Interactive CardioVascular and Thoracic Surgery (2020) 1–2
doi:10.1093/icvts/ivaa210

Cite this article as: Varela Barca L, Torralba Cloquell I, Herrero Cereceda J, Sáez de Ibarra JI. An unexplained death after routine cardiac surgery: how long have we dealt with coronavirus disease 2019? Interact CardioVasc Thorac Surg 2020; doi:10.1093/icvts/ivaa210.

An unexplained death after routine cardiac surgery: how long have we dealt with coronavirus disease 2019?

Laura Varela Barca [a,*], Isabel Torralba Cloquell [b], Jaime Herrero Cereceda[c] and Jose Ignacio Sáez de Ibarra[a]

[a] Department of Cardiac Surgery, Son Espases University Hospital, Palma de Mallorca, Spain
[b] Department of Pathology, Son Espases University Hospital, Palma de Mallorca, Spain
[c] Intensive Care Unit, Son Espases University Hospital, Palma de Mallorca, Spain

* Corresponding author. Department of Cardiac Surgery, Son Espases University Hospital, Palma de Mallorca, Spain. Tel: +34-617103918; e-mail: lauravarela21089@gmail.com (L. Varela Barca).

Received 19 June 2020; received in revised form 17 August 2020; accepted 31 August 2020

Abstract

We present a case report of fatal respiratory failure after cardiac surgery in the early stages of the coronavirus disease 2019 outbreak. Although not supported by epidemiological data nor clinical course, coronavirus disease 2019 infection was revealed post-mortem by immunohistochemical detection of the severe acute respiratory syndrome coronavirus 2 spike protein in lung tissue.

Keywords: COVID-19 infection • Immunohistochemical detection • Cardiac surgery • Post-mortem transmission

CASE REPORT

On 30 January 2020, a 76-year-old woman was presented to a secondary referring hospital with dyspnoea and ventricular tachycardia. After receiving medical treatment for arrhythmia, a transthoracic echocardiogram showed a severe aortic stenosis, normal heart function, severe hypertrophy and increased chamber size. The patient was afebrile and an X-ray was anodyne. Electrocardiogram showed a sinus rhythm with left bundle branch block. Dyspnoea was attributed to pulmonary oedema and was managed with diuretics and nasal oxygen. The calculated EuroScore-II was 6.67%.

The patient was transferred to our centre and an aortic valve replacement was performed 4 days after the admission. A Magna Ease® biological valve was implanted without complications. There were no sternum fractures nor any other problems during the sternum closure. During intensive care unit admission, she presented an adequate postoperative recovery. Mechanical ventilation disconnection was performed 8 h after surgery, and she was moved to hospitalization within the first 24 h.

Forty-eight hours after surgery the patient presented a worsening in respiratory function with non-productive cough, high respiratory rate and necessity for supplemental O_2 flow. Neither high temperature nor leucocytosis was observed. As her cough became stronger, the patient suffered a sudden sternum fracture with acute bleeding and cardiac tamponade in the transthoracic echocardiogram. An emergent sternum reparation surgery was required during which damage of the right ventricle was observed. Multiple tests were done in search for an infection including for: urine, blood and sputum culture. No infection was found.

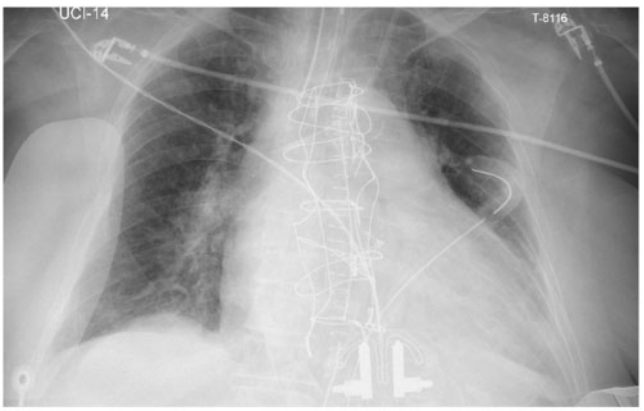

Figure 1: Chest X-ray: cardiomegaly. Pleural effusion with perihilar distribution.

After the second surgery, disconnection from mechanical ventilation was hampered by respiratory failure until the 9th postoperative day. Twenty-four hours after disconnection, a second sternum fracture coinciding with a bout of severe cough took place. This was also followed by high fever, and a second sternum reparation procedure had to be performed. Twenty-four hours later, the patient suffered distributive shock followed by cardiac arrest and asystole. She was pronounced dead on 1 March.

