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Transaminases elevation during the third trimester of the pregnancy (P)

A. Abergel, L. Rakotoarivonina, B. Aublet-Cuvelier, J.F. Viallard, R. Cesaire, Cécile Ralliere, C. Henquell, C. Divet, Y. Glanddier, E. Subtil, et al.

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**THE
AMERICAN ASSOCIATION
FOR THE
STUDY OF LIVER DISEASES**

**POSTGRADUATE COURSE
& 45TH ANNUAL MEETING
NOVEMBER 11-15, 1994**

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112 IDIOPATHIC STEATOSIS IN CHILDHOOD: ONSET, PRESENTATION AND ETIOLOGY. AD Baldrige*, F Grame-Cooke†, B Banner††, A Perez**, L Higgins*, JE Lavigne*: *Center for Childhood Liver Disease, Combined Program in Pediatric Gastroenterology; **Dept of Pathology, Children's Hosp (CH), Harvard Medical School; †Dept of Pathology, Massachusetts General Hosp (MGH), Boston, MA; ††Dept of Pathology, University of Massachusetts Medical Center (UMMC), Worcester, MA.

The onset, presentation and etiology of idiopathic hepatic steatosis in children is uncharacterized, and its relationship to the entity of nonalcoholic steatosis (NASH) has not been established. NASH (fatty infiltration of hepatocytes with inflammation and fibrosis) has been described in adults associated with diabetes mellitus, rapid weight loss, and obesity. An indolent, progressive clinical course may result in cirrhosis. Obese children in Japan with hepatitis have been reported to have histological changes similar to NASH, but idiopathic steatosis of the liver in children has not been reviewed.

Children (0-18yrs) with evidence of steatosis were identified by a retrospective review of all liver biopsies performed at CH, MGH, and UMMC from 1991-94. Fourteen of 82 patients had fatty infiltration of the liver without evidence of known causes of steatosis. All 14 were obese and weighed greater than 120% ideal body weight (IBW) and tall with height 106% (SD±19%) expected. Nine presented with abdominal pain, 2 had unexplained hepatomegaly, and 1 was detected incidentally. These 12 were biopsied for persistent transaminase elevation. Two patients were detected incidentally by biopsy during staging laparotomy and had no elevation in liver enzymes. The clinical and biochemical data are summarized below stratified by histologic group (I = steatosis alone, II = with inflammation, III = with fibrosis):

Group	N	M:F	Age (yr)	IBW (%)	AST	ALT
All	14	10:4	13.5(±2.6)	155 (±28)	77 (±38)	129(±73)
I	2	2:0	15.4(±2.6)	165 (±15)	31 (± 4)	28 (±3)
II	5	3:2	13.9(±2.1)	142 (±18)	81 (±33)	126(±66)
III	7	5:2	12.6(±2.6)	163 (±32)	87 (±38)	160(±63)

We conclude that idiopathic steatosis presents in prepubescent and pubescent symptomatic and asymptomatic children. All patients, usually males, without another identifiable cause of steatosis were obese. This group represented a common reason for liver biopsy in this age category. Obese children with evidence of liver disease may have significant histologic derangements and can present with bridging fibrosis.

913 DOUBLE BLIND PLACEBO CONTROLLED TRIAL OF RECOMBINANT HUMAN GROWTH HORMONE (rhGH) THERAPY IN CIRRHOSIS
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Malnutrition is of high prevalence in cirrhosis and has an adverse influence on clinical outcome. Low substrate availability and resistance to growth factors such as insulin and GH are prominent causes of this catabolic state. The anabolic effects of GH are mediated largely by insulin-like growth factor-I (IGF-I), the circulating form of which is of liver origin and low levels are seen in cirrhosis. RhGH therapy has proven efficacy in other catabolic states. To investigate the potential therapeutic role of rhGH therapy in cirrhosis we studied 20 consecutive cirrhotic patients in a double blind study, 10 in an active group (M:F 6:4, Childs groups A n=5, B n=3, C n=2) and 10 in a placebo group (M:F 3:7, Childs groups A n=2, B n=5, C n=3). Patients were treated with daily sc injections of either rhGH at a dose of 0.25 IU / kg or placebo for 7 days, after a 3 day baseline period. Levels of GH, IGF-I, insulin and IGFBP 3 (the dominant IGF binding protein of human serum) were measured by RIA and urinary nitrogen by the Kjeldahl technique. Results Pretreatment GH levels were high compared to a normal control group (mean 14.9 ± SEM 2.7 vs 3.64±2 p<0.05) with low levels of IGF-I (72.3±4.4 vs 151±7.6 p<0.0001) and IGFBP-3 (1.5±0.2 vs 5.3±0.2 p<0.0001). Pretreatment levels of GH, IGF-I, IGFBP-3 and Nitrogen balance were statistically similar in active and placebo groups. During rhGH therapy in the active group IGF-I levels increased in all patients from a mean 73.5±10 to 170.5±48 as did IGFBP-3 levels 1.9±0.4 to 2.9±0.6. No change in GH or insulin levels was seen. Daily nitrogen balance improved significantly in the rhGH treated group from +3.1±1.1 g/l (days 1-3) to +4.7±1.2 g/l (days 4-7) to +5.5±1.7 g/l (days 8-10). No significant change was seen in any of the hormone levels or nitrogen balance in the placebo group. No significant side effects of rhGH therapy were seen.

