Blood Glucose and Epicardial Adipose Tissue at the Hospital Admission as Possible Predictors for COVID-19 Severity

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Abstract

**Purpose:** to study the possible association of CT-derived quantitative Epicardial Adipose Tissue (EAT) and glycemia at the admission, with severe outcomes in patients with COVID-19.

**Methods:** 229 patients consecutively hospitalized for COVID-19 from March 1st to June 30th 2020 were studied. Non-contrast chest CT scans, to confirm diagnosis of pneumonia, were performed. EAT volume (cm$^3$) and attenuation (Hounsfield units) were measured using a CT post-processing software. The primary outcome was acute respiratory distress syndrome (ARDS) or in-hospital death.

**Results:** The primary outcome occurred in 56.8% patients. Fasting blood glucose was significantly higher in the group ARDS/death than in the group with better prognosis [114 (98-144) vs 101 (91-118) mg/dl, p=0.001]. EAT volume was higher in patients with vs without the primary outcome [103 (69.25;129.75) vs 78.95 (50.7;100.25) cm$^3$, p <0.001] and it was positively correlated with glycemia, PCR, fibrinogen, P/F ratio.

In the multivariable logistic regression analysis, age and EAT volume were independently associated with ARDS/death. Glycemia and EAT attenuation were risk factor for ARDS/death with a trend of statistical significance.

**Conclusions:** Our findings suggest that both blood glucose and EAT, measurable and modifiable targets, could allow the early identification of subjects at greater risk of developing severe complications.

1. **Introduction**

COVID-19 is a global pandemic and public health issue of ever-increasing proportions (1). Obesity and in particular excess visceral fat are implicated in development of heart and lung complications of COVID-19 due to a chronic inflammatory condition. Adipose tissue has been suggested to play a role as a reservoir for the virus and amplifier of the inflammatory response (2-5).

Epicardial adipose tissue (EAT), the visceral fat of the heart, is characterized by a dense macrophage infiltrates and secretion of proinflammatory cytokines, such as interleukin-6 (IL-6), overexpressed in COVID-19 patients with heart and lung diseases (7). Because its proinflammatory properties, EAT can contribute to the development of COVID-19 cardiac and pulmonary complications (7-10, 11). EAT volume and attenuation, a marker of inflammation, can be measured with chest computed tomography (CT) methods, currently considered a primary tool for detecting COVID-19 pneumonia (12,13).

Furthermore, hyperglycemia has been described in more than half of the patients with COVID-19 infection (14). Although stress-induced hyperglycemia is a physiological response, it may lead to further complications in hospitalized patient with pneumonia, independently of the previous diagnosis of diabetes (15).

To prevent the severity of the disease, our efforts are increasingly focused on identifying the best clinical parameters at the time of admission to predict the risk of complications and improve the stratification of patients to undergo more effective therapies from the earliest stages of the disease (16,17).

In this study we sought to examine the association of CT-derived quantitative EAT volume and attenuation and metabolic markers at the admission, in particular blood glucose, with worst outcomes (acute respiratory distress syndrome or death) in hospitalized patients with COVID-19.

2. **Materials And Methods**

2.1 **Study design and population**

This is a retrospective study conducted by the Latina Covid-19 Study Group on 229 patients consecutively hospitalized for Covid-19 from March 1st to June 30th, 2020 in Santa Maria Goretti Hospital, Polo Pontino of Sapienza University in Latina.

All patients who went to the emergency room with symptoms suggestive for Covid-19 (body temperature ≥ 37.5 °C, cough, dyspnoea) underwent triage in an out-of-hospital facility where vital signs were detected by a dedicated staff.

The diagnostic protocol included the execution of nasopharyngeal swab RT PCR and chest CT to determine and quantify the presence of interstitial pneumonia.

All patients with confirmed positivity to Sars-CoV2 and with chest CT scan suggestive of interstitial pneumonia, were hospitalized.
Routine laboratory tests were obtained at hospital admission. All patients underwent the same therapy used until June 30th, 2020 (according to internal hospital protocol) and based on methylprednisolone, azithromycin, lopinavir/ritonavir, hydroxychloroquine and enoxaparin. All subjects with P/F ratio < 200 underwent therapy with tocilizumab. Every patient received oxygen support with different FiO2 and different devices depending on respiratory distress degree and subjected to repeated blood gas analysis.

