ABSTRACT

Background: Hepatitis C virus infection seems to induce insulin resistance and type 2 diabetes by direct viral involvement. Prevalence of glucose metabolism disorders is higher in C virus infected non cirrhotic pacients in comparation to pacients with other etiology liver diseases. Insulin resistance may influence the evolution of hepatic disease and response to antiviral treatment.

The **aim** of this study was to identify a possible relationship between hepatitis C infection and diabetes, to estimate the role of diabetes risk factors in patients infected with hepatitis C virus compared with hepatitis B virus, to identify other important risk factors of insulin resistance in this particular case. Moreover, we investigated the effects of metabolic disorders on therapeutic response and the effects of viral eradication on glucose metabolism.

Material and method: 270 pacients with chronic C hepatitis were compared to 163 pacients with chronic B hepatitis, regarding to glucose metabolism before and after antiviral therapy respectively regarding to risk factors of diabetes. We divided our pacients in two cohorts:

- Studied cohort: HCV infected patients
- Control cohort: HBV infected patients

After measurement of initial glucose metabolism parameters, we have created 4 groups of patients:

- Hepatitis B with normal glucose metabolism
- Hepatitis B with insulin resistance/diabetes
- Hepatitis C with normal glucose metabolism
- Hepatitis C with insulin resistance/diabetes

Results: Prevalence of insulin resistance was 19% in hepatitis C and 6,7% in hepatitis B pacients ($p<0,0001$).

Known diabetes risk factors (higher body mass index, abdominal obesity, family history of diabetes, arterial hypertension, gestational diabetes, fetal macrosomia) appeard in most cases of insulin resistant HBV infected pacients, but only in few cases of insuln resistant HCV infected pacients, with a statistically significant difference ($p<0,0001$). Lipid metabolism disorders (hipercholesteroaemia, hipertrigliceridaemia) were oftener in both insulin resistant groups but they appeared

more frequently in pacients with HBV and diabetes than in pacients with HCV and diabetes ($p<0,0001$).

45,4% of HBV infectes pacients had low viral load (less than 800 000UI/ml), 54,6% high viral load (more than 800 000UI/ml); 34,6% of HCV infectes pacients had low viral load, 65,4% high viral load. Look at our group's results: group A: 44,7% low viral load 55,7% high viral load; group B: 54,5% low, 45,5% high; group C: 40,4% low, 59,6% high; group D: 9,8% low, 90, 2% high. Difference was statistically significant between C and D groups ($p<0,0001$) but not between A and B ($p=0,528$).

After viral eradication plasma glucose, insulin levels and HOMA value decreased significantly ($p<0,0001$) only in HCV infected, insulin resistant pacients.

In this group of pacients, eradication was obtained less(66,7%) than in non insulin resistent C hepatitis (84,4%) or insulin resistent B hepatitis group (80,0%) ($p=0,012$).

Conclusions: Hepatitis C virus infection increases the risk of diabetes comparing to hepatitis B virus, irrespectively of classic diabetes risk factors but dependent by viraemia. Iron load, expressed by higher ferritin level, could be one, but not the most important pathogenic link between hepatitis C and diabetes. Insulin resistance decreases therapeutic response only in hepatitis C, but viral eradication improves glucose metabolism in these pacients.