Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Letter to the Editor

Impact of nosocomial acquisition of COVID-19 in hospitalized cardiac patients

Sir,

The nosocomial transmission of SARS-CoV-2, which causes COVID-19, is a critical aspect of the pandemic due to its association with greater morbidity and mortality of hospitalized patients. Nosocomial transmission also highlights failures in the health system in preventing the propagation of this virus. It is therefore crucial to prevent this transmission through vaccinating the professional team, environmental disinfection, and appropriate isolation of suspected or confirmed cases [1,2]. To describe and quantify nosocomial transmission is essential for the safety of both patients and the assisting team [3]. Cardiac patients have worse outcomes when affected by COVID-19 [4]. Our goal was to compare the demographic, clinical, and laboratory features as well as outcomes of cardiac patients who contracted COVID-19 (hospital-acquired (HA)-COVID-19) in our cardiology institute with those who were admitted when already infected with COVID-19 (non-hospital-acquired (NHA)-COVID-19). This is a retrospective observational study of adults admitted between March and September 2020, diagnosed as infected with SARS-CoV-2 by a positive nasopharyngeal reverse transcription—polymerase chain reaction swab. The data were collected in accordance with the International Severe Acute Respiratory and Emerging Infection Consortium and complemented with a clinical cardiovascular evaluation questionnaire [4]. HA-COVID-19 was diagnosed when the patient showed a negative nasopharyngeal swab upon hospital admission which became positive $\geq$14 days after admission. Statistical analysis was carried out with Jamovi 1.6 e R 4.0.1. One hundred and twenty-one patients with a confirmed diagnosis of COVID-19 were included in the study. There were 20 patients (16.5%) with HA-COVID-19 and 101 (83.5%) with NHA-COVID-19.

The reasons for hospitalization in the HA-COVID-19 group were decompensated heart failure (35%), acute coronary syndrome (20%), cardiac surgery (25%), complete atrioventricular block (5%), pacemaker dysfunction (5%), stroke (5%), and haematological disease (5%). Median age and interquartile range were 64 years (61.8—69.3) for HA-COVID-19 and 63 (52—72) for NHA-COVID-19, without significant statistical differences. There were no differences between the two groups regarding the frequency of heart disease, chronic obstructive pulmonary disease, obesity, complicated diabetes mellitus, heart valve disease, acute coronary syndrome, and systemic arterial hypertension. However, patients with HA-COVID-19 had more dyslipidaemia (15/20 (75%) vs 49/101 (48.5%), $P = 0.030$) and chronic renal failure (7/20 (35%) vs 15/101 (15%), $P = 0.033$). They presented a more severe infectious syndrome, and more frequently had coagulation disorders, anaemia and acute renal failure. Mortality was considerably higher in HA-COVID-19 (10/20 (50%) vs 19/101 (18.8%), $P = 0.003$). Furthermore, the two groups were significantly different in relation to laboratory data: serum creatinine, D-dimer, ferritin, and aspartate aminotransferase all showed higher levels in the HA-COVID-19 cases, whereas lymphocyte count, platelet count, and haemoglobin levels were lower (Table I). Longer length of hospital stay was seen in patients with HA-COVID-19. These results clearly show the worse outcome of patients with HA-COVID-19, with a more complicated course and higher mortality, highlighting the impact of the pandemic in the outcomes of cardiac patients who were hospitalized due to causes not related to COVID. Few data exist about the risks and complications of COVID-19 acquired in the hospital environment.

