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# Serum Galectin-3 Levels in Patients with Hepatocellular Carcinoma, Liver Cirrhosis and Chronic Viral Hepatitis

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**ABSTRACT**

**Background:** hepatocellular carcinoma (HCC) represents a global health problem. It is the fifth most common solid tumor and the third cause of cancer-related mortality per year. In Egypt, it represents 75% of malignant liver tumors. Early detection and diagnosis of these cases are required for successful treatments and improved outcomes. **Aim of the Work:** this study aimed to detect serum galactin-3 levels in patients with HCC, liver cirrhosis and chronic viral hepatitis (HBV, or HCV) patients. **Patients and Methods:** this prospective study was conducted on a total of 60 patients, 20 of them with chronic viral hepatitis B or C, 20 with cirrhosis secondary to chronic viral hepatitis and 20 with HCC secondary to chronic viral hepatitis. It was carried out at the Clinical Pathology Department, Tanta University Hospital. **Results:** the mean galectin-3 levels were 15.5 ng/mL (±5.5) in HCC patients, 20.46 ng/mL (±7.56) in cirrhotic patients and 7.003 ng/mL (±4.24) in chronic viral hepatitis group. There were statistical differences between HCC and cirrhotic patients ($P < 0.03$), but they were lower in chronic hepatitis group statistically compared to cirrhosis and HCC ($P < 0.001$). **Conclusion:** serum galectin-3 levels in patients with chronic HBV or HCV may guide us about progression to cirrhosis or HCC and prognosis of the disease. In these patients, if galectin-3 levels were found to be high, serum alpha-feto protein level and ultrasonographic examination could be repeated at more frequent intervals. This may also guide us in terms of the treatment plan. **Recommendations:** it was recommended to measure changes of galectin-3 in hepatitis carries. Measurement of galectin-3 in a large scale of patients to explore its prognostic value.

Keywords: Galectin-3, HCC, Liver cirrhosis, Chronic viral hepatitis.

## INTRODUCTION

Hepatocellular carcinoma (HCC) represents a global health problem. It is the fifth most common solid tumor and the third cause of cancer-related mortality per year **(1)**.

In Egypt, HCC represents 75% of malignant liver tumors. Liver cancer is the 5th most common cancer in both genders, the 6th in female it represented 3.4% of cancers and 2nd in order in males after cancer urinary bladder representing 11.5% of all cancers. In 2010, liver cancer came in the 3rd order in both sexes (8.1%), 1st in males (12.1%) and 5th in females (4%) **(2)**.

The etiology of HCC included major risk factors such as infection with hepatitis B or C virus (HBV, HCV), alcohol abuse or dietary exposure to aflatoxin **(3)**. Regardless of the carcinogenic insult HCC usually develops in patients with cirrhosis due to chronic inflammation and advanced fibrosis **(4)**. Nonalcoholic steatohepatitis (NASH), a metabolic disorder resulting from insulin fibrosis and cirrhosis, is emerging as another important risk factor for HCC **(5,6)**.

During the past decade the management of HCC had significantly improved **(7)**. However, only about 10% to 20% of patients were currently eligible for potentially curative therapies at the time of diagnosis **(8)**, most of the patients with hepatocellular carcinomas were diagnosed at an advanced stage and their prognosis remained very dismal **(9)**. Thus, early detection and diagnosis of hepatocellular carcinomas still present the best chance for successful treatments and improved outcomes **(10)**. Galectins are members of a newly defined and growing family of the animal lectins. Galectin-3 is the most extensively studied. Oncogenic and viral stimulation can change galectin-3 expression and galectin-3 increases in several human tumors **(11)**. Normal hepatocytes do not express galectin-3, but this protein can be present in HCC. A study revealed that galectin-3 expression in HCC was independent of whether the patient had prior HBV infection. It was found that focal regenerating nodules of cirrhotic tissue also express galectin-3. A study showed that galectin-3 expression was involved in the tumor progression and related to the tumor prognosis of HCC **(12)**.This study discussed the possible role of galectin-3 in HCC. It highlighted the involvement of galectin-3 in other pathological settings of the liver, where chronic viral hepatitis B or C and/or cirrhosis take place.

