O-25 BACTERIAL INFECTION ENHANCES THROMBIN GENERATION IN PATIENTS WITH CIRRHOSIS

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Introduction and Aims: Current concept of coagulopathy in cirrhosis indicates that there is a rebalancing of hemostasis with plasma hypercoagulability. Bacterial infection can promotes releases of endothelial heparinoids. However, the effect of this condition on the thrombin generation is unknown. Our aim was to assess the effect of bacterial infection on thrombin generation in cirrhosis.

Methods: 36 patients with cirrhosis and bacterial infection (infected group) were evaluated within 24 hours after start antibiotic and at least 5 days after infection resolution. 28 patients with decompensated cirrhosis and not infected (not infected group) were also enrolled and reevaluated, without any intervention between evaluation times. Primary endpoint was the effect of bacterial infection on thrombin generation (TG) parameter ETP with TM (ETP TM). TM is a protein C activator added to mimic *in vivo* conditions. ROTEM assays, INTEM and HEPTEM (heparinase modified), was performed to evaluate the endogenous heparinoids effect. Protein C (PC) and antithrombin (AT) assays were performed. All results were compared within each group between evaluation times.

Results: ETP TM values in infected cirrhotics were significantly higher than after resolution of infection (from 1145.4 ± 360.7 nmol/L*min to 958.1 ± 254.8 nmol/L*min, p=0.005) - figure 1. A heparinoid effect was found only in infected cirrhotics, with CT_{INTEM} duration significantly longer than CT_{HEPTEM} (p=0.004). This effect disappeared after resolution of infection (p=0.75). PC and AT deficiencies were significantly more severe in infected patients (p<0,01). RNI/TP, aPTT was worsen at active infection (p<0.05). None of these parameters exhibited a significant difference between inclusion and revaluation times in not infected group.

Conclusion: patients with cirrhosis exhibits significant higher amount of TG during bacterial infection and it is associated with reduction of PC and AT levels. Despite the endogenous heparinoid effect during infection in cirrhosis, plasma hypercoagulability is preserved and cannot be assessed by conventional coagulation tests.

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O-26 ASPARTATE AMINOTRANSFERASE, AGE AND D-DIMER IN COVID-19 PATIENTS: A USEFUL PROGNOSTIC MODEL

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Introduction: Some patients with SARSCov-2 infection develop severe disease (SARS); however, the factors associated with severity are not yet fully understood. Some reports indicate that liver injury may be a poor prognostic factor.

Aim: To identify the biochemical factors related to the development of SARS with mechanical ventilation (MV) requirement in patients with SARSCov-2 and COVID-19.

Methods Type of study: Observational. Cohort study.

Procedure: Data from COVID-19 patients were collected at admission time to a tertiary care center. Differential factors were identified between seriously ill SARS+MV patients versus stable patients without MV. Transformation to the natural logarithm of significant variables was performed and multiple linear regression was applied, then a predictive model of severity called AAD (Age-AST-D dimer) was constructed.

Results: 166 patients were included, 114(68.7%) men, mean age 50.6±13.3 years-old, 27(16.3%) developed SARS+MV. In the comparative analysis between those with SARS+MV versus stable patients without MV we found significant raises of ALT (225.4± 341.2 vs. 41.3±41.1; *P*=0.003), AST 325.3±382.4 vs. 52.8±47.1; P=0.001), LDH (764.6±401.9 vs. 461.0±185.6; P=0.001), D dimer (7765±9109 vs. 1871±4146; P=0.003), age (58.6±12.7 vs. 49.1± 12.8; P=0-001). The results of the regression are shown in the Table, where model 3 was the one that best explained the development of SARS+MV; with these variables was constructed the model called AAD, where: [AAD= 3.896 + ln(age)x-0.218 + ln(AST) x-0.185 + ln(DD)x0.070], where a value ≤ 2.75 had sensitivity=0.797 and 1-specificity= 0.391, AUROC=0.74 (95%CI: 0.62-0.86; P<0.0001), to predict the risk of developing SARS+MV (OR=5.8, 95%CI: 2.2-15.4; P=0.001).