A nosocomial pneumonia was the main suspected cause of death. However, the X-rays did not show typical opacities (Fig 1). Given the absence of coronavirus disease 2019 (COVID-19) cases

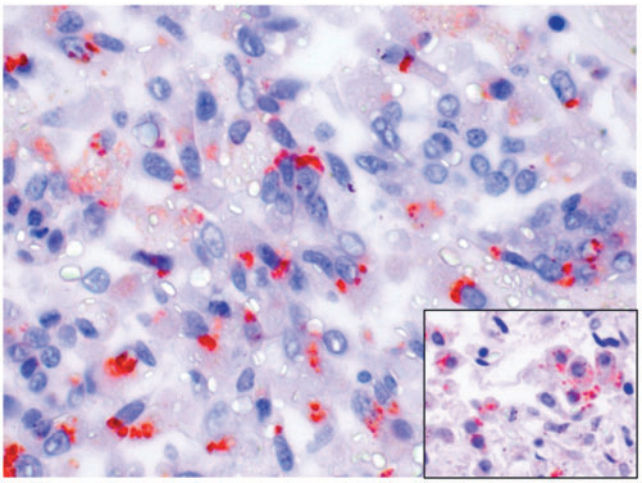

Figure 2: Immunohistochemistry for severe acute respiratory syndrome coronavirus 2 spike protein (red) in sloughed bronchiolar cells. Similar findings were seen in alveolar pneumocytes and macrophages (inset).

during that period in our region, polymerase chain reaction for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was not performed. There was no history of any risk contacts. Permission for a clinical autopsy was granted. The post-mortem results of 1 blood test showed the isolation of *Klebsiella pneumoniae*. Post-mortem examination revealed oedematous lungs with sloughing of bronchiolar epithelium and pneumocyte hyperplasia without hyaline membranes; within the alveoli some fibrin deposits, abundant foamy macrophages and focal oedema could be seen. Rare multinucleated cells were seen. No areas of neutrophilic infiltration, necrosis or viral inclusions were identified. Special stains failed to detect micro-organisms. Immunohistochemistry for SARS-Cov-2 spike protein (1A9 GTX632604 by GeneTex®) was positive in desquamated bronchiolar cells, detached pneumocytes and alveolar macrophages (Fig 2).

DISCUSSION

We are reporting a fatality due to a COVID-19 infection in the early stage of the outbreak, after a cardiac surgery was performed, for which no known transmission route can be traced.

In our case report, the cause of death may have been multifactorial due to patient comorbidities and a complicated postoperative course, which included a sternal fracture and 2 cardiac tamponades that required 2 complex reoperations. However, an undiagnosed concomitant SARS-CoV-2 infection may have precipitated the catastrophic postoperative course.

In agreement with other reports [1], the clinical course of SARS-CoV-2 infection could be modified by cardiac intervention, since surgery has been identified as a modifying factor in COVID-19. Other comorbidities also play an important role in this regard; for example older age, hypertension, dyslipidaemia or intensive care unit admission have all been described as risk factors for in-hospital mortality [2].

The suspicion for COVID-19 infection arose from the pathology findings. Macrophagic infiltration with areas of haemorrhage and thrombosis, alveolar fibrin and multinucleated cells has been described recently in early COVID-19 infection [3]. In the few reports dealing with post-mortem specimens from COVID-19 patients, much emphasis has been placed on the detection of the virus by molecular methods, so as to identify the involved tissues, in an attempt to understand the natural history of the infection [4]. Only a few studies have been done employing immunohistochemistry [5].

ACKNOWLEDGEMENTS

Carles Saus Sarrias and Carlos Santonja Garriga for the post-mortem examination and immunohistochemistry techniques. Sandra Pérez Buira and Rafael Alberto Rojas Silva validated and performed the immunohistochemical studies.

INFORMED CONSENT

Permission for a clinical autopsy was granted.

Conflict of interest: none declared.

Ethical approval: No applicable

Reviewer information

Interactive CardioVascular and Thoracic Surgery thanks Roman Gottardi and the other, anonymous reviewer for their contribution to the peer review process of this article.

REFERENCES

[1] Rescigno G, Firstenberg M, Rudez I, Uddin M, Nagarajan K, Nikolaidis N. A case of postoperative COVID-19 infection after cardiac surgery: lessons learned. Heart Surg Forum 2020;23:E231–3.
[2] Ky B, Mann DL. COVID-19 clinical trials: a primer for the cardiovascular and cardio-oncology communities. JACC CardioOncol 2020;254–269.
[3] Luo W, Yu H, Gou J, Li X, Sun Y, Li J *et al.* Clinical pathology of critical patient with novel coronavirus pneumonia (COVID-19). Preprints 2020; 202002.0407.v1.
[4] Puelles VG, Lütgehetmann M, Lindenmeyer MT, Sperhake JP, Wong MN, Allweiss L *et al.* Multiorgan and renal tropism of SARS-CoV-2. N Engl J Med 2020;383:590–2.
[5] Martines RB, Ritter JM, Matkovic E, Gary J, Bollweg BC, Bullock H *et al.* Pathology and pathogenesis of fatal COVID-19 cases associated with SARS-CoV-2 pandemic. Emerg Infect Dis 2020;26:2005–15.