## AIM OF THE WORK

This study aimed to detect serum galactin- 3 levels in patients with HCC, liver cirrhosis and chronic viral hepatitis (HBV, or HCV) patients.



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## SUBJECTS AND METHODS

This prospective study was carried out at Clinical Pathology Department, Tanta University Hospital and included 60 patients categorized into three groups:

- **Group I**: 20 patients with chronic viral hepatitis B or C.
- **Group II:** 20 patients with cirrhosis secondary to chronic viral hepatitis.
- **Group III:** 20 patients with HCC secondary to chronic viral hepatitis.

The patients were selected from Tropical Medicine Department, Tanta University Hospital. All patients with HCC didn't have metastatic diseases; their diseases were limited to liver.

**Studied groups were subjected to the following:**

1. Full history taking and thorough clinical examination.
2. Abdominal ultrasonography and ultrasound guided liver biopsy were performed by true-cut needle or liver biopsy gun for the cirrhotic patients when possible.
3. Triphasic C.T for patients with focal lesions.
4. Laboratory investigations including:
   - **a. Liver function tests included:**
   - ALT
   - AST
   - Prothrombin time.
   - Albumin
   - **b. Complete blood count.**
   - **c. Serum AFP level.**
   - **d. Viral hepatitis markers.**
   - **e. Serum Galectin-3 level was measured using enzyme linked immunosorbant assay (ELISA) according to manufacturer's instructions. All results are given as ng/mL.**

All data were recorded in Excel XP for windows. Statistics were performed using SPSS 16.0 for Windows. For comparison of the means of two groups, Student's t-test was used and a $P < 0.05$ was considered as significant.

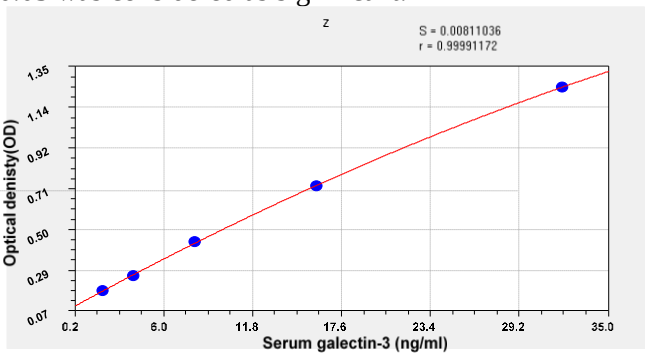


**Figure 1:** standard curve of serum galectin-3 assay.

**The study was approved by the Ethics Board of Ain Shams University.**

## RESULTS

There were 60 patients (HCC: 20 patients, cirrhosis: 20 patients, and chronic hepatitis B or C: 20 patients) in this study. All patients with HCC did not have metastatic diseases; their diseases were limited to liver. Male/female ratios were 13/7, 10/10, and 11/9, respectively. The mean age was 55.4 ± 9.757 in HCC and 49.2 ± 9.774 in the cirrhosis group. In the chronic hepatitis group, mean age was 43.15 ± 10.404. The demographic features and laboratory values are given in **Table 1.** AST, ALT, PT, albumin and AFP levels were statistically different in each group.

