**BACKGROUND:** Thrombocytopenia (TP) in chronic hepatitis C virus (HCV) is a common finding either directly due to viral infection of platelets or indirectly due to immune alteration triggered by the virus, the consequences of HCV-induced cirrhosis and portal hypertension, or induced by Interferon (IFN), the corner element of the standard of care (SOC) therapy for HCV.

**AIM OF THE WORK:** The aim of this study was to evaluate TP in patients with chronic HCV, and to mutual effect between SOC and TP.

**PATIENTS AND METHODS:** The study was conducted on 209 patients with chronic HCV from Railway Hospital, Cairo. Patients were divided into two groups, group (I): 144 patients who received SOC therapy, and group (II): 65 patients who were unfit for therapy served as control group. All patients were subjected to clinical examination, laboratory investigations [complete blood count (CBC), liver biochemical profile (LBP), viral load by PCR] and abdominal ultrasonography.

**RESULTS:** TP was a common finding (60/209; 28.7%), more in group I (33/60; 55%), and conversely, TP was significantly worse in group II (p=0.008). Along the course of treatment, 2 significant drops of platelet count took place, nadirs at W8 and W24. TP was significantly related to hepatitis activity and hepatic synthetic function, and not related to the viral load, whether pre-treatment or along the course of therapy. Four cases developed severe TP along SOC. Only 1 of them continued therapy on IFN dose reduction.

**Conclusions:** Moderate and severe TP constitute an evident barrier to candidacy of therapy. TP is a common complication along SOC therapy, influenced significantly by splenomegaly and advanced fibrosis.

**Key words:** Hepatitis C virus (HCV) – Thrombocytopenia (TP) - Interferon (IFN).