International Journal of Progressive Sciences and Technologies (IJPSAT)
ISSN: 2509-0119.

http://ijpsat.ijsht-journals.org
Vol. 27 No. 2 July 2021, pp.477-481

Covid-19 And Infective Endocarditis The Challenge of the 21st Century

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Abstract – COVID-19 can cause multiple organ dysfunction including cardiovascular system that affects the course and prognosis of disease. We present a case of a 65-year-old man with a history of arterial hypertension and severe COVID-19 complicated with acute pneumonia, acute respiratory distress syndrome, septic shock, infective endocarditis, severe stenosis and regurgitation of aortic valve, spontaneous pneumothorax and acute kidney failure.

Chest CT scan included bilateral lung involvement with subtotal ground-glass opacities and consolidations. Lung injury was considered as H type. Echocardiography revealed hyperechogenic formation (5mm in size) - vegetation on the right coronary cusp of aortic valve, that caused sever stenosis and regurgitation. On the extremities Janeway lesions and Osler's nodes were revealed. He became hemodynamically unstable. Besides leukocytosis, neutrophilia with a left shift, lymphopenia, eosinopenia, moderate thrombocytopenia and deep anemia, high level of CRP, procalcitonin and severely increased D-Dimer (83600 µg/L) was notable. In spite of adequate treatment with dexamethasone, antibiotics and anticoagulants patient developed first left sided and lately right sided complete spontaneous pneumothorax and was connected to mechanical ventilation. Though after thoracentesis and air evacuation from pleural cavities both lungs were expanded patient's state remained critical. Patient developed septic shock, acute renal failure, acute cardiovascular and respiratory insufficiency and lethal outcome was defined.

Presented case shows that COVID-19 causes different kind of multiple organ dysfunction that determines the outcome of the disease. It is important to follow and monitor the patients not only in critical and sever but in mild or common cases as well.

Keywords – COVID-19, acute respiratory distress syndrome, infective endocarditis.

I. INTRODUCTION

The World Health Organization (WHO) has declared coronavirus disease (COVID-19) a global pandemic on March 11, 2020. Since then more than 192 million people were infected. COVID-19 is caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection that can progress to pneumonia, acute respiratory distress syndrome and shock. Besides COVID-19 can cause multiple organ dysfunction including cardiovascular system that affects the course and prognosis of disease. It must be noticed that cardiovascular complications (due to hypercoagulation, myocarditis, coronary artery microvascular dysfunction et cet.), can occur not only in the common, severe and critical cases of the disease but even in mild cases as well [1], [2].

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II. Case presentation

We present a case of a 65-year-old man with a history of arterial hypertension and severe COVID-19 complicated with acute pneumonia (Candida spp, Citrobacter freundii, Klebsiella pneumonia), acute respiratory distress syndrome (ARDS), septic shock, infective endocarditis, severe stenosis and regurgitation of aortic valve, spontaneous pneumothorax and acute kidney failure.

The patient became ill from 26.03. 2020. He had fever (39C°), sore throat, dry cough, chills and was hospitalized in clinic, where was diagnosed COVID-19 and treated according to guideline protocol [3], [4]. In spite of treatment, the patient developed dyspnea, tachypnea, sever desaturation and with acute respiratory failure was transferred into the intensive care unit (ICU) of the Tbilisi State Medical University First University Clinic.

On admission, temperature (T) was 38°C, blood pressure (BP) was 133/68 mm Hg, heart rate - (HR) 92 bpm, respiratory rate (R) - 26 pm and peripheral oxygen saturation (SpO_2) - 84% on oxygen 2 L/min. On auscultation crackles and diminished breathing sounds were revealed bilaterally in the lower parts of lungs. Chest CT scan included bilateral lung involvement with subtotal ground-glass opacities (GGO). In upper fields the process was multifocal, while the middle and lower fields were covered totally by opacities. Comparatively more dense infiltrates (consolidations) were observed on the background of GGO. Several areas of bronchiectasis were also revealed. Thick trabeculae were observed in lower lobes. Little amount of effusion was revealed in both pleural cavities. Pulmonary involvement (PI) score was 21 (score range 0-24).

To assess PI score, semi-quantitative scoring system was used. Both lungs were divided into 3 fields (right lung according to the lobes, in left lung lower lobe was divided in 2 fields). All 6 fields were visually reviewed for GGO and consolidation and scored from 0 to 4 based on involvement percentage (0, no involvement; 1, ≤ 25%; 2, 26-50%; 3, 51-75%; 4, ≥76%). Total PI score was calculated as sum of GGO and consolidation score of all 6 fields ranging from 0 to 24.

Transthoracic echocardiography (TTE) revealed mild diminution of systolic function (ejection fraction (EF) equaled 50%) and diastolic dysfunction due to impaired relaxation, normal size right ventricle (tricuspid annular plane systolic excursion (TAPSE) was 20), mildly dilated atriums, fibrotic mitral valve leaflets and annulus with mild regurgitation +1.5/+4, calcification of aortic valve with mild regurgitation +1/+4, moderate pulmonary artery hypertension (pulmonary artery systolic pressure PASP equaled 55mmHg).