**Table 1: the demographic features and laboratory levels of all patients**

| | *HCC* | *Cirrhosis* | *Chronic viral hepatitis* |
|---|---|---|---|
| ***Male/female*** | 13/7 | 10/10 | 11/9 |
| ***Age (years)*** | 55.4 ± 9.75 | 49.2±9.774 | 43.15±10.4 |
| ***Hemoglobin (g/dL)*** | 10.74±1.575 | 11.285±1.222 | 11.28±0.647 |
| ***White blood cell (×10³/mm³)*** | 4.912±1.571 | 5.11±1.157 | 4.92±1.154 |
| ***Platelet(×10³ /mm³)*** | 96.75±26.347 | 92.55±26.482 | 122.7±25.515 |
| ***Prothrombin time (sec)*** | 14.2±2.298 | 17.55±1.826 | 12.15±1.122 |
| ***AST (U/L)*** | 43.00±10.895 | 43.45±9.603 | 40.425±9.989 |
| ***ALT (U/L)*** | 43.050±10.753 | 29.850±7.321 | 35.75±7.282 |
| ***Albumin (g/dL)*** | 3.316±0.351 | 2.93±0.425 | 3.55±0.268 |
| ***AFP (U/L)*** | 965.00±202.5 | 5.657±0.969 | 22.047±4.79 |

**Comparison between the three studied groups as regard galectin-3 (ng/ml):**

As regard serum galectin-3 levels, in chronic viral hepatitis group ranged from 2.45–13.5 ng/mL with a mean of 7.003±4.243, while, in cirrhotic group it ranged from 11.7–38.8 ng/mL with a mean of 20.465± 7.569 and in HCC group it ranged from 5.6– 29.5 ng/mL with a mean of 15.538 ± 5.59.There was a significant difference in galectin-3 between the three groups (**P**<0.001). Galectin-3 was significantly higher in HCC patients as compared to chronic viral hepatitis group (**P**<0.001), it was also significantly higher cirrhotic patients as compared to chronic viral hepatitis group (**P**<0.001), It was significantly lower in HCC patients as compared to cirrhotic group (**Table 2)**.

**Table 2**: **comparison between mean values of galectin-3 (ng/mL) in the studied groups.**

| Groups | Galectin-3 (ng/ml) | | | ANOVA | |
|---|---|---|---|---|---|
| | Mean | ± | SD | F | P-value |
| **Chronic viral hepatitis** | 7.003 | ± | 1.243 | 26.127 | <0.001* |
| **Cirrhotic** | 20.465 | ± | 3.569 | | |
| **HCC** | 15.538 | ± | 2.590 | | |
| **TUKEY'S Test** | | | | | |
| **Chronic viral hepatitis&Cirrhotic** | **Chronic viral hepatitis&HCC** | | **Cirrhotic&HCC** | | |
| <0.001* | <0.001* | | 0.030* | | |

**Correlation between galectin-3 and the studied parameters in the three groups (Table 3)**

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**able 3:** correlation between galectin-3 and studied parameters in the three groups.

| Correlations | | | | | | |
|---|---|---|---|---|---|---|
| | Galectin-3 | | | | | |
| | Chronic viral hepatitis | | Cirrhotic | | HCC | |
| | R | P-value | R | P-value | r | P-value |
| **Age** | 0.070 | 0.768 | 0.111 | 0.641 | 0.064 | 0.787 |
| **ALT** | 0.139 | 0.558 | -0.137 | 0.565 | 0.021 | 0.930 |
| **AST** | -0.104 | 0.661 | 0.102 | 0.670 | 0.023 | 0.923 |
| **AFP** | 0.135 | 0.570 | 0.166 | 0.485 | 0.539 | 0.014* |
| **PT** | -0.234 | 0.320 | 0.130 | 0.585 | 0.079 | 0.742 |
| **Albumin** | -0.193 | 0.415 | 0.228 | 0.334 | -0.123 | 0.605 |
| **Hb** | -0.310 | 0.184 | 0.022 | 0.926 | 0.210 | 0.375 |
| **WBC** | -0.442 | 0.051 | -0.086 | 0.717 | -0.220 | 0.352 |
| **Platelet** | -0.291 | 0.213 | 0.101 | 0.673 | 0.264 | 0.260 |

