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Oral presentation

O232 What's new in hepatitis C?

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Exposure to hepatitis C virus (HCV) is frequently followed by chronification. Contagion is more common through following parenteral exposure (e.g., intravenous drug use) than after sexual contacts. Since there is no protective immunity against HCV, re-infection after spontaneous HCV clearance accounts for a greater rate of chronicity in IDUs compared to persons sexually exposed to HCV. Current treatment of hepatitis C is based on the combination of weekly subcutaneous pegylated interferon plus daily oral ribavirin. Overall 75% of HIV patients infected with genotypes 2 or 3 and around 40% of subjects infected with genotypes 1 or 4 can achieve sustained virological response, which means eradication of HCV. Patients with known baseline serum HCV-RNA respond more than those with high baseline HCV levels, regardless of HCV genotype. The length of treatment must be adapted to patient's features, being recommended to be for 6 months in HCV genotypes 2/3 with low viremia and lack of cirrhosis, while it should be extended beyond 12 months in HCV genotypes 1/4 with high baseline viremia or lack of rapid virological response (negative serum HCV-RNA at week 4 of treatment). High exposure to ribavirin is associated with higher antiviral effects; however, haemolytic anemia is the most important limiting side-effect of ribavirin and often precludes the use of high dosing. Adjuvant weekly subcutaneous erythropoietin may help to manage anemia in some situations. However, the PERICO trial has shown that pre-emptive use of erythropoietin does not enhance virological response rates, most likely due to erythrocyte ribavirin sequestration. Due to inhibitory competition in the phosphorylation pathway within the cells as guanosine analogues, ribavirin should not be prescribed along with abacavir. On the other hand, the use of didanosine must be avoided along with ribavirin since toxic didanosine metabolites are increased, with enhanced risk of mitochondrial toxicities including lactic acidosis, hepatic decompensation and pancreatitis. The prospects for new specific anti-HCV agents are coming slowly. HCV protease inhibitors (e.g. telaprevir), nucleoside analogs (e.g. R-1626), and four different classes of non-nucleoside HCV polymerase inhibitors are rapidly progressing in clinical development. Drug resistance in HCV will be very challenging for most of these agents, and accordingly, as in the HIV field, combination therapy is the way to go on. Although the new drugs will be combined with pegylated interferon and ribavirin in a first moment, the prospects for combining new agents moving off interferons are eagerly searched.