Conclusions 1) RhGH therapy was able to overcome the GH resistance of cirrhosis, increasing production of IGF-I and IGFBP-3 and effecting a significant improvement in nitrogen balance. 2) RhGH may have an important role in the treatment of malnourished cirrhotic patients.

914 TRANSAMINASES ELEVATION DURING THE THIRD TRIMESTER OF THE PREGNANCY (P).

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The main causes of transaminases elevation beside pregnancy are : alcohol, drugs, hepatitis virus, steatosis and seldom auto-immune or metabolic diseases (hemochromatosis, Wilson disease, alpha-1 antitrypsin deficiency). The relative contribution of these factors during pregnancy are unknown. The main goals of this study were : 1) to determine the incidence of alanine aminotransferase (ALAT) elevation during pregnancy, 2) to state the role played by pregnancy related and non related liver diseases. From December 91 to October 92, ALAT (Mérieux, -30°C) was determined in 1352 patients who delivered in public maternity hospitals. All the women with ALAT > N (N=41) were tested for Ag Hbs (Pasteur), IgM anti-HAV (Abbott), IgM anti-Hbc (Abbott), anti-HCV (Abbott), IgM anti-CMV (Wellcome) and antinuclear, antismooth muscle, antimitochondrial, anti-LKM antibodies by indirect immunofluorescence. ALAT was superior to N in 41 patients (3,03%). Presumed etiologies were as follow (Table) :

ETIOLOGY	ALAT > N	N<ALATs1,5 N	ALAT > 1,5 N
Cholestasis (ICP)	10	4	6
Preeclampsia	6	1	5
Urinary infection	5	2	3
Hepatitis C	3	2	1 (PCR-)
Drugs	2	0	2
Unknown	15	12	3

This study shows that 1) the pregnancy related liver diseases are the main causes of the transaminases elevation, 2) the prevalence of the ICP is 0,74% (10/1352) and more elevated than generally accepted in France (0,2%), 3) the aetiological diagnosis of an ALAT elevation during P is often clinical.

915 FK 506: A PROMISING THERAPY FOR PATIENTS WITH AUTOIMMUNE LIVER DISEASES.

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The demonstration of the superior therapeutic index of FK 506 "Prograf" for hepatic allotransplantation triggered an open label trial for its use among patients with fully documented autoimmune hepatic diseases; chronic active hepatitis (CAH-A), primary sclerosing cholangitis (PSC) and primary biliary cirrhosis (PBC). From June 1990 until February 1994 a total of 59 patients who failed long term conventional therapy for each disease were enrolled in the study; 25 had CAH-A, 14 had PSC, and 20 had PBC. Cirrhosis was histologically documented from the outset in 22 (37%) patients. FK 506 was given with a starting oral dose of 0.03 to 0.05 mg/Kg/day every 12 hours and dose adjustments were dictated by disease gravity, FK 506 plasma trough levels and adverse drug reactions. Liver transplantation was required for 12 (20%) patients (5 CAH-A, 3 PSC, 4 PBC) 1 to 30 months after initiation of FK 506 treatment and all of them had concomitant cirrhosis and were already listed for transplant (UNOS status 2) before treatment. In another 9 patients (3 CAH-A, 4 PSC, 2 PBC), FK 506 therapy was discontinued after 6 to 29 months because of either recurrent cholangitis (n=4), recent detection of concomitant C hepatitis (n=2), peripheral vascular disease (n=1), thrombocytopenia (n=1) or development of cryptococcal pneumonia (n=1) that was successfully treated with antifungal therapy. With a mean follow-up of 23 ± 9 months (range: 3 to 36), the biochemical responses of the remaining 38 (64%) patients who continue to receive FK 506 are (mean ± SD):

Disease	F.U. (mos.)	Bilirubin(mg/dL)		AST (IU/L)		GGTP(IU/L)	
		Pre	Current	Pre	Current	Pre	Current
CAH-A(n=17)	21± 8	2.9±4	0.9 ± 0.3*	328±540	58±29*	315±391	145±174
PSC (n=7)	27± 7	1.0±0.4	0.9 ± 0.6	159±106	66±69*	807±511	385±422
PBC (n=14)	23±10	1.8±2.3	1.1 ± 0.7	119±140	63±32	678±606	532±505
Total(N=38)	23± 9	2.1±3	1.0 ± 0.5*	218±374	61±39*	540±523	331±403*

* P < 0.05
 There were no significant changes in liver volume. None of these cases showed clinical, endoscopic or radiologic evidences of progressive portal hypertension and the histopathologic examination of the 12 month follow up liver biopsies (n=11) showed significant improvement in the hepatic activity index. There were no significant side effects of FK 506 or any opportunistic infections. In conclusion, FK 506 is an effective and safe therapeutic agent for patients with early stages of CAH-A, PSC and PBC.