

INTRODUCTION

Hepatocellular carcinoma (HCC) is one of the most common causes of death from cancer accounting for over one million deaths per year world wide. It represents about 83% of all primary malignant hepatic tumours (*Edmondson and Craig, 1987*).

In Egypt, the incidence of HCC was 3.7% of the total number of malignant cases received at the pathology department, national cancer institute (NCI), Cairo University in 1985 - 1987 period (*Abdel-Razek, et al., 1989*).

Interleukin 8 (IL-8) and granulocyte macrophage colony stimulating factor (GM-CSF) are important mediators of inflammation and immune response in human diseases (*Al-Wabel, et al. 1995*).

It was reported that GM-CSF is increased in some patients with liver disease and it may play a significant role in host defence and disease. *Itch et al. (1994)*, investigated the serum levels of GM-CSF in acute and chronic liver diseases. It was significantly higher in acute hepatitis and chronic active hepatitis (CAH) than in healthy volunteers. These findings suggest that the serum level of GM-CSF represent ongoing hepatocellular necrosis in acute and chronic liver disease.

Martin et al. (1994) reported that the administration of recombinant human GM-CSF may induce reduction in hepatitis B virus DNA levels, perhaps by altering the immune status and increasing cytokine production.

Interleukin-8 was referred to as neutrophil activating factor as it strongly attracts and activates neutrophil (*Hebert et al., 1990*). It was found to increase in certain pathological conditions and it is involved in the pathogenesis of inflammatory infections and neoplastic diseases (*Al-Wabel et al., 1995*).

AIM OF THE WORK:

The aim of this work is to detect the level of IL-8 and GM-CSF in hepatocellular carcinoma patients and study the role of these cytokines in the pathophysiological process of the disease.