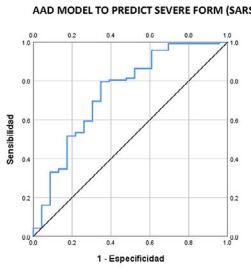
Conclusions: Elevation of AST (probable marker of liver damage) is an important predictor of progression to SARS, together with elevation of D-dimer and age early (at admission) and efficiently predict which patients will potentially require MV.

Model		Non-standarized Coeficients		StandarizedCoeficients	P	95% Confidence Interval for B		Colinearity statistics	
		B	Error Desv.	Beta		Inferior limit	Superior limit	Tolerance	VIF
1	C	2.721	.131		.000	2.462	2.980		
	AST	-.229	.033	-.512	.000	-.293	-.164	1.000	1.000
2	C	3.161	.198		.000	2.770	3.551		
	AST	-.194	.034	-.435	.000	-.261	-.127	.878	1.139
	DD	-.081	.028	-.221	.004	-.135	-.026	.878	1.139
3	C	3.896	.414		.000	3.077	4.714		
	AST	-.185	.034	-.413	.000	-.252	-.118	.860	1.163
	DD	-.070	.028	-.190	.014	-.125	-.014	.844	1.185
	Age	-.218	.108	-.148	.046	-.433	-.004	.915	1.093

AST, aspartate aminotransferase; C, constant; DD, D dimer; VIF, variance inflation factors.

Resume of the model:

1. R=0.512, r^2=0.262, r^2 adjusted=0.256, standard error=0.331.
2. R=0.552, r^2=0.305, r^2 adjusted=0.294, standard error=0.322.
3. R=0.570, r^2=0.325, r^2 adjusted=0.310, standard error=0.318. Durbin-Watson=1.53.



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O-27 IMPACT OF HBV GENOTYPE F IN THE DIAGNOSIS AND EVOLUTION OF PATIENTS WITH HBEAG-NEGATIVE CHRONIC HBV INFECTION

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Background: The quantitative hepatitis B surface antigen (qHBsAg) threshold of 1,000 IU/ml has been proposed to distinguish HBeAg-negative chronic infections from HBeAg-negative chronic hepatitis, to assess risk of liver disease progression, and to predict HBsAg clearance. There is evidence that qHBsAg vary significantly among genotypes, however, there is scarce data on genotype F, the most prevalent in Latin America.

Aims: To analyze the impact of HBV genotype F on qHBsAg inpatients with HBeAg-negative chronic infection and to describe clinical and virological outcomes.

Methods: HBV-DNA and qHBsAg serum levels of 141 patients with HBeAg-negative chronic infection were correlated with HBV genotype, who were followed for 10.6±7.4 years.

Results: The overall genotype distribution was as follows: F 46.8%, D 26.1%, A 25.2%, and B 0.7% and C 0.7%. While no impact of the HBV genotype on HBV DNA levels was observed, qHBsAg differed significantly among genotypes (p<0.001). The highest HBsAg levels were observed in genotype F (4.0±1.1 Log_{10}IU/ml) followed by genotype A (3.9±0.6 Log_{10}IU/ml) and genotype D (2.4±0.9 Log_{10}IU/ml). In genotype A and F, qHBsAg <3.0 Log_{10}IU/ml were only observed in 10.7% and 11.5% respectively.

Regardless of the HBV genotype, spontaneous clearance of HBsAg was observed in 17 cases. Of these, 12 patients presented qHBsAg <100 IU/ml one year before clearance. Despite, 101 (71.6%) patients showed qHBsAg >3.0 Log_{10}IU/ml, no cases of advanced liver disease or hepatocellular carcinoma were observed at the end of follow-up.

Conclusions: This study provides new insights into the impact of HBV genotypes on serum HBsAg levels, emphasizing the need to implement genotype-specific cut-off to achieve diagnostic certainty in the identification of HBeAg-negative chronic infection and the risk of liver disease progression, particularly on infections with genotypes A and F. Moreover, HBsAg serum levels can became a reliable biomarker to predict HBsAg clearance.

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O-28 DRUG-INDUCED LIVER INJURY IN LATINAMERICA: First ten years' experience of the ongoing LATINDILI Network

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