2.2 Anthropometric, clinical and laboratory assessments

The following anthropometric data were extrapolated from the medical records of the subjects included in this analysis and refer to the time of admission: age, gender, height, weight, BMI. P/F ratio was determined in all patients by blood gas analysis. Among the blood chemistry tests it was possible to collect the following parameters: complete blood count with differential, glycemia, creatinine, total cholesterol, high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), triglycerides, uricaemia, albumin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), Gamma Glutamyl transferase (GGT), Lactate dehydrogenase (LDH), erythrocyte sedimentation rate (ESR), c-reactive protein, ferritin, fibrinogen, D-dimer. Data from the medical records of 229 hospitalized patients were collected and a database was set up for subsequent analysis.

The study was approved by the local institutional review board (IRB), and written informed consent was obtained from all study participants.

2.3 EAT quantification

Non-contrast chest CT scans were performed with TC Force Siemens Dual Energy. Isovolumetric, thin slice (1.25 mm) chest CT scan was used for volumetric quantification of EAT by means of advanced CT post-processing software (Intuition; Terarecon). The software made it possible to perform the semi-automatic volumetric segmentation of the EAT. To estimate the EAT volume, the pericardium was manually contoured, by the same operator, and considered as the outer segmentation limit.

EAT density (Hounsfield Unit) was determined by the positioning of a 6 mm ROI (region of interest) at the pulmonary arteries. The Hounsfield Units (HU) scale ranges from -1024 HU to 3071 HU. It is defined by the following: -1024 HU is black and represents air (in the lungs). 0 HU represents water. Fat is around -100 HU.

2.4 Outcomes and definitions

The composite primary outcome was defined as any degree of acute respiratory distress syndrome (ARDS) or death. ARDS is classified into mild (200 mm < PaO2/FiO2 ≤ 300), moderate (100 g < PaO2/FiO2 ≤ 200), and severe (PaO2/FiO2 ≤ 100), in accordance with the 2012 Berlin criteria (18). P/F was recorded at baseline (P/F baseline) and at the worst clinical condition during hospitalization (P/F nadir).

Diagnosis of type 2 diabetes was defined by a self-reported history of diabetes.

3. Statistical Analysis

Continuous variables were expressed as mean and standard deviation or median and interquartile range (IQR) according to their distribution. Categorical variables were recorded as frequencies and percentages. Comparisons between patients with and without the primary outcome were performed using, Mann–Whitney U test, T test or the chi-square test, as appropriate. Kruskal-Wallis test was used to assess differences among groups with different degrees of ARDS and post hoc, p-value were adjusted according to Benjamini and Hochberg to account for multiple comparisons.

Spearman's correlation coefficient was used to assess the relationship between CT parameters, serum biomarkers, and clinical variables. Multivariable logistic regression analysis was performed to investigate the relationship between the risk of having ARDS or death and Glycemia, EAT HU, EAT volume, age and gender. Model selection was performed by stepwise procedure based on the Akaike Information Criterion (AIC).

All tests were two-tailed, and a p-value < 0.05 was considered as statistically significant. Analyses were performed using R version 4.0.1 (The R Project for Statistical Computing).

4. Results

4.1 Patients characteristics
A total of 229 patients (age 63 (16); 56.8% male) with laboratory-confirmed COVID-19 who underwent chest CT during their admission were included. The primary outcome (ARDS or in-hospital death) occurred in 130 (56.8%) patients. The remaining patients (n = 99; 43.2%) had been discharged alive or otherwise did not develop ARDS during hospitalization at the time of data collection. Mild, moderate and severe ARDS occurred respectively in 34 (27%), 68 (54%) and 24 (19%) patients. The clinical and laboratory features of patients with and without the primary outcome are reported in Table 1. Patients with ARDS or died were older than the other group (age respectively 57 [47-68.5] and 66 [55-78.75]; p < 0.001). The ratio of men to women in the two groups did not differ significantly: the prevalence of male patients in the group ARDS/death was 40% vs 47.5% in the comparison group (p 0.319).