There was a significant positive correlation between galectin-3 and AFP in HCC group **(Figure 2).**

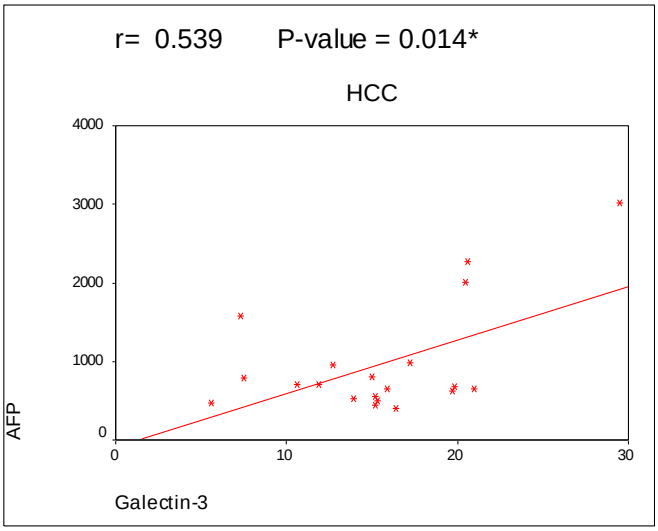


**Figure 2:** correlation between galectin-3 and AFP in HCC group**.**

**Table 4**: diagnostic performance of serum galectine-3 (ng/ml) discriminating group I from group II.

| ROC curve between Chronic viral hepatitis and Cirrhotic | | | | | |
|---|---|---|---|---|---|
| **Cutoff** | **Sens.** | **Spec.** | **PPV** | **NPV** | **Accuracy** |
| >12.6 | 90.0 | 95.0 | 94.7 | 90.5 | 97.7% |

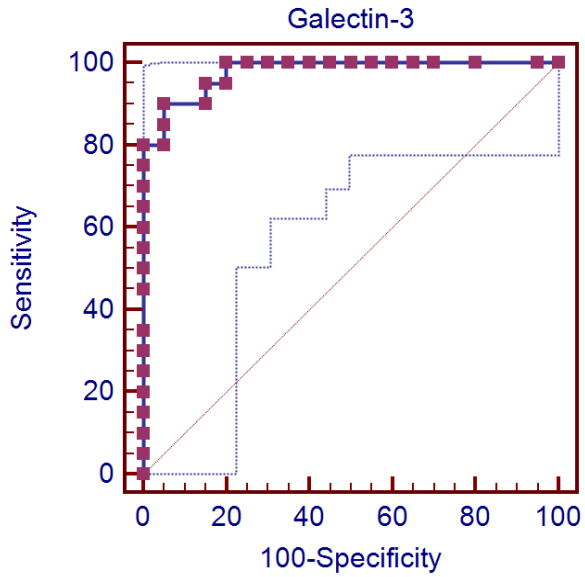


**Figure 3:** diagnostic performance of serum galectine-3 (ng/ml) discriminating group I from group II.

**Table 5:** diagnostic performance of serum galectine-3 (ng/ml) discriminating group I from group III.

| ROC curve between chronic viral hepatitis and HCC | | | | | |
|---|---|---|---|---|---|
| **Cutoff** | **Sens.** | **Spec.** | **PPV** | **NPV** | **Accuracy** |
| >12.6 | 75.0 | 95.0 | 93.7 | 79.2 | 89.9% |

