI in non-survivor group was 19. 5Kg/m² according to the modified BMI for cirrhotic patients there is increase in mortality if BMI is less than 20Kg/m². TST and MAMC were less than 5$^{th}$ percentile and 10$^{th}$ percentile respectively. According to McWhirter, these results mean severe malnutrition. The percentage of malnourishment in the non-survivor individuals was 69.4% which was much higher the survivor individuals 9.2% ($P$=0. 001)

**Table 3:** Comparison between survivor and non-survivor regarding nutritional parameters:

| Parameter | Survivor | Non-survivor | P value |
|---|---|---|---|
| Total protein | 6. 23±0. 941 | 5. 09±1. 55 | 0. 006 |
| albumin | 2. 4±0. 66 | 2. 0±0. 55 | 0. 248 |
| BMI | 22. 6±2. 78 | 19. 5±5. 4 | 0. 004 |
| TST | 10. 1±3. 16 | 5. 9 ±1. 43 | 0. 003 |
| MAMC | 24. 5 ± 3. 6 | 19. 4 ±1. 5 | 0. 002 |
| CP score | | | |
| A | 111 | 0 | |
| B | 131 | 28 | 0. 000 |
| C | 9 | 21 | |
| MELD | 21. 7±7. 5 | 29. 8±9. 9 | 0. 007 |
| Percentage of malnutrition | 9. 16% | 69. 38% | 0. 001 |

## DISCUSSION

Malnutrition is a common complication of end-stage liver disease and is an important prognostic indicator of clinical outcome (survival rate, length of hospital stay, post transplantation morbidity, and quality of life) in patients with cirrhosis. Several studies have evaluated nutritional status in patients with liver cirrhosis of different etiologies and varying degrees of liver insufficiency [20,21].

The present study tried to find out the role of nutritional status in the prognosis of Egyptian cirrhotic patients. The study represented 300 patients followed-up for a period of 6 months. Overall 57(19%) of the participants were severely malnourished. 134 (44. 66%) and109 (36. 33%) were well nourished and moderately malnourished, respectively. The Child Pugh score revealed that majority of patients i. e. 159 (53%), belonged to Child B group and 111 patients (37%) to Child A with only 30 (10%) Child C group. A higher MELD score (28. 8) in the severely malnourished patients. The non-survivors had higher CP score, mostly belong to Child B and C ($P$=0. 00). Moreover, the non survivors had a higher MELD (29. 8) than that of survivors (21. 7) ($P$=0. 007), that's means the higher CP and MELD scores are associated with higher mortality rate, these results were supported by Kamath *et al., 2001* who found direct relation between MELD score and the mortality rate [22]. Also, Sempere *et al., 2009* stated that Child score ≥ 10 were the variables associated with mortality [23]. D'Amico *et al., 2006* have shown that the CP score was the most common significant predictor of death (identified in 63% of the studies evaluated), followed by all its components: albumin, bilirubin, ascites, encephalopathy, and prothrombin time[24].

The results revealed higher total protein in survivors ($p$=0. 006). BMI among the survivors (83. 7%) was 24. 5Kg/m²; while, BMI was 19. 5Kg/m² (16. 3) among the non-survivors. MAMC was 19. 4cm, <10$^{th}$ percentile, and TST was 5. 9mm, <5$^{th}$ percentile, which means severe malnutrition in 16. 3% of total number of cirrhotic patients on first presentation. Although different percentage, but comparable results of other studies. These results agreed

with that of Sasidharan *et al.,* 2012, The BMI was higher among survivor 21. 5, while was 19. 2 among non-survivor. The MAMC was 24. 1 and 21. 9 among the survivor and non-survivor respectively, patients with poor nutritional status are more likely have poor survival as compared to their better nourished counterparts, irrespective of their Child or MELD scores [25].

Moreover Tai *et al.,* 2010, demonstrated that the patients with cirrhosis exhibited a range of nutritional abnormalities, with protein-energy malnutrition of 50% (MAMC < 5th percentile) and fat store depletions of 30% (TST <5th percentile) [26].

Alberino *et al.,* 2001, showed that 34% of cirrhotics with MAMC < 5th percentile [8]. A hospital-based study by Campillo B *et al.,* 2003, who studied 315 patients from France (58. 7% of Child-Pugh C cirrhotic patients with MAMC < 5th percentile) [27].