As regards anthropometric parameters in the two groups, with the available data (115 observations), body weight in the worst group appeared to be significantly higher, while the difference in BMI was close to statistical significance. The prevalence of diabetes mellitus was 18.2% in patients with better outcome and 36.2% in patients with worse prognosis, with a statistically significative difference (p=0.005). The prevalence of hypertension too was higher in the ARDS/death group (p 0.004). Fasting blood glucose values were significantly higher in the group ARDS/death than in the group with better prognosis, showing values of 114 (98-144) mg/dl versus 101 (91-118) mg/dl (p=0.001) respectively.

As regards the other nutritional and metabolic parameters, liver function indices (Alanine Aminotransferase - AST, Aspartate Aminotransferase- ALT and Gamma Glutamyl Transferase - GGT) reached higher values in the worst outcome group, while higher HDL levels appeared to be associated with a better prognosis. Differences in serum inflammatory markers were found between the two groups. Patients with worse outcome showed a reduction of lymphocyte counts and an increase in the percentage of neutrophils. C-reactive protein, ESR, D-Dimer and fibrinogen were significantly higher in the worst outcome group, as well as LDH concentration.

### 4.2 Chest CT measurements

The chest CT processing showed a median EAT volume of the whole cohort of 91.7 cm$^3$ (57,13-122), while median EAT attenuation overall was -88,78 (-102,83; -71,68) HU. EAT volume was higher in patients with versus without the primary outcome (103 cm$^3$ [69,25;129,75] vs 78,95 cm$^3$ [50,7;100,25], p<0,001). The comparison for EAT HU is not significant (Fig1 A-B).

Depending on the degree of ARDS, median (IQR) EAT volume was 77.10 cm$^3$ (51.40-101.00) in the group NoARDS and 93.40 cm$^3$ (60.98-119.75) and 99.50 cm$^3$ (68.30-133.50), 121.50 cm$^3$ (89.75-132.75) in patients with mild, moderate and severe ARDS respectively, with a statistically significant difference among groups (p=0.0006094, Kruskall–Wallis test). The pairwise comparison showed that EAT volume was significantly different between of No ARDS and moderate ARDS groups (p=0.0147) and between No ARDS and severe ARDS groups (p=0.0015), (Fig1 C-D).

Median (IQR) EAT density was -89.62 HU (-100.78—-70.47) in patients without ARDS, and 87.47 HU (-107.08—-74.89), -90.00 HU (-102.73—-74.51) and -80-53 HU (-101.73—-73.88) in patients with mild, moderate and severe ARDS respectively (p=0.975, Kruskall–Wallis test), (Fig1 C-D).

### 4.3. Correlation of EAT with clinical, inflammatory and metabolic variables

Spearman's rho correlation coefficient between CT parameters, serum biomarkers, and clinical variables are presented in Table 2. As also showed in Fig 2 the EAT was positively correlated with glycemia (rho=0,17, p=0,0002), and with inflammatory markers as PCR (rho=0.30, <0,001), and fibrinogen (rho=0.16, p=0,032). An inverse correlation was observed between EAT and P/F ratio (rho= -0,24, p=0,0006) and between EAT and P/F nadir (rho= -0,30, p=0,001)

### 4.4 Predictors of ARDS/death in COVID-19 patients

Univariate analysis showed a significant association between age glycemia, EAT volume and ARDS onset or death. In the multivariable logistic regression analysis, age (OR 1.025, 95%CI: 1.003 - 1.049, p-value=0.0291) and EAT volume (OR 1.009, 95%CI: 1.002 - 1.018, p-value=0.0207) were independently associated with ARDS onset or death.
Glycemia (OR 1.006, 95% CI 1.000; 1.014) and EAT attenuation (OR 1.011, 95% CI 0.999; 1.024) remained in the model as risk factors for ARDS/death with a trend of statistical significance (Table 3).

5. Discussion

The objective of this retrospective study was to identify biochemical parameters and CT characteristics associated with ARDS related to COVID-19 or death. The need to identify early clinical markers of COVID-19 severity is compelling.

Obesity has been identified as a risk factor for hyperventilation syndrome in Intensive Care Unit (ICU) patients (19) and for respiratory failure in patients with ARDS (20).

Obesity is also associated with chronic inflammation and visceral adipose tissue is capable of secreting inflammatory mediators such as IL-6, TNF, INF gamma, IL1 beta (21-23). Moreover, in patients with obesity and diabetes, the expression of Angiotensin-converting enzyme 2 (ACE2), the functional receptor for SARS-CoV-2, is upregulated in adipocytes and turns adipose tissue into a potential target and viral reservoir. This may explain why the excess of VAT and diabetes are potential empowering factors for COVID-19 infection (24).

