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 20 years 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).
Introduction

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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