These results mean that under nutrition is common among cirrhotic patients and are meeting the concept that the liver plays a central role in the regulation of nutrition by trafficking the metabolism of nutrients, and many factors disrupt this metabolic balance in end-stage liver failure. Consequently, when the liver fails, numerous nutritional problems occur. Several factors contribute to malnutrition in liver failure including inadequate dietary intake of nutrients, reduction in their synthesis or absorption (diminished protein synthesis, malabsorption), increased protein loss, disturbances in substrate utilization, a hypermetabolic state as well as increased energy-protein expenditure and requirements. Because of decreased glycogen stores and gluconeogenesis, energy metabolism may shift from carbohydrate to fat oxidation while insulin resistance may also develop. Consequently, liver cirrhosis frequently results in a catabolic state resulting in a lack of essential nutrients[28].

From the study, the severity of malnutrition associated with bad prognosis and higher mortality. Platuth M *et al.,* 1997, stated that Protein energy malnutrition is associated with unfavorable clinical outcomes. In cirrhotic patients in general there is an association between nutritional status and mortality. Furthermore, within Child A and B, there is an association between nutritional status and mortality. Malnutrition when defined by low dietary intake is associated with high mortality. Malnutrition has been shown an independent predictor of both the first bleeding episode and survival in cirrhotic patients with eosophageal varices. Malnutrition is also associated with refractory ascites or the persistent ascites [29], this may explain the bad prognosis and higher mortality among malnourished cirrhotics.

In controlled trials, the rate of complications (ascites, gastrointestinal bleeding, encephalopathy, infection and mortality) tend to respond favorably to intervention that successfully increased nutrient intake in treated patients over controls [30,31,32].

## CONCLUSION

The nutritional status of cirrhotic patients is an important tool, together with MELD and Child score, to predict the prognosis of such patients, and the severity of malnutrition is associated with higher mortality. Early referral of cirrhotic patients with bad nutritional status for transplantation is recommended.

## REFERENCES

1. Schuppan D and Afdhal NH . Liver cirrhosis. Lancet, 2008 371, 838-851.
2. Orloff MJ , Halasz NA , Lipman C, Schwabe AD, Thompson JC and Weidner WA . The complications of cirrhosis of the liver. Ann Intern Med, 1967, 66(1), 165-198.
3. Norstrom T. The impact of per capita consumption on Swedish cirrhosis mortality. Br J Addict, 1987 82(1), 67-75.
4. Lautz HU, Selberg O, Korber J, M. Burger M and Muller MJ. Protein-calorie malnutrition in liver cirrhosis. Clin Investig, 1992, 70(6), 478-486.
5. Moriwaki H . Protein-energy malnutrition in liver cirrhosis. J Gastroenterol, 2002, 37(7), 578-579.
6. Coltorti M, Del Vecchio-Blanco C, Caporaso N, Gallo C, & Castellano L. Liver cirrhosis in Italy. A multicentre study on presenting modalities and the impact on health care resources. National Project on Liver Cirrhosis Group. Ital J Gastroenterol, 1991,23(1), 42-48.
7. Sobhonslidsuk A, Roongpisuthipong C, Nantiruj K, Kulapongse S, Songchitsomboon S, Sumalnop K, & Bussagorn N. Impact of liver cirrhosis on nutritional and immunological status. J Med Assoc Thai, 2001, 84(7).
8. Alberino F, Gatta A, Amodio P, Merkel C, Di Pascoli L, Boffo G andCaregaro L. Nutrition and survival in patients with liver cirrhosis. Nutrition, 2001, 17(6), 445-450.
9. Dan AA, Kallman JB, Srivastava R, Younoszai Z, Kim A and Younossi ZM . Impact of chronic liver disease and cirrhosis on health utilities using SF-6D and the health utility index. Liver Transpl, 2008, 14(3), 321-326.
10. Krige E and Beckingham J. ABC of diseases of liver, pancreas, and biliary system: Portal hypertension :1-Portal hypertension-2: Ascites, encephalopathy, and other conditions. BMJ, 2001, 17,322(7283), 416-418.
11. Giannini E, Botta F, Fumagalli A, Malfatti F, Testa E, Chiarbonello B, Polegato S, Bellotti M, Milazzo S , Borgonovo G and Testa R. Can inclusion of serum creatinine values improve the Child–Turcotte–Pugh score and challenge the prognostic yield of the model for end-stage liver disease score in the short-term prognostic assessment of cirrhotic patients? Gastroenterology, 2003, 125, 993–4.
12. Pugh RN, Murray-Lyon IM, Dawson JL, Pietroni MC and Roger Williams . Transection of the oesophagus for bleeding oesophageal varices. Br J Surg, 1973, 60, 646–9.
13. Aldo J. Montano-Loza . Clinical relevance of sarcopenia in patients with cirrhosis. World J Gastroenterol, 2014, 20 (25), 8061-8071.