Some reports showed that CT-based quantification of VAT and upper abdominal circumference in routine chest CTs were associated with worse clinical outcome in patients with SARS-CoV-2 infection and as EAT closely correlated with abdominal visceral adiposity and metabolic risk factors (25-26).

Some authors have investigated the association between EAT and COVID-19 severity showing how epicardial fat measured in terms of thickness at the level of the right coronary artery origin on the axial plane (8,25) was similar among the groups of COVID-19 severity.

Other authors, using a fully automated, three-dimensional measurement of epicardial fat, which gives a better estimate of visceral deposits, have shown that EAT volume and attenuation were associated with the quantitative burden of Covid-19 pneumonia and that both parameters independently predicted clinical deterioration or death (9,27).

In our study, using a semiautomatic three-dimensional EAT volume quantification system, a significantly higher value of EAT volume was observed in the ARDS/death group and it was a strong independent predictor of negative outcome. It was also interesting to note that our sample was stratified based on the severity of ARDS (mild, moderate and severe), EAT volume showed a progressive increase in parallel with the degree of ARDS. In fact, not only P/F at baseline but also P/F nadir was positively correlated with EAT.

The lack of difference in EAT attenuation between the two groups and among patients with different degrees of ARDS, could be probably explained by the attenuation calculated exclusively on a ROI of 6 mm.

The role of EAT as a viral reservoir and cytokine storm amplification site, could explain not only a worsening of lung function, but also the clinical deterioration up to multiorgan failure and death, through a direct/paracrine and indirect/systemic effect (27-30).

Interestingly, our study showed that EAT volume correlated with metabolic parameters, in particular with blood glucose.

We found that fasting glucose was higher in the ARDS/death group and that fasting glucose at admission, had a trend as an independent risk factor for ARDS or death. This data could be confirmed by increasing the sample size.

This was in line with previous reports highlighting that admission hyperglycemia was a strong predictor of radiographic findings of ARDS (15) and that not only diabetes but also infection-related hyperglycemia at admission were associated with higher risks of adverse outcomes among patients with COVID-19 (31).

Further studies are needed to evaluate both the role of tight glycemic control in the outcome of COVID-19 pneumonia and the possibility of modifying EAT with target therapies to prevent the outbreak of the immune response.

Our study has some limitations. First, the nature of this study was retrospective and only selected biochemical and clinical parameters were available. Second, we didn't have the possibility of applying cardio-synchronization software to the CT acquisition. Third, the epicardial fat attenuation was calculated on a 6 mm ROI which did not allow an average estimate of the whole EAT attenuation.

Some strengths should also be emphasized. First, our sample size was larger than in other studies. Second, we were able to obtain the BMI of about half of the patients, despite the critical health and isolation conditions that prevented the physical examination of the patients, which allowed a preliminary anthropometric comparison between the two groups, even if we were unable to include this obesity measure in our multivariable model. Third, the same decision-making protocol for hospitalization and treatment for all patients made the
sample homogeneous in the severity of COVID-19 pneumonia or in hospital outcomes. Fourth, chest CT indications and acquisition protocols were standardized. Fifth, the semiautomatic quantification of the epicardial fat volume and the positioning of the ROI for the estimation of tissue attenuation were performed by the same operator.

In conclusion, we found that EAT was correlated strictly with inflammatory markers, as C-reactive protein and fibrinogen and fasting blood glucose. Our findings suggest that hyperglycemia in hospitalized patients should be adequately treated in patients with or without diabetes and that both blood glucose and EAT, as measurable and modifiable targets, could be included in a risk score for patients hospitalized with Covid-related pneumonia, in order to predict a worst prognosis and improve the therapeutic approach.

Indirect parameters of inflammation, such as hyperglycemia and EAT, easily obtained by chest CT in patients with suspicion of COVID-19, could allow the early identification of subjects at greater risk of developing severe complications.

6. Declarations

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Declaration of Competing Interest

The authors declare no conflict of interest.

