

GALLBLADDER VOLUME AND CONTRACTION IN GALLSTONE PATIENTS AND CONTROLS.

K.J. van Erpecum, G.P. van Berge Henegouwen, M.F.J. Stolk, W.P.M. Hopman*.
Dept. of Internal Medicine, Malberg-G.Z. Arnhem and *Dept. of Gastroenterology,
University Hospital, Nijmegen, The Netherlands.

It has been suggested that impaired gallbladder contraction may be important for cholesterol gallstone formation by providing time for nucleation of cholesterol crystals in supersaturated bile.

We compared gallbladder volume and contraction in 20 gallstone patients and 20 normal subjects. There were no significant differences in sex ratio or age between both groups and no more than 20% of fasting gallbladder volume was occupied by stones. Fasting gallbladder volume was determined using ultrasonography (sum-of-cylinders method). Subsequently, after oral administration of a fatty meal (Sorbitract®) volume was determined every 10 minutes during 90 minutes.

Table		Controls	Patients	P	Values denoted as mean (SEM) ;
Vo	(ml)	18.9 (1.6)	29.9 (4.3)	< 0.05	Vo = fasting gallbladder volume ;
Δ V max	(ml)	10.0 (1.2)	12.9 (2.6)	n.s.	V min = minimal gallbladder volume
Δ V max	(%)	52.0 (4.0)	42.9 (4.9)	n.s.	after maximal contraction ;
V min	(ml)	9.0 (1.1)	17.0 (2.8)	< 0.05	Δ V max = maximal decrement of
					gallbladder volume in ml resp. %

Significance according to Mann-Whitney U-test.

Conclusions : 1. Gallstone patients have significantly larger mean fasting and residual volumes than controls. This may be an epiphenomenon due to the presence of gallstones. Alternatively large residual gallbladder volumes may increase the risk of gallstone formation. 2. Gallbladder emptying is not decreased in gallstone patients.

SAA VERSUS CRP SERUM LEVELS QUANTIFIED BY SANDWICH ELISA'S IN CHRONIC ACTIVE HEPATITIS (CAH), AND THEIR RELATION TO NUMERICAL SCORED HISTOLOGICAL ACTIVITY

B. van Hoek*, B.P.C. Hazenberg°, P.C. Limburg°, J. Bijzet°, J.R. Huizenga*, A.J.K. Grong°, M.H. van Rijswijk, C.H. Gips. Divs. of Hepatology and Rheumatology, Dept of Med, and Dpt of Pathology, University Hospital, Oostersingel 59, 9713 EZ Groningen, The Netherlands.

Study objective: The acute-phase proteins CRP and SAA are (almost) entirely synthesised in the liver and they react on inflammation. We studied CRP and SAA in chronic inflammation of the producing organ, i.e. chronic active hepatitis.

Design: SAA and CRP were quantified by newly developed sandwich ELISA's§; SAA-ELISA was modified by using a monoclonal anti-human-SAA as a second antibody. **Patients:** 85 patients with moderate to severe CAH of various etiology, 25 subsequently treated (group 1), 60 untreated (group 2). Biopsies and frozen sera were studied, in group 1 at 0,2,14 and 26 months of standardized treatment, with additional sera during infections, in group 2 during active CAH. **Histological activity scores (HA)** were assigned by two of us reviewing the biopsies. **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.

Results: 1. There was no apparent difference in CRP and SAA values between the various subgroups of CAH studied. 2. In active CAH serum-SAA is normal, and CRP is elevated, resulting in a low SAA/CRP-ratio. 3. During infections in CAH-patients this ratio remains below normal. 4. After institution of immunosuppression CRP normalizes, SAA remains unchanged. 5. SAA has no, and CRP a weak correlation with histological activity of CAH.

Conclusions: 1. In CAH there is a clear CRP preponderance with normal SAA.

2. CRP only weakly correlates with histological activity of CAH.

Reference §: Janssen S, Limburg PC, Bijzet J, et al. SAA versus CRP in chronic inflammatory diseases. In: XXXIVth Colloquium Protides of the Biological Fluids. Peeters H, ed. Pergamon Press Ltd, Oxford 1986; 34: 347.