Summary and Conclusion

Chronic hepatitis C is one of the major prevalent infectious hepatic diseases in Egypt where the highest prevalence in the world.

AFP is a glycoprotein, produced by the liver and its level decreases below 10ng/ml after birth. It was observed that AFP levels are elevated in chronic HCV infection, liver regeneration and significantly elevated in patients with HCC.

In present study, we aimed to construct one simple model consisting of routine laboratory data to predict the response to treatment with interferon and ribavirin therapy. We have focused on evaluating AFP as a practical, non invasive, inexpensive and simple biomarker method. This is especially important in Egypt where there is a heavy chronic liver disease burden since diagnosing sever fibrosis or cirrhosis could initiate strategies for treatment of HCV and screening for hepatocellular carcinoma.

This retrospective study was conducted on 1000 patients with chronic hepatitis C who attended the Liver Research Unit at Tanta fever Hospital, Egypt, during March 2008 to July 2009 and in whom antiviral therapy was initiated during this period.

They were 348 females and 652 males with their age ranged between 24-55 years with mean age (43.07$\pm$8.56) years.

All patients were subjected to history taking, clinical examination, routine laboratory investigations as aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase enzymes, bilirubine, albumin, platelets count, white blood cells count and haemoglobin.

Measurement of serum HCV RNA were done before starting treatment and six months after treatment.Hepatitis C and B markers by ELISA technique, abdominal ultrasonography for liver and spleen assessment and for presence or absence of ascites had been done.

Liver biopsy was done before starting treatment to determine the degree of fibrosis so as to be able to make the decision of treatment.

Serum AFP levels are estimated before treatment and at 24 and 48 weeks after treatment.

The results of the study revealed significant increase in AFP in patients with chronic hepatitis C specially those with fibrosis. The severity of liver fibrosis was correlated significantly with gradual increase of AFP level (p-value=0.05). There were also significant positive correlation between AFP levels and response to antiviral therapy. Low levels of AFP before starting treatment predict better response to antiviral therapy including pegylated interferon and ribavirin treatment. There was significant positive correlation between AFP levels and PCR at 24 weeks of treatment (p-value =0.044) and at 48 weeks of treatment (p-value =0.021). There were significant positive correlation between stage of fibrosis and the response to treatment at 48 weeks, as increased the severity of liver fibrosis, the low response to treatment (p-value=0.021).

Conclusion:

The identification of AFP as a bio-marker that may predict virological response to anti-HCV treatment is a very important tool for the correct management of the condition. Our findings suggest that pretreatment levels of AFP should be included in the routine assessment of factors potentially predictive of the anti-HCV treatment outcome.

A prolonged follow-up is required to confirm the predictive role of AFP levels at baseline in determining EVR, ETR and sustained virological response to anti-HCV treatment.

Serum AFP levels may be appropriate marker to give an idea about liver dysfunction in patients with chronic hepatitis. It can also provide guidance as to whether the patient has minimal hepatic fibrosis or more sever lesion which may assist in making the therapeutic decision.

Serum AFP levels can be used as a marker for early detection of HCC but not alone as it is recommended to be used with other image studies.