Ethical approval

All procedures performed were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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8. Tables
## Table 1
Anthropometric, clinical and laboratory characteristics of patients on admission

|                                | ARDS or death | P value |
|--------------------------------|---------------|---------|
|                                | No (N = 99)   | Yes (N = 130) |   |
| **Anthropometric and clinical characteristics** |               |          |
| Age, years                      | 57 (47–68,5)  | 66 (55-78.75) | < 0,001 |
| Male sex                        | 52 (52,5)     | 78 (60)     | 0,319 |
| Weight, kg (126)                | 76,5 (65–85), 50 | 80 (72–90), 76 | 0,022 |
| Body mass index, kg/m2 (126)    | 26,23 (23,57 – 29,41), 50 | 27,78 (24,58, 30,91), 76 | 0,05 |
| Systolic blood pressure         | 122,5 (120–140) | 120 (120–140) | 0,932 |
| Diastolic blood pressure        | 80 (70–80)    | 80 (70–80)  | 0,862 |
| Diabetes mellitus (%)           | 18,2          | 36,2       | 0,005 |
| Hypertension (%)                | 27,5          | 56,3       | 0,004 |
| Cardiovascular events (%)       | 10            | 10,8       | 0,901 |
| P/F ratio                       | 400 (359,5-457) | 298 (218–356) | < 0,001 |
| P/F nadir                       | 381 (388–439) | 155 (123,25–211,25) | < 0,001 |
| **Blood biomarkers**            |               |          |
| Leukocytes (*10³/µL)            | 5,810 (4,275-7,708) | 5,630 (4,115-7,780) | 0,864 |
| Neutrophils (*10³/µL)           | 3,705 (2,505-5,667) | 4,330 (2,345-6,00) | 0,485 |
| Lymphocytes (*10³/µL)           | 1,440 (0,993-1,868) | 0,990 (0,735-1,360) | < 0,001 |
| Neutrophils (%)                 | 61,83 ± 12,66 | 70,85 ± 14,83 | < 0,001 |
| Lymphocytes (%)                 | 26,45 ± 10,26 | 20,31 ± 10,99 | 0,002 |
| Hemoglobin (g/dl)               | 13,51 ± 1,64  | 13,23 ± 1,93 | 0,264 |
| Platelets (*10³/µL)             | 209 (176–259)  | 197 (153–257) | 0,394 |
| Glycemia (mg/dl)                | 101 (91–118)  | 114 (98–144) | 0,001 |
| Total cholesterol (mg/dl) (73)  | 165,28 ± 32,45 | 152,34 ± 36,01 | 0,116 |
| High-density lipoprotein cholesterol (mg/dl) (73) | 36 (25,5–48) | 31(24,5–37) | 0,044 |
| Low-density lipoprotein cholesterol (mg/dl) (73) | 107,4 ± 28,09 | 99,59 ± 34,94 | 0,306 |
| Triglycerides (mg/dl) (73)      | 95 (68–130)   | 120 (88,5-136,5) | 0,103 |
| Uricaemia (mg/dl) (66)          | 4,7 (3,75 – 5,62) | 4,65 (3,73 – 6,7) | 0,568 |
| Aspartate Aminotransferase (AST, U/L) | 21 (16,5–26,5) | 27 (20–42) | < 0,001 |
| Alanine Aminotransferase (ALT, U/L) | 21 (14,5–29,5) | 24 (15,5–40) | 0,160 |
| Gamma Glutamyl Transferase (GGT, U/L) (78) | 25,5 (15,75 – 42,75) | 35 (20–57) | 0,015 |
| Creatinine (mg/dl)              | 0,84 (0,73 – 1,09) | 0,95 (0,77 – 1,27) | 0,113 |
| Lactate dehydrogenase (U/L)     | 198,5 (177,75-246m25) | 288,5 (216,25–375,25) | < 0,001 |
| Erythrocyte sedimentation rate (ESR) (mm/h) | 35 (17,5–50,75) | 56 (35–72,5) | 0,008 |
| C-reactive protein (mg/dl)      | 0,67 (0,20 – 2,69) | 4,62 (1,53 – 13,54) | < 0,001 |
| Ferritin (ng/mL) (61)           | 258 (130–429)  | 339,5 (171,5-948) | 0,113 |
|                | ARDS or death | P value |
|----------------|---------------|---------|
|                | No (N = 99)   | Yes (N = 130) |
| Fibrinogen     | 386,5 (318–448,5) | 464 (397,5-567) | < 0,001 |
| D-dimer (mg/L) | 0,42 (0,28 – 0,99) | 0,96 (0,59 – 1,63) | < 0,001 |
| Albumin (40)   | 3,81 ± 0,46   | 3,28 ± 0,53 | 0,002 |

Radiological assessments

|                |                |                |
|----------------|----------------|----------------|
| EAT (cm³)      | 78,95 (51,52–99,95) | 103,00 (69,92–128,75) | < 0,0001 |
| EAT density (HU) | -87,76 (-100,72- -70,38) | -89,99 (-103,70- -74,73) | 0,684 |

Data are n (%), median (IQR), or mean ± SD, n if fewer patients had laboratory results available than the total study population.

