Introduction

Platelets are produced in the bone marrow; the progenitor cell for platelets is the megakaryocyte. This large, multinucleated cell sheds platelets into the circulation. Thrombopoietin (c-mpl ligand) is a hormone, mainly produced by the liver, that stimulates platelet production. It is bound to circulating platelets; if platelet levels are adequate, serum levels remain low. If the platelet count is decreased, more thrombopoeitin circulates freely and increases marrow production**(Rob etal 2005).**

The circulating life of a platelet is 9-10 days. After this it is sequestered in the spleen. Decreased function (or absence) of the spleen may increase platelet counts, while hypersplenism (overactivity of the spleen, e.g. in Gaucher's disease or leukemia) may lead to increased elimination and hence low platelet counts**(Robert etal 2005).**

Blood platelets have several important functions. They adhere to sites of vascular injury, generate biological mediators, secrete their granule contents, form multicellular aggregates and serve as a nidus for plasma coagulation reactions. In order to carry out these tasks, the platelet undergoes dramatic structural rearrangements, utilizes multiple membrane receptors, which bind small molecule mediators, adhesive glycoproteins and constituents of the vascular subendothelium, and activates a network of complex signaling pathways. All of these events

occur within seconds of vascular injury. Collectively, they help to maintain the integrity of the vascular system. It should not be surprising that mutations occasionally arise that perturb these complex reactions and lead, in some cases, to disordered hemostasis. While inherited disorders of platelet function are relatively rare, they have provided important information about normal platelet physiology**(Robert etal 2005).**

Thrombocytopenia is a frequent complication of chronic liver disease and is considered an indicator of advanced disease. The low platelet count is due partly to the effects of portal hypertension and hypersplenism, decreased thrombopoietin production, and virus-induced bone marrow suppression**(Giannini2006) (G,Bordin G,Ballare and M,Zigrossip etal 1995)**.

Patients with chronic liver disease due to infection with the hepatitis C virus (HCV) who have thrombocytopenia (<75,000 platelets per cubic millimeter) have been routinely excluded from clinical trials of interferon and ribavirin, and few published reports have described the treatment of chronic HCV infection in patients with platelet counts of less than 50,000 per cubic millimeter. Although a reduced platelet count is not an absolute contraindication to treatment with pegylated interferon (peginterferon) and ribavirin, product labels advise that caution be used in treating patients with clinically significant thrombocytopenia. Furthermore, if thrombocytopenia develops during antiviral therapy, peginterferon may need to be delivered at a reduced dose or discontinued. Currently, there is no

approved treatment for thrombocytopenia in patients with HCV infection**(shiffman Ml,Ghany MG andMorgant Retal 2007).**

Persistent hepatitis C virus (HCV) infection evokes autoimmune response including production of autoantibodies and concomitant autoimmune disorders. Numerous types of autoantibodies such as non-organ-specific autoantibodies and liver-specific autoantibodies have been identified in sera of patients with HCV-related chronic liver disease (CLD). The production of these autoantibodies in HCV-related CLD reflects "virus-induced autoimmunity." Molecular mimicry between the HCV polyprotein and self-proteins, and polyclonal B cell activation by chronic HCV infection have been proposed as possible mechanisms for the occurrence of autoantibodies in HCV-related CLD. Some autoantibodies are tightly associated with concurrent autoimmune diseases, and others closely associated with peculiar human leukocyte antigen (HLA) haplotypes. Changes in the titers of autoantibodies during the antiviral treatment may predict the sustained virological response in individuals. In this article, we mainly focus on the interpretations of autoantibodies in HCV-related CLD **(Takashihimoto and Mikionishioka etal 2008)**

Human interferon (IFN) is the standard therapy for chronic hepatitis C to prevent its progression to liver cirrhosis and hepatocellular carcinoma. Thrombocytopenia is one of the major adverse effects of IFN- and often leads to dose reduction or treatment discontinuation. However, there is little information on how IFN inhibits human megakaryopoiesis **(Akikio yamane , Takanori nokamera HIdenori suzukirtal 2008).**