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NASH AFTER STEROID TREATMENT FOR ULCERATIVE COLITIS EXACERBATION

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A 25 year old man with a history of UC was admitted in our hospital for the occurence of bloody diarrhea. Blood exams showed normal ALT (32 U/L) and AST (27 U/L), and increased ERS (45 mm). Abdominal ultrasound where performed with no evidence of liver diseases. Colonoscopy showed left colitis consistent with UC exacerbation. He started 40 mg/day of methylprednisolone therapy with improvement of his symptoms. Therapy was tapered to 16 mg in 4 weeks. After 40 days he performed blood exames and a raise of ALT value to 106 was observed. AST remained normal (41 U/L). An abdominal ultrasound examination was repeated with the evidence of fat infiltration consistent with liver steatosis. Hepatitis virus (HAV, HBV, HCV, CMV and EBV) and antibodies against mitochondria, smooth muscle and nuclear antigens were not detected by serological tests performed. Liver biopsy showed macrovescicular hepatocellular fat accumulation, periportal inflammation and mild fibrosis. The patient denied a history of alcohol abuse and a diagnosis of nonalcoholic steatohepatitis was made. He stopped the steroid treatment in two weeks and ALT value gradually decrease to normality. After six months abdominal ultrasound showed a normal liver. Steatohepatitis appears after a short course of steroid for ulcerative colitis. Although drug discontinuation seems effective to induce remission of this condition a careful follow up of patients submitted to steroids has to be performed for the possibility to develop liver failure.

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PREVALENCE OF HELICOBACTER PYLORI CAG-A POSITIVE STRAINS INFECTION IN NON-ALCOHOLIC STEATOHEPATITIS.

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Background and aim: non-alcoholic steatohepatitis (NASH) is a chronic liver disease that can run its course towards cirrhosis. Genetic and metabolic factors, largely unknown, are likely involved in its pathogenesis. Some authors hypothesised that the Helicobacter Pylori (HP) might promote progressive liver disease by inducing pro-inflammatory cytokines production in the portal system. The seroprevalence of HP infection in subjects with NASH is presently unknown. Aim of our study was to evaluate the prevalence of *H. pylori* infection and of more virulent strains in patients with NASH. **Material and Methods:** Thirteen consecutive male patients, aged 35.6±7.5 yr (mean±SD), with a clinical and histological diagnosis of NASH, were studied. The control group consisted of 13 male blood donors, matched for age (mean 34.8±3.6 years), resident in the same area and comparable for socio-economic status. Patients and controls were tested for serum IgG against *H. pylori* and the CagA antigen (ELISA Helori-test® Eurospital, Trieste, Italy). **Results:** Antibodies against *H.pylori* were present in 3/13 (23%) patients and 4/13 (31%) blood donors (p= n.s.). The anti-CagA antibodies were detected in 3/3 (100% of infected patients, 23% of all cases) patients compared to 1/4 (25% of infected controls, 7,7% of all controls) in the blood donors. **Conclusions:** the prevalence of *H. pylori* infection in NASH group and in controls was similar in our study. However, the impressive prevalence of more virulent strains in patients with NASH needs to be further investigated.

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TEN YEARS EXPERIENCE OF TREATMENT OF PATIENTS WITH CHRONIC HEPATITIS C IN CLINICAL PRACTICE

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Aim: To retrospectively compare the efficacy, the tolerability, in terms of discontinuation of therapy (DT), and the predictor factors of therapy success of three regimens of treatment for chronic hepatitis C, in current clinical practice. End points considered were: 1) SVR: sustained viral responses; 2) R: relapse; 3) NR: Nonresponse; 4) DT
Patients and methods: We retrospectively analyzed the data of 164 consecutive naive patients treated from 1992 to 2001. Group A: 109 pts received recombinant alpha Interferon 6MU Tiw for 12-16 weeks followed by 3MU Tiw, over a period of 48 weeks. Group B: 28 pts received recombinant alpha Interferon 6 MU Tiw for 12-16 weeks followed by 3MU Tiw , plus 1000-1200 mg Ribavirin over a period of 24 (genotype 2-3) or 48 weeks; Group C: 27 patients received Peg-Interferon alpha-2b 80 mcg Ow (27 pts) , plus 1000-1200 mg Ribavirin over a period of 24 (genotype 2-3) or 48 weeks.
Results: The characteristic of patients in the treatment groups were similar (age, sex, duration of infection, HCV genotype, histologic stage of hepatic disease: p<0,05). The results are shown in the following table:

	IFN (group A)	IFN + RBV (group B)	PEG-IFN + RBV (Group C)
	N=109	N= 28	N=27
DT	13 (12%)	2 (7%)	5 (19%)
NR	46 (48%)	10 (38%)	6 (27%)
R	29 (30%)	6 (24%)	3 (14%)
SVR	21(22%)	10 (38%)	13 (59%)

Conclusions: According to the clinical trials, our results confirm in clinical practice that: significantly more patients treated with PEG-Interferon alpha plus ribavirin had a SVR than those treated with Interferon alone (59%vs22%, p=0,002) or Interferon plus ribavirin (59% vs 38%, p<0,05). The tolerability was similar in the three groups. In univariate analyses, predictors of SVR were: low levels of GGT, ALP, TGL (p<0,05); HCV genotype non-1 (p<0,005); stage 1 at biopsy (p<0,001).

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SAFETY OF PEG-IFN α2b PLUS RIBAVIRIN IN NONCIRRHOTIC PATIENTS WITH CHRONIC HEPATITIS C.

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Aims: The tolerability profile of PEG-IFN α2b is similar to the one of IFN α2b in patients (pz.) with chronic hepatitis C. We have considered the safety profile of PEG-IFN α2b+Ribavirin compared with the one of Interferon α2b+Ribavirin in the treatment of chronic hepatitis C.
Methods: 43 patients have been treated with PEG-IFN α2b at the dose of 1 µg/kg weekly+Ribavirin 1-1,2 g/die (Group A) and compared with 40 patients previously treated with IFN α2b 3 MU/ 3 times weekly +Ribavirin at the same dosage (Group B). Contraindications for interferon therapy have been excluded.
Results: In Group A 7 patients (16.3%) have discontinued the therapy, 6 for adverse events and 1 for severe neutropenia; in group B only 2 (5%) patients have interrupted the therapy for side effects (depression). A dose modification has been required in 7 pz (16.3%) for neutropenia in group A compared with 2 (5%) pz of group B; 11 pz (25,6%) for anaemia in group A against 5 pz. (12.5%) of group B; 4 pz (9%) for thrombocytopenia opposite to 2 (5%) in Group B. The frequency of flu-like syndrome has been similar in the two groups (40%vs 35%).
In Group A, 6 cases of thyroid dysfunction have been shown (1 hyperthyroidism, which has required therapy discontinuation and 5 hypothyroidism, with a consequent substitutive hormonal therapy), a young man has presented a severe arrhythmias (FA which required electrical cardioversion), 2 patients have developed severe depression, which has caused interruption of therapy, 5 patients have instead developed mild–moderate depression, which has not determined a discontinuation of the therapy. In Group B we have found 2 cases of asymptomatic hypothyroidism and no hyperthyroidism; 2 patients have shown severe depression, with consequent dose discontinuation; none has had cardiac syndrome.
Conclusion: In our experience PEG-IFN has been associated with a higher frequency of rare and severe side effects or laboratory abnormalities compared with IFN α2b.