Table 2

- Correlation matrix between investigated variables.

|        | Age   | EAT volume | Glycemia | BMI | HDL | Gamma GT | AST | ALT | uricaemia | P/F ratio | fibrinogen | PCR |
|--------|-------|------------|----------|-----|-----|----------|-----|-----|-----------|-----------|------------|-----|
| Age    | 1,00  |            |          |     |     |          |     |     |           |           |            |     |
| EAT volume | 0,31* | 1,00       |          |     |     |          |     |     |           |           |            |     |
| Glycemia | 0,17* | 0,28*      | 1,00     |     |     |          |     |     |           |           |            |     |
| BMI    | 0,09  | 0,47*      | 0,17     | 1,00|     |          |     |     |           |           |            |     |
| HDL    | -0,08 | -0,27*     | -0,22    | -0,22*| 1,00|         |     |     |           |           |            |     |
| Gamma GT | -0,07 | 0,33*      | 0,25*    | 0,33*| -0,30*| 1,00     |     |     |           |           |            |     |
| AST    | -0,065 | 0,14      | 0,17*    | 0,24*| -0,20| 0,556*   | 1,00|     |           |           |            |     |
| ALT    | -0,22* | 0,15*      | 0,21*    | 0,41*| -0,13| 0,66*    | 0,72*| 1,00|           |           |            |     |
| uricaemia | 0,04  | 0,36*      | 0,19     | 0,37*| -0,18| 0,30*    | 0,33*| 0,11| 1,00     |           |            |     |
| P/F ratio | -0,40* | -0,25*    | -0,11    | -0,33| 0,32*| -0,24*   | -0,15| -0,16| -0,08    | 1,00     |            |     |
| fibrinogen | 0,10  | 0,16*      | 0,29*    | 0,00| -0,14| 0,29*    | 0,312*| 0,29*| -0,07    | -0,01    | 1,00     |     |
| PCR    | 0,23* | 0,30*      | 0,20*    | 0,23*| -0,42*| 0,41*    | 0,452*| 0,26*| 0,13     | -0,50*    | 0,58*    | 1,00|

* Correlation is significant at the 0,01 level
* Correlation is significant at the 0,05 level
Table 3
Odds Ratio (OR) of Risk Factors for developing ARDS or death at univariate and multivariate analysis.

| Variable               | Univariate |               |            | Mutivariate |               |            |
|------------------------|------------|---------------|------------|-------------|---------------|------------|
|                        | OR         | 95% CI        | p-value    | OR          | 95% CI        | p-value    |
| Glycemia (mg/dl)       | 1.009      | 1.003–1.017   | 0.0122     | 1.006       | 1.000–1.014   | 0.0904     |
| Age (yrs)              | 1.035      | 1.014–1.058   | 0.00108    | 1.025       | 1.003–1.049   | 0.0291     |
| EAT density HU         | 1.004      | 0.994–1.015   | 0.403      | 1.011       | 0.999–1.024   | 0.0875     |
| Gender F vs M          | 0.730      | 0.402–1.321   | 0.299      | -           | -             | -          |
| EAT volume (cm$^3$)    | 1.011      | 1.004–1.018   | 0.00218    | 1.009       | 1.002–1.018   | 0.0207     |

Figures

Figure 1
A-B: Differences in epicardial adipose tissue volume (A) and attenuation (B) in patients with and without clinical deterioration or death; C-D: Differences in epicardial adipose tissue volume (A) and attenuation (B) in patients without ARDS, and with increasing degrees of ARDS (mild, moderate and severe).

Figure 2

Correlation between EAT and Glycemia, P/F ratio at baseline, P/F nadir, PCR and Fibrinogen

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- APPENDIXA.docx