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Sars-Cov-2 In Pleural Effusion of Patients with Covid-19-A Case Report

A. DAFIR^{1*}, A. Raja¹, K. CHAWKI¹, S. LAMGHARI¹, C. El KETTANI¹, H. BARROU, F. ESSODEGUI², N. ELBENNA², N. ELMDAGHRI³, M. SOUSSI³

¹Department of anesthesia and intensive care, Ibn Rochd University Hospital Center, Casablanca, Morocco. ²Department of radiology Ibn Rochd University Hospital Center, Casablanca, Morocco. ³Parasitology and Microbiology laboratory, Ibn Rochd University Hospital Center, Casablanca, Morocco.

***Corresponding author:** DAFIR.A, COVID-19 Dedicated ICU, Ibn Rochd University hospital of Casablanca, Hassan II University, Faculty of Medicine and Pharmacy, Casablanca Morocco. Email: dafirasmae1987@gmail.com

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Abstract

On March 11, 2020, the World Health Organization (WHO) announced infection with SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2), called COVID-19 (coronavirus disease 2019), as a pandemic.

Early diagnosis of atypical cases of SARS-CoV-2 infection is essential if precautions based on the modes of transmission continue to be implemented in order to reduce transmission and control the current pandemic.

We report the case of a patient with a history of recurrent pleurisy admitted for Covid-19 viral pneumonia diagnosed through screening, and whose SARS COV-2 test was positive in pleural fluid.

Keywords: COVID-19, SARS-COV-2, PCR, Pleural fluid, Intensive care.

Introduction

The COVID-19 (coronavirus disease 2019) pandemic has affected more than 200 countries with more than eight million cases confirmed as of June 1, 2020. Similar to severe acute respiratory syndrome in 2003 and Middle East respiratory syndrome in 2012, COVID-19 infection primarily causes life-threatening pneumonia, suggesting that the virus primarily targets the respiratory system and is spread through droplets of air and through contact. In addition to oropharyngeal swabs, SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) has also been detected in blood, urine and facial / anal swabs, suggesting other potential means of transmission. However, it is unknown whether the virus can be detected in samples from other sites [1].

Medical Observation

77-year-old male patient with type 2 diabetes on oral antidiabetics and recurrent right pleurisy punctuated 4 times (etiology not labeled).

Initially admitted to the peripheral hospital center where he stayed 10 days for viral pneumonia at Covid-19 diagnosed as part of a screening, then transferred to the Ibn Rochd University Hospital Center in Casablanca for additional treatment. On admission, the patient was eupneic with 98% oxygen saturation in the open air with reduction of the vesicular murmur on the right at pleuropulmonary auscultation, normocardium 85 bpm / min, normotension at 106/68 and apyretic.

Biological balance: Hb at 8.9 g / dl, GB at 6490 / μ L, PNN at 4000 / μ L, Lymphocytes at 1770 / μ L, Fibrinogen at 3.69 g / l, D-dimers at 350 μ g / l, Urea at 0.83 g / l, Creatinine 14.3 mg / L, ASAT 20UI / L, ALAT 13UI / L, Albumin 31 g / L, Ferritin 419 ng / ml, CRP 21 mg / L, NT-Pro BNP 3211 pg / ml and Troponin at 34 ng / L.

The electrocardiogram (ECG) showed a regular sinus rhythm at 90bpm, an incomplete left branch block, corrected QT lengthened to 538ms hence the decision to stop the hydroxychloroquine (on D10 of the treatment).

A transthoracic ultrasound was made revealing a little dilated left ventricle (LV) in systolic dysfunction with an ejection fraction (FE) at 25%, a mitral profile (PM) type restrictive E / A> = 2.6, mitral insufficiency (MI) moderate to severe without stenosis, an undilated right ventricle (LV) of good longitudinal systolic function (TAPSE = 22), an average tricuspid insufficiency (IT) estimating the PAPS at 23mm hg and a complying lower vena cava at 11cm.

A thoracic computed tomography was made revealing a systematized right postero-basal parenchymal condensation associated with a right basi-thoracic pleural collection suggesting a bacterial superinfection with a large

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compressive right basi-thoracic pleural empyema measuring 17 x 11 cm.

Then we completed with a thoracic CT angiography which showed a right pleural effusion of great abundance encysted with lower lobar atelectasis. (Figure 1).

ICONOGRAPHY:



Figure 1: Thoracic CT angiography.

The indication for a pleural drainage was posed, bringing back 2 500 liters of purulent liquid over 48 hours with the presence of Pseudomonas aeruginosa (PARI) sensitive to Ceftazidime in culture and therefore the patient was put on Céftazidime + Metronidazole + Vancomycin.

A PCR test for SARS-COV 2 in the pleural fluid was made and returned Positive

Subsequently, our patient developed hemodynamic instability with confusion and agitation. We realized:

- An ECG objectifying a complete BBG with a septo-apicolateral ST elevation.
- Troponins were at 4700 ng / L.
- A cardiac evaluation revealed hypokinetic dilated cardiomyopathy with a 15% LVEF, fused PM, dilated OG, moderate IT estimating PAPS at 36 + 5 = 41 mm Hg, very high PRVG with an ITV under aortic at 10 mm Hg.
- A cerebral computed tomography showing a left capsulo-lenticular lacunar hypodensity with a sequellaire-like appearance. (Figure 2).



Figure 2: Cerebral tomography.

• A control chest CT scan found a left para-cardiac pulmonary embolism with a Qanadli score of 25%, a decrease in the volume of the right pleural collection and an increase in the volume of the left pleural effusion. (Figure 3).



Figure 3: Thoracic CT angiography.

The patient benefited from:

- Oxygenation based on high concentration mask.
- Pleural puncture on the left side which brought back 1 L 50 of citrine yellow liquid, transudative to 9 g of proteins with absence of germs visible on direct

examination and a PCR of the pleural liquid which had returned negative.

- Introduction of vasopressors based on norepinephrine and dobutamine.
- Introduction of diuretics.
- Continuation of antibiotic therapy.

- Discontinuation of unfractionated heparin and introduction of fondaparinux 7.5 mg / day before thrombocytopenia at 55,000.
- The evolution was marked by the death of the patient by the decompensation of his heart failure.

Discussion

The latest research indicates that the most infectious body fluids are nasopharyngeal, nasal, and lower respiratory tract secretions [2].

SARS-CoV-2 was detected for the first time in Japan in the cerebrospinal fluid by the trans-synaptic route after invasion of the peripheral nerve endings [5,7].

The virus has also been found in feces and a lower percentage of infection has been reported in the blood [8]. There are reports of COVID-19 in the urine of infected patients; however, the reported percentages are much lower compared to SARS and MERS [3].

There is another potential issue that has not been resolved for COVID-19 infection: "the presence of COVID-19 in pleural effusions". The presence of viral particles in pleural fluid has also not been studied earlier for previously reported SARS and MERS outbreaks [4].

The importance of such an investigation lies in the low level of viremia in the blood reported for COVID-19, which makes it possible for the virus to spread to the organs of the body, including the pleura [2,6].

Conclusion

Although pleural effusion is a rare clinical manifestation in COVID-19, because the content of pleural effusion has never been studied in patients infected with COVID-19, we here

for the first time reported the presence of SARS COV-2 in pleural effusion. We look forward to such studies.

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