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Fifty-two patients admitted to the Navarra Hospital Complex

from 22nd March to 5th April 2020 with confirmed SARS-CoV-2

infection were prospectively included. Patients with pathologies

other than SARS-CoV-2 infection as a cause of admission were

excluded. The CURB65 clinical scale was calculated⁴ for stratifica-

tion of patients. Blood tests were performed upon admission with the following inflammatory markers: lymphocyte count $(\cdot 10^9)$,

fibrinogen (mg/dl), dimer-D (ng/ml), ferritin (µg/l), LDH (U/l), troponin-I (pg/ml) and CRP (mg/l). These determinations were

repeated at 48 h of admission after the implementation of the treat-

ment protocols indicated at that time. The main outcome variable

was a combined variable that included death during admission

or the need for transfer to the ICU due to SARS-CoV-2 infection.

Univariate logistic regression models were constructed for the out-

come variable and ROC (receiver-operating characteristics) analysis

of the models was performed.

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Scientific letter

Risk factors and severity predictors in COVID-19 hospitalized patients: Analysis of 52 patients

Factores de riesgo y predictores de gravedad en pacientes hospitalizados por COVID-19: análisis de 52 casos☆

To the Editor:

The SARS-CoV-2 infection presents, in most cases, with mild symptoms.¹ However, up to 13% of cases develop severe symptoms,² with acute respiratory distress associated with a massive release of pro-inflammatory mediators.³ The identification of patients at risk of developing severe symptoms would allow optimization of therapeutic algorithms. This study analyses demographic, clinical-radiological and analytical data of admitted patients to try to establish predictive mortality and ICU admission varia

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variables.	-			
Table 1 Characteristics of the patients.				
Variables	Total (N = 52)	Death or admission to ICU $(N=8)$	Discharge without admission to ICU ($N = 44$)	р
Age in years, mean (SD)	65.5 (15.2)	74 (10.6)	64.0 (15.5)	0.04 ^a
Male, n/N (%)	36/52 (69.2)	8/8	28/44	0.04 ^b
Hypertension, n/N (%)	29/52 (55.8)	6/8	23/44	0.21 ^b
Diabetes mellitus, n/N (%)	14/52 (26.9)	3/8	11/44	0.67 ^b
Smoking	3/47 (6.4)	1/8	2/39	0.77 ^b
Non-ischemic heart disease, n/N (%)	9/50 (18)	1/7	8/43	1.0 ^b
Previous respiratory disease, n/N (%)	6/49 (12.2)	1/8	5/41	1.0 ^b
CKD with GFR < 45%, n/N (%)	4/49 (8.2)	1/8	3/41	0.52 ^b
Days of progression at admission, median (IQR)	8 (5-11)	5 (4-6)	8.5 (6-12)	0.01 ^c
SatO ₂ \leq 91% in the emergency department, n/N (%)	9/49 (18.4)	3/7	6/42	0.11 ^b
Days with T>37.5°, median (IQR)	6.5 (2-10)	1 (0-2)	7 (3-10)	0.02 ^c
CURB65 score, median (IQR)	1 (0-1)	2.5 (1-3.5)	1 (0-2)	< 0.01 ^c
Chest X-ray findings, n/N (%)				
Normal	4/49 (8.2)	0/7	4/42	
Interstitial pattern <2 lobes	7/49 (14.3)	1/7	6/42	
Interstitial pattern> 2 lobes	18/49 (36.7)	1/7	17/42	
Interstitial pattern + opacities	20/49 (40.8)	5/7	15/42	
Anosmia/dysgeusia, n/N (%)	11/50 (22)	3/7	8/43	0.17 ^b
Lymphopenia on admission, n (%)	23/52 (44.2)	6/8	17/44	0.07 ^b
Fibrinogen on admission in mg/dl, median (IQR)	703.5 (599-825)	705.5 (593.5-948.5)	703.5 (599-789)	0.74 ^c
D-dimer on admission (ng/ml), median (IQR)	763 (426-1,051)	1,445 (953-2,053)	701.5 (421-914.5)	< 0.01 ^c
Ferritin on admission (μ g/l), median (IQR)	557 (216-925)	597 (394-1,372)	557 (193-924)	0.46 ^c
LDH on admission (U/l), median (IQR)	276.5 (236-321)	317.5 (278-439)	266 (229-315)	0.08 ^c
Troponin I on admission (pg/ml), median (IQR)	5 (3-11)	49 (6-66)	4 (3-8)	0.01 ^c
CRP on admission (mg/l), median (IQR)	94 (42.5-136.5)	115 (60.5-320)	87.5 (38-135)	0.20 ^c
Deaths, n (%)	5/50 (10)	\downarrow	\downarrow	\downarrow
Admission to ICU, n (%)	3/50 (6)	Ļ	Ļ	\downarrow

SD: standard deviation; CKD: chronic kidney disease; GFR: glomerular filtration; LDH: lactate dehydrogenase; CRP: C-reactive protein; IQR: interquartile range 25-75; ICU: intensive care unit.

