Evaluating serum biomarkers LIVERFASt ™ surrogates of liver fibrosis and steatosis could identify risks in a clinical population experiencing SARS-COV2 infection (COVID19).

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Background and aim

Coronavirus disease-2019 (COVID-19) is a life-threatening infection caused by the severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) virus. Age, diabetes and metabolic factors has rapidly emerged as a major comorbidity for COVID-19 severity. However, the phenotypic characteristics of patients (pts) in COVID-19 are unknown. For clinicians, it's imperative to predict the outcome of a given patient following a positive test for SARS-CoV2—it is known that prior health history and demographics are informative towards describing the wide range of prognostic outcomes for COVID19 pts.

Aim.

We approach this problem from a new angle: by directly evaluating blood serum markers LIVERFASt™ (LF) for fibrosis and steatosis prospectively collected from 10,623 patients, we examined whether there exist any notable differences between those who would eventually contract COVID19 (~30%), and those who have not (or have not yet) contracted the disease. LIVERFASt™ is a serum-based proprietary algorithm assessing liver fibrosis (LF-Fib) and steatosis (LF-Ste).

Methods

We amassed a database prospectively collected (10,623) from primary, secondary and tertiary settings before and during the COVID19 pandemy. Pts were selected with available LF and LF select two surrogate markers for fibrosis and steatosis risk evaluation. LIVERFASt is computed with sex, age at test, alpha2 macroglobulin (a2m), haptoglobin, apolipoprotein a1, bilirubin ,GGT, ALT, BMI, fasting glucose, cholesterol and triglycerids. Patients that tested positive for Covid-19 were matched based on the main characteristics including age and gender with those not Covid experimented.

Results

We focused on 276 pts Covid (-) or (+) having complete LF -Fib and LF-Ste, mean age 59.6yrs, 49% males, 38,4% having history of cardiovascular disease, 68.4% having 2 or more metabolic syndrome factors.

A strong correlation was observed between LF-Ste and the number of metabolic factors (Spearman correlation coefficient 0.39 , p<0.001). (Figure 1) with a linear increase of LF-Ste score with the increase in the number of metabolic factors.

9/276 pts Covid (+) positive were matched with 66/276 Covid (-) negative pts. 77% pts with Covid (+) had more than one cardiovascular complications and 67% had at least one metabolic factor.

Median (95%CI) estimated liver fibrosis in Covid (+) pts was significantly higher than in matched without Covid (92) as per surrogate marker of fibrosis A2M [3.30g/l (2.56-3.78) vs 2.40 g/l(2.07-2.74), p=0.04] and LF-Fib [0.6 (.45-.77) vs 0.39(.32-.59) p=0.05] . Covid (+) pts had higher triglycerids levels [2.34 (1.33-2.78) vs 1.02 (.97-1.27), p<0.001].

10,028 Covid (-) pts with LF Select to detect steatosis risk were identified and 319 Covid (+) that will make the object of further analyses for the outcomes.

Conclusion

LIVERFASt Steatosis and Fibrosis scores are noninvasive tests that could be used to screen populations at risk of metabolic syndrome and liver disease and Covid.

Figure 1. LIVERFASt Steatosis according to Metabolic factors

Box Plot of LF_Ste