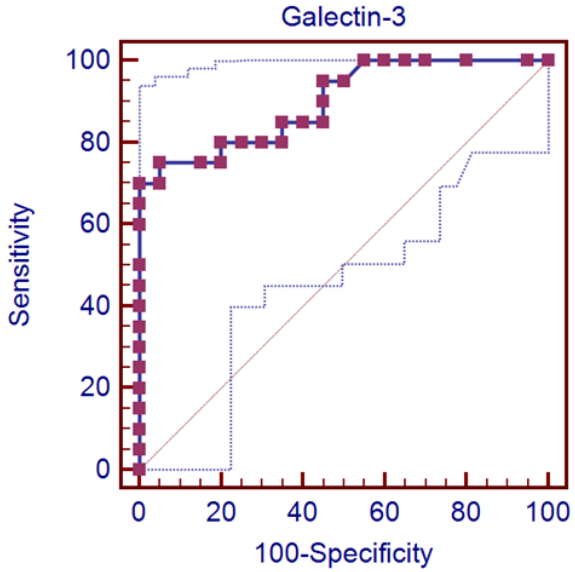


**Figure 4:** diagnostic performance of serum galectine-3 (ng/ml) discriminating group I from group III.

## DISCUSSION

Hepatocellular carcinoma (HCC) represents a global health problem. It is the fifth most common solid tumor and the third cause of cancer-related mortality per year [(1)].

In Egypt, HCC represented 75% of malignant liver tumors. Liver cancer is the 5th most common cancer in both genders, the 6th in female it represented 3.4% of cancers and 2nd in order in males after cancer urinary bladder representing 11.5% of all cancers. In 2010, liver cancer came in the 3rd order in both sexes (8.1%), 1st in males (12.1%) and 5th in females (4%) [(2)].

The etiology of HCC included major risk factors such as infection with hepatitis B or C virus (HBV, HCV), alcohol abuse or dietary exposure to aflatoxin. Regardless of the carcinogenic insult, HCC usually develops in patients with cirrhosis due to chronic inflammation and advanced fibrosis [(4)]. Nonalcoholic steatohepatitis (NASH), a metabolic disorder resulting from insulin fibrosis and cirrhosis, was emerging as another important risk factor for HCC [(5,6)].

AFP is the most widely used tumor biomarker currently available for the early detection of HCC. A previous clinical study carried out by **Yan-Jie** [(13)] demonstrated that serum AFP had a sensitivity of 41–65% and specificity of 80–94% when the cut-off value was 20 ng/ml. However, certain issues should be considered with regard to early diagnosis that the positive rate of AFP in HCC was only 60–80%, the false-positive AFP results were positive during pregnancy, as well as for active liver disease, embryonic tumor and certain gastrointestinal tumors and also false-negative, limitations in terms of sensitivity in different detection methods.

Galectin-3 is a multifunctional member of the galectin family. It plays an important part in the biological behavior of various tumors and may have diagnostic and prognostic significance[(14)]**.** A study showed an association between galectin-3 expression and HCC. It has been reported that galectin-3 expression is induced in cirrhotic livers and HCC and that galectin-3 overexpression inhibited the immune response by inducing apoptosis in lymphocytes and thus promoted tumor growth [(15)].

In this study 20 patients with chronic viral hepatitis either HBV or HCV were chosen. Their age ranged between 25-60 years and with a mean value of 43.150±10.404 years and 20 cirrhotic patients on top of chronic viral hepatitis with age range between 27-63 years and a mean value of 49.2±9.774 years, while the last group was of 20 patients who had HCC on top of chronic viral hepatitis ranged from 41-70 years with mean value of 55.4±9.757 years.

In a previous study, age incidence of HCV infection was 45-60 yrs [16]. This was in agreement with the present study. As regards to sex there were predominance of HCC and chronic viral hepatitis in males than in females which is in agreement with a previous study which suggested that the reason why HCC is more common in men than women is due to the fact that HBV, HCV and alcohol consumption were more prevalent and possibly more carcinogenic in males [17].

In this study ALT levels in the chronic viral hepatitis patients ranged from 10 – 91u/l with a mean of 35.750±19.282 u/l and in cirrhotic patients it ranged from 10-65 u/l with a mean of 29.850±14.321u/l, while in HCC patients it ranged from 10-112 with a mean of 43.050±31.753 u/l. While AST in chronic viral hepatitis patients ranged from 2.5-77 u/l with a mean of 40.425±17.989/L and in cirrhotic patients it ranged from 19-101 u/l with a mean of 43.450±22.603 u/l while in HCC patients it ranged from 10-125 with a mean of 43.000±30.895 u/l.