Several studies, however, address the prevalence of nosocomial COVID-19 [1—3,5—8]. An early systematic review found that the proportion of nosocomial infection in patients with COVID-19 was 44% in the early outbreak [5]. A Chinese study at the start of the pandemic with 138 patients showed that 41% of cases were hospital-acquired. Another study carried out in a London hospital revealed that 15% of patients with COVID-19 were definitely or probably nosocomial and the lethality in these patients was 36% [6]. Another study in the UK during the first wave of COVID-19 estimated that 11.3% (95% confidence interval: 11.1—11.6) of patients acquired COVID-19 in hospital, across 314 UK hospitals [1]. Another UK study identified 505 COVID-19-positive inpatients between March and April 2020, and the HA-COVID-19 cases represented 11.3%; patients were older (mean: 77.4 years) compared to community cases (mean: 67.7) [3]. In a large survey in Brazil, in the second wave of COVID-19, from August 2020 to September 2021, 48,634 cases of HA-COVID-19 were reported from 1428 hospitals, with a peak in March 2021; it was estimated that 3.17% of patients in adult ICU acquired COVID-19 in hospital [7]. Other series estimated the rate of nosocomial acquisition to be 7%, 8%, 15%, and 44%, respectively [2,3,5,7]. Elderly patients who are hospitalized are fragile and show comorbidities which make them more vulnerable to the resulting complications of

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Table I
Comparison of clinical and laboratory features of patients with hospital-acquired COVID-19 (HA-COVID-19) with those with non-hospital-acquired COVID-19 (NHA-COVID-19)

Variables	*N*	HA-COVID-19	NHA-COVID-19	*P*-value
Length of hospital stay[a] (days)	121	29 (18.75–60.2)	16 (7–31)	<0.001
Lymphocyte count[a] (cells/μL)	121	1763 (970.2–2437.5)	1309 (762–1928)	<0.001
Glucose[a] (mg/dL)	121	138 (100–157)	120 (96–166)	<0.001
Alanine aminotransferase[a] (IU)	87	16.5 (14.5–36)	30 (18–47)	<0.001
Creatinine[a] (mg/dL)	119	1.4 (1.05–2.58)	1.07 (0.85–1.46)	<0.001
C-reactive protein[a] (mg/dL)	106	7 (1.6–24)	6.2 (1.5–14)	<0.001
Ferritin levels[a] (μg/L)	54	1031 (225–1637)	676 (346–1610)	<0.001
D-dimer[a] (ng/mL)	62	2180 (820–3945)	1130 (535–2012)	<0.001
Aspartate aminotransferase[a] (IU)	87	43.5 (21.2–70.5)	31 (20–54)	<0.001
Haemoglobin[a] (g/dL)	121	11.7 (10.02–13.15)	12.5 (11–14)	<0.001
Leucocyte count[a] (cells/μL)	121	8665 (7050–10,840)	6740 (4910–9300)	<0.001
Total bilirubin[a] (mg/dL)	65	0.57 (0.55–1.17)	0.5 (0.35–0.83)	<0.001
Platelets[a] (/μL)	121	238,000 (199,250–334,000)	212,000 (162,000–293,000)	0.003
Vasoactive drugs	121	13/20 (65%)	26/101 (25.7%)	<0.001
Severe infectious syndrome	121	10/20 (50%)	18/101 (17.8%)	0.002
Pleural effusion	119	7/19 (36.8%)	16/100 (16%)	0.035
Cardiac arrest	120	10/20 (50%)	23/100 (23%)	0.014
Coagulation disorder	121	6/20 (30%)	8/101 (7.9%)	0.005
Anaemia	121	10/20 (50%)	25/101 (24.8%)	0.023
Acute renal failure	121	10/20 (50%)	27/101 (26.7%)	0.039

Reference values: ferritin: <341 ng/mL; D-dimer: <500 ng/mL; leucocyte count: 4000–10,000/μL; lymphocyte count: 800–4500/μL; haemoglobin: 11.5–16.4 g/dL; C-reactive protein: <0.5 mg/dL.
[a] Median (interquartile range).

COVID-19. Although in our study patients with and without HA-COVID-19 did not differ in age, those with HA-COVID-19 had more dyslipidaemia and chronic renal failure. Furthermore, longer length of hospital stay increases the risk of acquiring COVID-19 in the hospital, which in turn prolongs hospitalization and aggravates outcomes [2,6]. We conclude the COVID-19 pandemic was a challenge both in the community and hospital settings, and the prevention of healthcare-associated infection remains cornerstone, due to known higher prevalence of older and sicker patients in healthcare units.

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None declared.

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