INTRODUCTION

Chronic hepatitis C virus (HCV) infection affects approximately 300 million people worldwide and currently is the most frequent cause for liver transplantation in the United States and Europe (**Lauer and Walker, 2001**). Natural history studies suggest that up to 20 % chronic HCV patients develop liver cirrhosis after 20year of infection. Moreover, The incidence of chronic liver failure is expected to increase over the next 10 years as a result of the "silent epidemic" of HCV infection (**Seeff, 2002**)

The recommended treatment for patients with HCV genotypes 1 and 4 is pegylated interferon plus ribavirin for 48 weeks (**Fried *et al.*, 2002)**. Such treatment has yielded overall sustained viral response (SVR) rates of 54-63 % in randomized controlled phase III clinical trials (**Hadziyannis *et al*. 2004 and Zeuzem *et al*. 2005)**. However, treatment responses are not uniform across all populations, and are dependent on various viral and host factors (**Dienstage and Mchutchison, 2006**)

Treatment of patients with chronic hepatitis C virus (HCV) infection remains suboptimal, with the current pegylated interferon (PEG-IFN) and ribavirin combination therapy providing sustained viral response (SVR) rates of 54 - 63 %. The aim of this study was to identify clinical, laboratory and histological findings that can predict non-response to this treatment (**Nachnani *et al.*, 2007**)

The aim of treatment in chronic hepatitis C is to achieve a sustained virological response (SVR) defined as undetectable HCV RNA with a sensitive PCR assay (<50 IU/ml) 24 weeks after end of antiviral therapy. In patients who achieved an SVR following standard interferon (IFN) – based antiviral therapy, virological relapse after 5 years of follow – up was observed in 2-4 % only, and no relapse was reported after 5-10 years (**Veldt *et al*., 2004**). Moreover, the 5- year durability of an SVR was in excess of 99 % in patients treated with pegylated (PEG) IFN. A number of host and viral factors have been identified that influence treatment outcomes (**Swain *et al*., 2007 and Lindsay *et al*. 2008**)

Review of the fate from a large multi-centre trial evaluating PEGIFN RBV combination therapy showed equal or worse AEs in almost all areas compared with standard IFN RBV combination. (**Manns Mp, *et al* 2001**).

The most prominent among these AEs were injection site reactions and dose reductions for neutropenia. A similar comparison of adverse events with PEG-IFN a-2a/RBV combination therapy shows somewhat less side-effects that standard combination therapy. (**Fried Mw, *et al* 2002**) It is difficult to determine the importance of these trial differences in clinical practice until a significant clinical experience with both compounds has accumulated. It will also not be possible to accurately compare the respective side-effect profiles of these two pegylated compounds with one another until a head –to-head trial is done.

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