These results showed that in some patients there was an increase in ALT above normal value. In a previous study there were persistent elevated ALT levels in Egyptian adults infected with HCV [18]. Most chronically infected patients have slight elevations in ALT levels [19]. This was in agreement with the present study. However, in another study it was concluded that patients with chronic HCV infection have normal or borderline ALT values [20]. Another study performed on HCV infected patients showed that elevated ALT levels above 70u/L was strongly associated with the incidence of HCC. This finding indicated that ALT level is a good independent determinant of the need for intervention and that clinical application of these findings may help decrease HCC-associated mortality in hepatitis virus-endemic regions [21].

In this study, prothrombin time (PT), in chronic viral hepatitis group was ranged from 10.5– 14.8 seconds with a mean of 12.15± 1.122, while, in cirrhotic group it ranged from 14.6–20 seconds with a mean of 17.55± 1.826 and in HCC group PT ranged from 12.4 – 20 seconds with a mean of 14.2 ± 2.298. It is prolonged in cirrhotic and HCC groups.

In severe hepatocellular disease, decreased synthesis of liver-produced plasma proteins led to reduced serum levels of several blood clotting factors. Hemorrhage may occur as a complication of chronic liver disease because of a lack of one or more liver-produced blood clotting factors, thrombocytopenia, and/or defective platelet function. Hemorrhage in such patients may also occur from esophageal or gastric varicose secondary to portal hypertension. The biosynthetic pathways of blood coagulation factors II, VII, IX and X are within the hepatocyte and are dependent on vitamin K. Low serum levels of these factors were associated with prolongation of the prothrombin time (PT) [22].

The PT is usually not elevated until cirrhosis is present and the liver fibrosis is fairly significant. PT measures only the formation of fibrin from thrombin and does not assess the effect of fibrinolysis factors [23].

In this study albumin level in the chronic viral hepatitis patients ranged from 3.2– 3.9 g/dl with a mean of 3.55 ± 0.268g/dL and in cirrhotic it ranged from 2.4 – 3.6 g/dl with a mean of 2.93± 0.425 g/dl, while in HCC patients it ranged from 2.6 – 3.82 g/dl with a mean of 3.316 ± 0.351g/dl.

In a previous study of 454 patients with HCV, only 25 showed low albumin level and rest of patients had normal albumin level [24] and this is in agreement with the present study whose chronic viral hepatitis patients had normal albumin level. While, patients with advanced cirrhosis almost always have hypoalbuminemia caused both by decreased synthesis by the hepatocytes and water and sodium retention that dilutes the content of albumin in the extracellular space, also due to increased transcapillary transport rate [25] and this is in agreement with the present study whose cirrhotic patients had low albumin level. In this study most of HCC patients have low serum albumin level and some have normal level. A previous study stated that low recurrence rate of HCC was associated with high serum albumin levels in patients; therefore, high levels of serum albumin are a major indicator of a favorable prognosis [26].

In this study, WBCs in chronic viral hepatitis patients ranged from 2.8-6.8 x $10^3$cells/$mm^3$ with a mean of 4.92± 1.154 x $10^3$cells/$mm^3$ and in cirrhotics it was ranged from 2.4– 6.8 x $10^3$cells/$mm^3$ with a mean of 5.11± 1.157 x $10^3$cells/$mm^3$, while in HCC group WBCs ranged from 2.9 – 7.8 x $10^3$cells/$mm^3$ with a mean of 4.912±1.571x $10^3$cells/$mm^3$.

