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Assessment of IL-23 serum levels on hepatitis-C infected patients

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Abstract---This study includes 100 total samples divided into 40 control, 60 patients suffering from HCV infection from Al-Saddar Medical City in Najaf governorate from December 2020 to May 2021, aging (20-70) years old and has shown higher frequency of hepatitis C infection in males 35sample (58 %), also the age group of (40-50) years more frequency. Moreover, it was observed that the concentration of Interleukin 23 (IL-23) (pg/ml) in (60) patients with diabetic foot ulcer infection was increase significantly over (P value was (< 0.0001))

Keywords---HCV, interleukin 23, serum level.

Introduction

HCV infection is a global health problem that affects >184 million people worldwide. HCV is associated with several hepatic and extrahepatic disorders, including several malignancies. The burden of HCV-related disorders is influenced by the number of new and existing cases, number of existing cases and the natural history of the infection. The natural history of HCV is affected by several demographic, virological, clinical and lifestyle factors (Thrift, et al., 2017). Hepatitis C virus (HCV) is a hepatotropic RNA virus that causes progressive liver damage, which might result in liver cirrhosis and hepatocellular carcinoma. Globally, between 64 and 103 million people are chronically infected. Major risk factors for this blood-borne virus infection are unsafe injection drug use and unsterile medical procedures (iatrogenic infections) in countries with high HCV prevalence (Manns, et al., 2017). Interleukin-23 (IL-23) is a pro-inflammatory cytokine composed of two subunits, p19 and p40. The p40 subunit is shared with IL-12. IL-23 and IL-12 have different receptors and different effects.

Whereas IL-12 induces development of Th1 cells, which produce interferon- γ , IL-23 is involved in differentiation of Th17 cells in a pro-inflammatory context and especially in the presence of TGF- β and IL-6 (Lee, et al., 2015). Activated Th17 cells produce IL-17A, IL-17F, IL-6, IL-22, TNF- α , and GM-CSF. Inflammatory macrophages express IL-23R and are activated by IL-23 to produce IL-1, TNF- α , and IL-23 itself. These effects identify IL-23 as a central cytokine in autoimmunity and a highly promising treatment target for inflammatory diseases. IL-23 is found in the skin of patients with psoriasis, in the bowel wall of patients with chronic inflammatory bowel disease, and in synovial membrane of patients with rheumatoid arthritis (Kurzeja, et al., 2011). IL-23 is expressed and secreted by professional antigen-presenting cells (APCs), chiefly dendritic cells, macrophages and monocytes. Epithelial cells were also shown to contribute to IL-23 production.

These include keratinocytes, intestinal epithelial cells [4] and glomerular podocytes (epithelial cells in the Bowman's capsule especially during nephrotoxic serum (NTS) nephritis (NTN)). Furthermore, human fibroblast-like synoviocytes (ex vivo and in vivo) and human colon subepithelial myofibroblasts were shown to produce IL-23 upon IL-1 β and TNF- α all of which suggest that non-hematopoietic sources may also contribute to IL-23 production to some extent, given the right stimulation (Oukka, et al., 2016). IL-23 exposure programs Th17 cells transcriptionally to have a unique effector cytokine profile compared to nonpathogenic Th17 cells which are not exposed to IL-23. Unlike nonpathogenic Th17 cells, which express only IL-17, IL-23-activated pathogenic Th17 cells express IFN- γ and GM-CSF in addition to the IL-17. Various lines of evidence suggest that Th17 cells, and hence IL-23R signaling, is critical for the development of chronic inflammatory conditions (Gaffen, et al., 2014).

Results and Discussion

The current study includes 100 samples, which were separated into groups for further analysis in the AL-Sader hospital in Al-Najaf City, Iraq, HCV patients = 60 (60%), and healthy control 40 (40%). According to the results of the study, the male patients was 35(58 %) and female 25(42 %) infected with HCV. For control samples male constitute 24 (60%) samples while female 16 (40%) sample .This study showed age group ranging (20 years-70 years) comprises 40 female and 50 male .

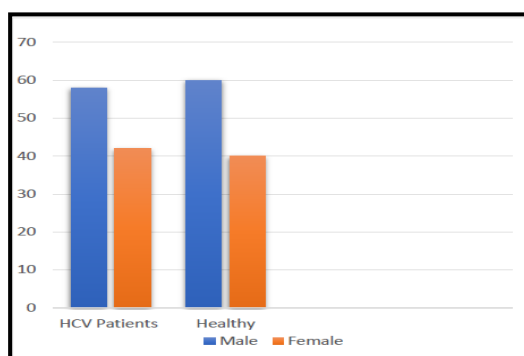


Figure 1. Distribution of patients with and without HCV according to sex.

