A MODIFIED ANTI-LIVER SPECIFIC PROTEIN (LSP)-RIA FOR IMMUNEMONITORING OF AUTOIMMUNE CHRONIC ACTIVE HEPATITIS (AI-CAH): A LONGITUDINAL STUDY.

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Study objective: To establish a quantitative, reproducible RIA for anti-LSP, and determine its applicability in immunemonitoring[1] of AI-CAH. Patient groups: 32 consecutive patients with moderate to severe AI-CAH, 24 subsequently treated (group 1), 8 untreated (group 2), and 31 controls (group 3). Design: We modified the anti-LSP RIA[2]. Frozen sera and biopsies were coded and studied, in group 1 at 0,2,14 and 26 months of standardized treatment. Coded biopsies were reviewed and assigned a histological activity score (HA)[3] by two of us. Therapeutic intervention: (group 1) oral prednisolone 15 mg and azathioprine 75 mg daily for two months, followed by 2 years of treatment with 10 resp. 50 mg daily.
Main Results: 1.Method:binding percentages as determined in London(KCH)[4] and in our assay did not differ, the interassay variation was intralabelling <10%, interlabelling 15%, and the intra assay variation <10%. Using 3 dilutions calculated anti-LSP titers did not differ from measured titres. The normal binding range was 5-24%. 2.Clinical relevance: Before start of therapy in 23/24 patients of group 1 anti-LSP was detected. In histological remission (HA≤2) 14/23 became negative. Correlation of anti-LSP with HA: r=.62; In group 2 4/8 patients, and in the control group 0/31 were anti-LSP positive .
Conclusions: 1. We established a simplified, reproducible, quantitative RIA for anti-LSP. 2. Anti-LSP reflects immunological activity of AI-CAH, confirming ref. 1.

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IS MITOCHONDRIAL ASPARTATE AMINOTRANSFERASE (m-AST) USEFUL IN THE DIAGNOSIS OF ALCOHOLIC LIVER DISEASE?

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The clinician is often confronted with the problem whether liver function test abnormalities are due to excessive consumption of ethanol. Recently, m-AST and the ratio of mitochondrial to total AST (m:t-AST) have been proposed as sensitive and specific markers for chronic alcoholism. In the present study we tested the hypothesis that m-AST helps in the identification of alcoholics among a group of outpatients with biochemical evidence for liver disease. M-AST activity in serum was measured using an immunological assay supplied as a kit by Poli Industria Chimica (Italy). In 10 healthy controls m-AST was 0.7±0.5 (mean±SD) U/l corresponding to 4.0±2.3% of t-AST. In 10 patients with documented alcoholic liver disease and elevated GGT, m-AST ranged from 1.0 to 16.2 U/l, and m-AST:t-AST from 4.9-12.4%. The median values in our outpatients (3.6 U/l and 8%, respectively) were lower than in previously published studies of alcoholic patients who had required hospitalization. Most likely, this reflects less severe liver disease in our population. Indeed, only 3 alcoholics had a moderately decreased aminopyrine breath test, suggesting well preserved hepatic function in most subjects. Although significantly ($p<0.001$) different from controls, these values for m-AST were not higher than in 11 patients with non alcoholic liver disease (m-AST 1.7-15.4 U/l; m:t-AST 4.1-12.9%). In patients with fatty liver who pose a particularly difficult diagnostic problem, m-AST and m:t-AST were elevated to the same extent in patients with alcoholic and non alcoholic etiology. We conclude that m-AST and the m:t-AST do not discriminate between an alcoholic and non-alcoholic etiology in outpatients with liver function test abnormalities.