^a Calculated by one-sided Student's t.

^b Calculated by Fisher's exact test.

^c Calculated by Wilcoxon test.

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A total of 52 patients were analysed (Table 1). At least 3 of the 7 parameters of inflammation evaluated were elevated in all of them. A correlation was observed between CRP levels and LDH levels (Pearson's r coefficient, 0.44, p < 0.01), fibrinogen (0.74, p < 0.01) and ferritin (0.40, p < 0.01). D-dimer levels at admission were correlated with troponin-I levels (0.66, p < 0.01) but not with acute phase reactants (CRP, LDH, fibrinogen, and ferritin).

The score on the CURB65 scale was associated with a higher risk of dying or requiring admission to the ICU (OR 4.27; CI 95%: 1.6-11.25). Among the inflammatory parameters on admission, troponin-I levels (OR 2.21; CI 95%: 1.17-4.16) and D-dimer (OR 11.98; CI 95%: 1.72-83.27) were associated with a worse prognosis. Increasing D-dimer levels above laboratory normal limits (500 ng/ml) showed a negative predictive value of 100%. The ROC analysis of the predictive ability of D-dimer levels showed an AUC of 0.81 (CI 95%: 0.69-0.92), and a cut-off point>1,200 ng/ml showed a sensitivity of 71.43% and a specificity of 90.91%.

Increasing levels of troponin-I above laboratory normal limits (34 pg/ml) associated a worse prognosis (Wilcoxon test, p < 0.01). ROC analysis for troponin-I levels yielded an AUC of 0.81 (CI 95%: 0.67-0.92), with a cut-off point of 34 pg/ml to obtain the best sensitivity (66.67%) and specificity (91.89%) data.

D-dimer values (OR 4.89; CI 95%: 1.26-18.93) and CRP at 48 h were associated with the risk of dying or requiring admission to the ICU (OR 5.36; CI 95%: 1.19-24.09). A prognostic scale was created with the number of inflammatory parameters increased above the normal limits, associating the score at admission to a worse prognosis (for each increased value: OR 2.6; CI 95%: 1.17-5.76).

Some of the factors described in the literature as predictors of a worse prognosis in SARS-CoV-2 infection (age and male sex) are maintained in our study.¹ All of the deceased patients (5/52) and those who required ICU were male, and advanced age was associated with higher mortality. In addition, higher scores on the CURB65 scale were associated with higher mortality, as in other published studies.⁵

Previous studies have shown an increase in inflammatory parameters and mild cytopenia in cases of severe progression.³

Comparison of telehealth and traditional face-to-face model during COVID-19 pandemic*

Comparación de la teleconsulta con el modelo presencial tradicional durante la pandemia COVID-19

To the Editor,

As early as 1974, telehealth was being discussed as a link between hospitals and homes.¹ There were few publications on the subject until 1992, the turning point when publications on this model started to emerge. Face-to-face interactions will always play a central role in our healthcare system. But a system based on highquality remote care might work better for many patients and quite possibly for some doctors as well.² Since the advent of SARS-CoV-2, telehealth has become a useful tool in certain healthcare systems.³ This disruptive experience has meant a sudden and total shift from Elevated levels of D-dimer and troponin-I on admission were associated with higher mortality and disease severity in our population, with a negative predictive value of 100% in the case of D-dimer.

In conclusion, the use of inflammatory parameters such as troponin-I or D-dimer, as well as clinical scales such as CURB65, help to predict a worse COVID-19 disease progression. Their implementation in clinical practice makes it possible to optimize therapeutic algorithms and rationalize resources in situations of health crisis.

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face-to-face consultations to a virtual model, unprecedented in many health systems.

Our department implemented a comprehensive telecardiology model from 16th March until 1st May, time when we progressively returned to face-to-face activity. We do not yet have a specific tool, therefore, the model relied on two simple pillars, the electronic medical record and the telephone call as a means of communication with users and colleagues.

A total of 1721 teleconsultations were carried out, of which 1339 came from general consultations, 67 from the cardiac rehabilitation consultation and 315 from the monographic consultation on Advanced Heart Failure.

For the analysis of the results we propose 3 possibilities: (1) follow-up (it is resolved by teleconsultation and requires a check-up/complementary test); (2) resolved (it is resolved by teleconsultation without the need for further follow-up) and (3) re-appointment (requires a face-to-face visit).

Of the total of 1721 patients contacted by teleconsultation, 1156 (67.2%) were referred for a follow-up, 332 (19.3%) were resolved and only 233 (13.5%) required re-appointment



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