14. Lucey M, Brown K, Everson G, Fung J, Gish R and.Keefe E. Minimal criteria for placement of adults on the liver transplant wait list: a report of a national conference organized by the American Society of Transplant Physicians and the American Association for the Study of Liver Diseases, Transplantation, 1998, 66, 956–62.

15. Moctezuma-Velázquez C, García-Juárez I, Soto-Solís R, Hernández-Cortés J and Torre A. Nutritional assessment and treatment of patients with liver cirrhosis. Nutrition, 2013, 29 (11-12), 1279-85.

16. Jones JM. Reliability of nutritional screening and assessment tools. Nutrition, 2004, 20(3), 307-311.

17. Frisancho A. New norms of upper limb fat and muscle areas for assessment of nutritional status. Am J Clin, 1981, Nutr. 34, 2540-2545.

18. Frisancho A. Triceps skinfold and upper arm muscle size norms for assessment of nutritional status. Am J Clin Nutr, 1974, 27, 1052-1058.

19. McWhirter JP and Pennington CR. Incidence and recognition of malnutrition in the hospital. BMJ, 1994, 308, 945-948.

20. Italian Multicentre Cooperative Project on Nutrition in Liver Cirrhosis. Nutritional status in cirrhosis. J Hepatol, 1994, 21 (3), 317-325.

21. Muller MJ . Malnutrition in cirrhosis. J Hepatol, 1995, 23 (1), 31-35.

22. Kamath PS, Weisner RH, Malinchoc M, Kremers W, Therneau TM, Kosberg CL, D'Amico G , Dickson ER, and Kim WR . A model to predict survival in patients with end stage liver disease. Hepatol, 2001, 33, 464-470.

23. Sempere L, Palazón M, Sánchez-Payá J, Pascual S, de Madaria E, Poveda J, Carnicer F, Zapater P and Pérez-Mateo M. Assessing the short- and long-term prognosis of patients with cirrhosis and acute variceal bleeding. Rev EspEnferm Dig, 2009, 101(4), 236-48.

24. D'Amico G, Garcia-Tsao G and Pagliaro L. Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. J Hepatol, 2006, 44, 217–231.

25. Sasidharan M, Nistala S, Narendhran RT, Murugesh M, Bhatia SJ and Rathi PM. Nutritional status and prognosis in cirrhotic patients. Trop Gastroenterol, 2012, 33(4), 257-64.

26. Tai MS, K. L. Goh KL , Mohd-Tai SH, Rampal S and Mahadeva S. Anthropometric, biochemical and clinical assessment of malnutrition in Malaysian patients with advanced cirrhosis. Nutrition Journal, 2010, 9, 27.

27. Campillo B, Richardet JP, Scherman E and Bories PN. Evaluation of nutritional practice in hospitalized cirrhotic patients: results of a prospective study. Nutrition, 2003, 19(6), 515-521.

28. Bémeur C, Desjardins Pand Roger Butterworth F. Role of Nutrition in the Management of Hepatic Encephalopathy in End-Stage Liver Failure. J Nutr Metab, 2010, Vol. 2010, 12.

29. Platuth M, Merl M, Kondrup J, Weimann A, Ferenciandand P and Mullér MJ. ESPEN guidelines for nutrition in liver disease and transplantation. Clinical nutrition, 1997, 16, 43-55.

30. Bunout D, Aicardi V, Virsch S, Petermann M, Kelly M, Silva G, Garay P, Ugarte G and Iturriaga H. Nutritional support inhospitalized patients with alcoholic liver disease. Eur J Clin Nutr, 1989, 43, 615-621.

31. Keatus PJ, Young H, Garcia G, Blaschke T, O'Hanlon G, Rinki M. , Sucher K and Gregory P. Acellerated improvement of alcoholic liver disease with enteral nutrition. Gastroenterology, 1992, 102, 200-205.

32. Mezey E, Caballeria J, Mitchell MC, Parés Herlong HF and Lodés J. Effect of parentral amino acid supplementation on short-term and long-term outcomes in severe alcoholic hepatitis, a randomized controlled trial. Hepatolog, 1991, 14, 1090-1096.

**CITE THIS ARTICLE AS:**
Engy Yousry Elsayed, Inas Elkhedr Mohamed, Iman Mahmoud Ezzat. Effect of Nutrition on Prognosis of Egyptian Cirrhotic Patients. *International Journal of Bioassays* 4.11 (2015): 4499-4503.

**Source of support:** Nil
**Conflict of interest:** None Declared