This study showed that chronic viral infected patients had low level of WBCs in their peripheral blood which may be attributed to the main cause of neutropenia in chronic viral hepatitis patients was the treatment they receive to eradicate the virus [27]. Causes of leukopenia in cirrhosis include portal hypertension-induced splenic and splanchnic sequestration, alterations in granulocyte-colony stimulating factor and granulocyte macrophage-colony stimulating factor and bone marrow suppression mediated by toxins (eg, alcohol, hepatitis B and C) [28].A previous

study showed that the majority of cancer patients developed neutropenia [29]. Another study revealed that there were various hematologic disorders associated with HCV [30].

This is in agreement with the present study. In this study, Hb level in chronic viral hepatitis patients ranged from 10.7 – 12 g/dl with a mean of 11.28 ± 0.647g/dL and in cirrhotics it ranged from 8.1–12.9 g/dl with amean of 11.28 ± 1.222 g/dl, while in HCC patients it ranged from 7.9– 13 g/dl with a mean of 10.74 ± 1.575. g/dL. Values for Hb in the three groups were almost decreased. Anemia occurred in about 75% of patients with chronic liver disease due to acute or chronic gastrointestinal hemorrhage, and hypersplenism secondary to portal hypertension, bleeding and defective blood coagulation are the most severe causes of anemia, with a high mortality. Also aplastic anemia secondary to previous hepatitis or side effects of treatment of hepatitis with interferon and ribavirin [22].

In this study, chronic viral hepatitis group platelets count ranged from 83 – 160 $\times 10^3/mm^3$ with a mean of 122.7± 25.515, and in cirrhotic patients, platelets count ranged from 50 – 139 $\times 10^3/mm^3$ with a mean of 92.55± 26.482, while in HCC group platelets count ranged from 54 – 147 $\times 10^3/mm^3$ with a mean of 96.75± 26.347. These results showed thrombocytopenia in the three groups. Causes of thrombocytopenia in cirrhosis are portal hypertension-induced splenic sequestration, alterations in thrombopoietin, bone marrow suppression mediated by toxins (alcohol, hepatitis B and C), consumptive coaguloapathy and increased blood loss [28]. Anemia, neutropenia, leukopenia, and thrombocytopenia are among the numerous side effects of currently available HCV treatments by various mechanisms including, suppression of hematopoietic progenitor cell proliferation and activation of programmed cell death (apoptosis) in erythroid progenitor cells [30].

Alpha-fetoprotein is a protein of fetal component produced during the embryonic period by the visceral endoderm of the gestational sac and, later on, by the liver [31]. Any tumor arising from organs derived from the same endodermal lining as the hepatic diverticulum can be associated with elevations in serum AFP levels, including cancers of the stomach, pancreas and biliary tree. Pregnancy and non-seminomatous germ-cell tumors must also be considered. Chronic hepatitis or cirrhosis raise AFP in 20% and 40% of patients, respectively and tend to fluctuate in parallel with underlying inflammatory activity [32]. In this study, AFP in HCC infected patients was ranged from 407-3023ng/ml with a mean of 965.000±702.544ng/ml while in chronic viral hepatitis patients AFP ranged from 0.5-247ng/ml with a mean of 22.047±56.794 ng/ml and in cirrhotic patients it ranged from 0.5-16.5 ng/ml with amean of 5.657±4.969 ng/ml.

HCC can produce a range of AFP values from normal to>100000 ng/ml. Normal AFP levels were present in as many as 30% of patients at time of diagnosis and usually remain low, even with advanced HCC. AFP >400–500 ng/ml was considered diagnostic for HCC, although fewer than half of patients may generate levels that high. With values of that magnitude, the specificity of AFP was close to 100%, but at a cost to the sensitivity which fell below 45%.The positive predictive value (PPV) of AFP was low, ranging from 9% to 32% [33]. The role of AFP in the diagnosis and surveillance of HCC was getting smaller owing to the advances in imaging modalities [34].

