

Hepatic Profile in the Patients receiving hepatitis treatment

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ABSTRACT

The aim of the study was to evaluate the hepatic profile in the patients suffering from hepatitis C and receiving interferon treatment (after two months). Individuals were divided into two groups including control (n=10) and patients (n=45). The hepatic profile (ALT, AST, ALP, total protein and bilirubin) was determined by commercially available kits. Variations in the hepatic profile was statistically significant ($p < 0.05$) between the control and patients groups. It can be concluded that continuous attention towards the patients regarding the hepatic profile must be delivered while they are receiving long therapy including interferon. Moreover, hepatic protective treatment should also be considered.

Keywords: Hepatic profile, hepatitis C, interferon

INTRODUCTION

Hepatitis C is a life threatening disease caused by virus. Scientists are making more and more efforts to discover the proper treatment of this problem. Development of Interferon alpha (IFN α) was a big achievement against HCV. The mode of action of Interferon alpha described that I is a potent cytokines which after binding with IFN α receptor activated number of signaling pathways and ultimately blocked the virus RNA replication (Jonasch and Haluska, 2001, Parmar and Plataniias, 2003). Different researches believed that IFN α is an essential mediator of the innate antiviral immune response and produced very prominent effects on cellular physiology and cells of the immune system (Biron and Sen, 2001; Samuel, 2001). It has been proved by different vivo and vitro studies that IFN α improve the liver histology and reduces the chances of hepatocellular carcinoma in chronic HCV patients (Yoshida *et al.*, 2004, George *et al.*, 2009). According to different studies it is concluded that about 50% patients with chronic hepatitis C did not show fully clearance of hepatitis C virus sustain with IFN α . However there is strong evidence that IFN α when used therapeutically in chronic hepatitis C patients can participate or exacerbate autoimmune endocrine diseases, especially of thyroid gland. Mostly about 40% patients discontinue the treatment of IFN α . It is also documentary proved that about 14% HCV patients develop thyroid dysfunction. The aim of the study was to evaluate the hepatic profile in the patients suffering from hepatitis C and receiving interferon treatment (after two months), the continuation of the previous research article.

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MATERIALS AND METHODS

The current study was conducted at Jinnah hospital, Lahore from January-2015 to April-2015. Total 45 individuals were selected suffering from hepatitis C and receiving interferon treatment and 10 individuals were selected as control group. 1ml blood was taken from all the individuals and plasma was separated from the blood for the evaluation of hepatic profile. The patients have completed their two months interferon therapy. Patients were reported with various side effects since they were treated with interferon. Some of the patients were reported with diabetes and some were reported with decreasing their weight. The thyroid gland test were evaluated and it was found that these individuals also had a little bit thyroid malfunctioning. Some of the patients stopped their treatment and some of them showed intension to complete interferon therapy. The hepatic profile (ALT, AST, ALP, total protein and bilirubin) was determined by commercially available kits.

RESULTS

Table 1: Comparison of hepatic profile

	N	Mean	Std. Deviation	P value
ALT (IU/L)				
Control	10	22.60	5.08	.000
HCV	45	110	49.30	
AST (IU/L)				
Control	10	18.30	3.40	.000
HCV	45	94.80	41.44	
ALP (IU/L)				
Control	10	80.95	10.67	.000
HCV	45	168.90	25.10	
Bilirubin (mg/dl)				
Control	10	0.84	0.34	.012
HCV	45	3.25	2.90	
TP(g/dl)				
Control	10	6.32	0.34	.218
HCV	45	6.56	0.60	

The hepatic profile of the patients receiving interferon therapy after two months showed statistically significant variation (Table 1) among the various hepatic parameters. The level of ALT (110 ± 49.30 IU/L), AST (94.80 ± 41.44 IU/L), ALP (168.90 ± 25.10 IU/L), bilirubin (3.25 ± 2.90 mg/dl) and total protein (6.56 ± 0.60 g/dl) was elevated in the patients receiving interferon as compared to control (Table 1).

DISCUSSION

Liver enzymes are localized in the cell cytoplasm and cell mitochondria as well as in the bile. Increased levels of serum AST, ALT, ALP and total bilirubin are indicative to cellular damage and loss of functional integrity of cell membrane in liver. Therefore in our study, the levels of three liver enzymes ALT, AST, ALK and total bilirubin in patients with interferon induced thyroid diseases were estimated and compared with controls. In our recent research serum levels of ALT and AST and ALP were found elevated in hypothyroid and hyperthyroid state after interferon therapy.

Hepatitis C is one of the main reasons of chronic liver infections and cirrhosis worldwide. Interferon alpha has been widely used as a curative agent for many infectious and malignant diseases including chronic hepatitis C (Fried *et al.*, 2002). A major and common adverse effect of interferon therapy is the development of thyroid disease. In patients treated with IFN, the activation of the immune system is important for the development of thyroid disease. Moreover, IFN has direct inhibitory effects on thyroid hormone synthesis, release, and metabolism by altering the anomalous expression of major histocompatibility antigens on thyroid cells and supporting the cytokine microenvironment, which may lead to the immune-mediated damage of thyroid tissues (Wang *et al.*, 2002).

Thyroid Disease can be caused by many factors including hyperthyroidism (too much thyroid hormone) and hypothyroidism (too little thyroid hormone) released by the thyroid gland. The direct

link between hepatitis C and thyroid disease (usually hypothyroidism) is unclear, but thyroid disease is more commonly seen in people with hepatitis C than in the general population. HCV treatment can also induce thyroid disease, but thyroid function will return to normal for about 95% of people who develop treatment-related hypothyroidism when treatment is stopped.

REFERENCES

1. Fried MW, ML Shiffman, KR Reddy, C Smith, G Marinos and FL Goncales (2002). Peginterferon alfa-2a plus ribavirin for chronic hepatitis C virus infection. *NEngl J Med.* 347(13):975-982.
2. Wang SH, JD Bretz, E Phelps, E Mezosi, PL Arscott, S Utsugi, JR Baker (2002). A unique combination of inflammatory cytokines enhances apoptosis of thyroid follicular cells and transforms nondestructive to destructive thyroiditis in experimental autoimmune thyroiditis. *J Immunol.* 168:2470-2474.
3. Biron C, Knipe D, Howley P, Griffin D, Lamb R, Martin M, Straus S and Sen GC. Interferons and other cytokines. *Fields virology.* Lippincott Williams & Wilkins, Philadelphia 2001; pp. 321-351.
4. George SL, Bacon BR, Brunt EM, Mihindukulasuriya KL, Hoffmann J and Bisceglie AM. Clinical, virologic, histologic, and biochemical outcomes after successful HCV therapy: a 5-year follow-up of 150 patients. *Hepatology* 2009; 49: 729-738.
5. Jonasch E and Haluska FG. Interferon in oncological practice: review of interferon biology, clinical applications, and toxicities. *Oncologist* 2001; 6(1): 34-55.
6. Parmar S and Plataniias LC. Interferons: mechanisms of action and clinical applications. *Curr Opin Oncol* 2003; 15(6): 431-439.
7. Samuel CE. Antiviral actions of interferons. *Clin Microbiol Rev* 2001; 14: 778-809.
8. Yoshida H, Tateishi R, Arakawa Y, Sata M, Fujiyama S, Nishiguchi S, Ishibashi H, Yamada G, Yokosuka O, Shiratori Y and Omata M. Benefit of interferon therapy in hepatocellular carcinoma prevention for individual patients with chronic hepatitis C. *Gut* 2004; 53: 425-430.