hepatocytes are more likely to develop chronic hepatitis followed by hepatocellular carcinoma.

Lymphotoxins are implicated indirectly in the induction of endothelogenesis, lymph-angiogenesis and inflammation by their direct action on NK cells to activate stromal cells and the production of Vascular endothelial growth factor A and C (VEGF-A, C) which are a crucial inflammatory mediator [14,15]. In addition, the production of chemokines and adhesion molecules by LT α could be implicated in the recruitment of macrophages which produce VEGF-C. On the other hand, tumor necrosis factor has been shown to up-regulate the expression of VEGF-C by macrophages [16].

Many investigations have documented the role of LTα in host defense mechanisms and reaction to infections, as mice deficient of this cytokine increases their susceptibility to infection with *Staphylococcus aureus* [17]. It was also reported that LTα is required for the granuloma formation and resistance to infection by *Mycobacterium*, *Leishmania*, *Plasmodium and Toxoplasma gondii* infections in mice [18-21]. In a study on transgenic mice it has been proposed that LTα plays a smaller role in the maintenance of lymphoid organs and has no direct involvement in the regulation of TNF [22].

In vivo and *in vitro* studies have indicated that infection with HBV or HCV leads to an increase in the LT expression in hepatocytes [23,24]. Another study performed *in vitro*, revealed that components of LTβR signaling pathway are required for HCV replication [25].

This study has revealed that there is no correlation between lymphotoxin α and the gender of patients, HAI or the stage of the disease. On the other hand, a significant correlation exists with that of the age of patients. This age related increase of incidence of hepatic neoplasia in HCV infected subjects appears to be a attributed to the inherent decline of the immune system and macrophage surveillance in old patients in addition to the increasing incidence of mutations of HCV infected hepatocytes [26].

Hepatocellular carcinoma is considered as the most common primary liver malignancy where the average age at diagnosis ranges from 60 to 80 years. During infection with HCV hepatic cirrhosis develops replacing injured liver cells. Formation of regenerative nodules is one of the healing processes that are usually happening in cases of hepatic cirrhosis and adenomatous hyperplasia. Development of the neoplasm is believed to evolve following cellular mutations that happen at the regenerative nodules which are then transform into malignancy [27].

The results of lymphotoxin β staining, shown in table 2, this result revealed increase in the expression of lymphotoxin β among studied group. This agrees with many authors who indicate that lymphotoxin β is expressed in chronic liver injury [25,28], and with Heliken-Walder *et al.*, (2005) [29] who indicate high expressed LTα and -β in the liver during analysis of two transgenic mouse lines. On the other hand this study did not reveal significant correlation between the positive signals of lymphotoxin β and different clinicopathological variable.

Lymphotoxin α is recognized by the same receptor of tumor necrosis factor and lymphotoxin beta is recognized by its receptor [30].

The current result demonstrated a significant increase in the cellular expression of lymphotoxin β receptor (TLβRs), among patients with chronic HCV infection and those with hepatocellular carcinoma. Previous studies demonstrated the role of LT-α1β2/LT-βR in the transduction of both apoptotic and non-apoptotic signaling pathways [31,32]. Moreover, it has also been reported that activation of LT-β receptor can induce inflammation through the production of chemokines and endothelial adhesion molecules necessary for recruitment of lymphocytes to sites of insult [33-35].

The work by Ruddell and colleagues demonstrated that LT β R signaling regulates hepatic stellate cell function and hepatic wound healing as well as controlling liver homeostasis in both health and disease [36].