Galectin-3 is a nonintegrin β-galactoside-binding lectin that has a role in tissue homeostasis and cancer progression. This protein is expressed in a variety of tissues and cell types and mainly found in the cytoplasm. Galectin-3 exhibits pleiotropic function, playing a key role in many physiological and pathological processes [35]. Increased galectin-3 expression was found to be related to cellular motility and extracellular matrix invasion, thus related with tumor metastasis [36]. Over expression of galectin-3 can cause mitochondrial homeostasis and protect the cell from damage and apoptosis. Although galectin-3 is minimally expressed in normal hepatocytes, it was found to be significantly highly expressed in the liver biopsies of patients with HCC or hepatic cirrhosis [37]. In addition, galectin-3 was abundantly expressed in cirrhotic liver in peripheral distribution within regenerating nodules, which may be a result of the high mitotic index [38].

A study evaluated the changes in serum galectin-3 concentration during liver diseases and the results were compared to hyaluronic acid concentration in combination with prothrombin index as a HAPRI index, showed that galectin-3 is a good marker of fibrosis in cirrhosis and toxic hepatitis, which reflects the stage of liver damage, like the HAPRI index. The serum galectin-3 concentration was significantly elevated in non-alcoholic cirrhosis group (mean±SD; 18.14±6.99 ng/mL) in comparison to the control group (9.90±2.21 ng/mL). For the non-alcoholic cirrhotic group, the cut off was 12.6 ng/mL, while sensitivity, specificity, PPV, NPV and accuracy were, 83.3, 90.0, 92.6, 78.3 and 86.0% respectively [39].

Another study was done to compare serum galectin‑3 levels in three patient groups with chronic hepatitis (HBV, HCV), hepatic cirrhosis secondary to HBV or HCV and HCC secondary to HBV or HCV and evaluated the role of galectin‑3 during HCC progression [(11)].Their study included 65 patients (HCC: 19 patients, cirrhosis: 22 patients, and chronic hepatitis B or C: 24 patients) and they considered the group of chronic viral hepatitis patients as the control group. There was no statistical difference between HCC and cirrhotic patients (P = 0.5) in case of galectin‑3 serum levels, but chronic hepatitis group statistically compared to cirrhosis and HCC (P < 0.001, P = 0.002, respectively).

In this study, serum galectin‑3 levels were found to be significantly higher in HCC and cirrhosis than in chronic viral hepatitis. In chronic viral hepatitis patients (Group 1) and there was a significant difference between the three groups (P < 0.001). As regard to chronic viral hepatitis and cirrhotic groups, the cut off for galectin-3 was > 12.6, while sensitivity, specificity, PPV, NPV and accuracy were 90.0, 95.0, 94.7, 90.5 and 97.7% respectively, while in chronic viral hepatitis and HCC groups, the cut off for galectin-3 was >12.6, while sensitivity, specificity, PPV, NPV and accuracy were 75.0, 95.0, 93.7, 79.2 and 89.9% respectively.

This study selected chronic hepatitis B and C patients as the control group as healthy individuals who were not exposed to hepatitis were not included. However, literatures showed no studies had been conducted so far to measure changes in galectin‑3 levels in case of hepatitis carriers in whom liver fibrosis or carcinogenesis has not yet begun.

## CONCLUSION

Serum galectin‑3 levels in patients with chronic HBV or HCV may guide us about progression to cirrhosis or HCC and prognosis of the disease. In these patients, if galectin‑3 levels were found to be high, serum alpha‑feto protein level and ultrasonographic examination could be repeated at more frequent intervals. This may also guide us in terms of the treatment plan.

Measurement of galectin-3 in sera of large number of patients and follow up may pave the way to pick up early fibrosis and showed its prognostic effect.

## RECOMMENDATIONS

- It was recommended to measure changes of Galectin-3 in hepatitis carries.
- Measurement of Galectin-3 in a large scale of patients to explore its prognostic value.

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