Increased incidence of HCV in male deal with the results of (Puga, et al., 2017) they mentioned that (15.4%) were female and 2,848 (84.6%) were male infected with HCV. Sex disparity in incidence is notable in almost all countries, with rates among males being 2- to 3-fold higher than rates among females (Ozakyol, 2017). Incidence HCV among male repeat testers exceeded that of female repeat testers across all years, although the gap narrowed over time. From 2000 to 2011, there were approximately 15% more female than male repeat testers each year (Kuo, *et al.*, 2015). The prevalence of males HCV infection significantly higher than that of females in 2011 (male 56.62 % vs female 43.38 %,) and 2012 year (male 53.29 % vs female 46.71 %), respectively (Niu, *et al.*, 2016). Patients were placed into three groups based on their age ranges, with each group being separated into two groups. According to figure (2) the distribution of HCV according to age groups that present 40-50 had the highest affected group, followed by those aged 50-60, 30-40, 60-70, and those aged 20-30, was the less affected age group.

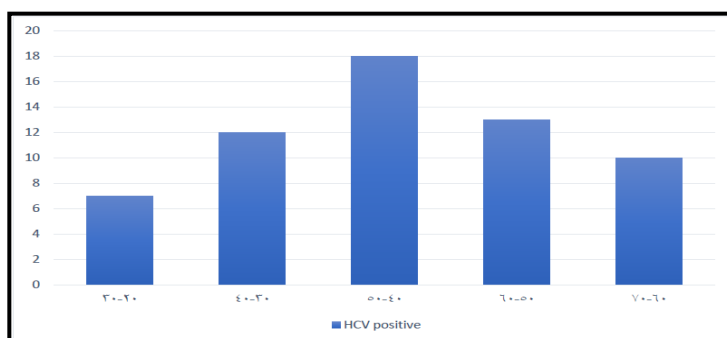


Figure 2. Distribution of the HCV patients according to the age group

The prevalence of HCV infection showed different variations with age, as the highest incidence was shown for the age group 50–59 (25.85 %) and the lowest prevalence was 0–9 (0.93 %) (Niu, *et al.*, 2016). Among both males and females, a bimodal age distribution was observed with infections highest among persons aged 20–39 years (peak: 29 years) and a second apex around 55–70 years (peak: 59 years) (CDC, 2019). Sixty positive samples for HCV were measured by ELISA according to the methods that is explained in previous chapter. The result were obtained by ELISA reader device by measuring optical density for the examined samples. The result were separated for both control group and patients group and then the result were converted from O.D to concentration.

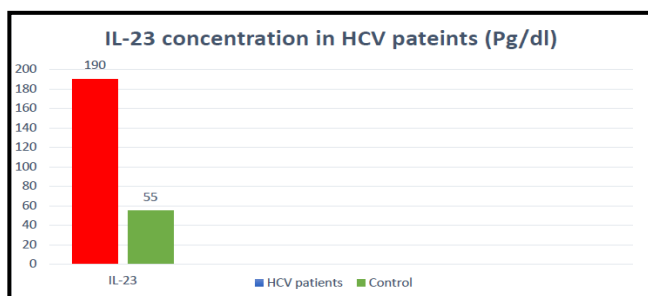


Figure 3. Concentration of IL-23 in serum of HCV patients, and control group

Our result statically evaluated to find if there is a significant effect of IL-23 on HCV patients in comparison to healthy control persons and the results was enrol standard error for HCV patients 146.3 ± 2.520 and for control group it was 48.36 ± 0.7858 and P value was < 0.0001 ***.

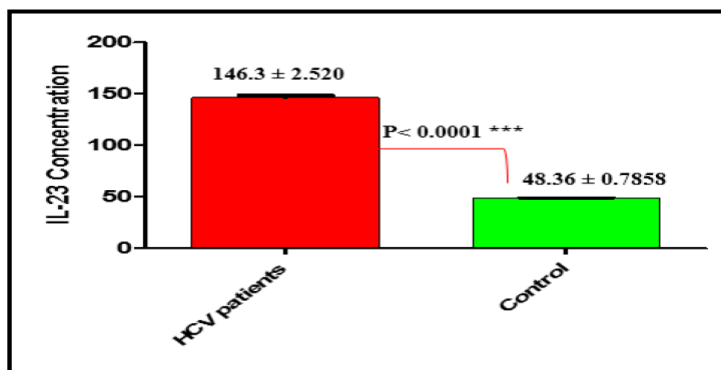


Figure 4. This figure show statically analysis of IL-23 result

The individuals with HCV outcome had statistically higher serum levels of IL-23 than controls ($P < 0.01$). Further analysis in HCV tissues showed that CD14⁺ inflammatory macrophages were the major IL-23 producers (Meng, *et al* 2018).

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