Common blood count showed relative neutrophilia, lymphopenia, eosinopenia, moderate thrombocytopenia; Coagulation tests were in normal range. Troponin I <0.01ng/ml, (N< 0.03); D-Dimer - 1710 µg/L (N< 654); C-Reactive Protein (CRP) -254 mg/L (N< 5); Procalcitonin (PCT) < 0.5 ng/mL (N<0.5). Liver and Kidney function tests were in normal range.

Patient was started on iv infusion of antibiotics, detoxification therapy, unfractionated heparin (UFH), oral hydroxychloroquine 600 mg every 12 hours for 1 day followed by 400 mg daily for 4 additional days. Patient's state was severe, fever continued. Sputum bacteriology revealed Candida spp. antifungal (Fluconazole 200mg/100ml infusion) treatment was added. Ventilation/oxygenation indices worsened, D-Dimer, CRP, ferritin level increased. In 48 hour D-dimer increased from 1710->3780->5990->15750 -> 36500 (though being treated with Nadroparin 0,4ml bid). Patient became hemodynamically unstable. Clinical onset of severe ARDS was developed. Patient was started on iv norepinephrine and oral rivaroxaban 20mg/day. On the background of CT scan, lung compliance and V/Q data lung injury was considered as H type. The ratio of arterial oxygen partial pressure (PaO_2) and fraction of inspired oxygen (FIO_2) - PaO_2/FIO_2 <150 pointed on sever ARDS. Dexamethasone was included in treatment but with little benefic effect. Chest X-ray showed GGO and consolidations bilaterally. Bacteriology of endotracheal aspirate revealed abundant growth of Candida spp. Antimycotic therapy was continued.

On 14.04 stabilization of hemodynamic indices was achieved and inotropic support was quitted. Mechanical ventilation parameters also improved comparatively, but ceasing of medical sedation was not possible. Patient was awaking with anxious, tachypnea, desynchronization and desaturation. Besides sedatives, increase of the parameters of mechanical ventilation was needed. As continuing artificial ventilation was necessary on 16.04 tracheostomy was performed. On 17.04 patient's state worsened, fever, leukocytosis with a left shift, increase of CRP (284 mg/L), PCT (2 ng/mL) and D-dimer (83600 µg/L) was observed. For the hemodynamic stabilization again inotropes were used. TTE revealed hyperechogenic formation (5mm) on the right coronary cusp (RCC) of aortic valve, supposedly, vegetation (Fig 1), that caused sever stenosis (mean pressure gradient (Vmean) equaled 58 mmHg) and regurgitation (+3.5/+4). Mitral valve leaflets and annulus were fibrotic and mild regurgitation (+1.5/+4) was observed; PASP was increased up to 58 mmHg. On the extremities Janeway lesions and Osler's nodes were revealed.

On the 25th day from the onset of COVID-19 patient was diagnosed sepsis, septic shock, and infective endocarditis with severe aortic stenosis and regurgitation according to modified Ducke criteria. Sputum microbiology revealed again Candida spp. while blood culture was sterile. Antibiotic treatment was changed by Meropenem and Vancomycin combination, and antifungal treatment was going on as well. Fever was reduced, but patient's state still remained sever needing mechanical ventilation (MV) on high parameters.

On 28.04 patient developed complete left sided spontaneous pneumothorax and thoracentesis was performed. Sputum bacteriology revealed *Citrobacter freundii* and in addition Colomycin was included in treatment. Though repeated real PCR on SARS-COV-2 was negative and serological tests on Anti-SARS-COV-2 IgG and Anti-SARS-COV-2 IgM were positive, patient's state still remained severe because of COVID-19 induced multi-organ dysfunction.

Periodically tachycardia, tachypnea, desynchronization with respirator was observed. Patient needed deeper sedation, relaxation. MV was going on by tracheostomy tube according to ABG data: Peak inspiratory pressure (PIP) - 22 cmH_2O, pressure support ventilation (PSV) - 12 cmH_2O; positive end-expiratory pressure (PEEP)- 7 cmH_2O; frequency - 20; Fraction of inspired oxygen (FiO_2) – 50% (0.5); tidal volume (VT) - 560 ml. SpO_2 - 97%-98%;

Microbiology of bronchoalveolar lavage revealed *Klebsiella Pneumoniae* sensitive to Colomycin. Air evacuation was going on from pleural drainage. On X ray lungs were expended, though infiltrates were observed on both sides. TTE showed no changes in dynamic. Norepinephrin was infused to maintain hemodynamic stability, feeding was provided by nasogastric tube. Patient had sub febrile temperature, diuresis was adequate. Lymphopenia, leukocytosis with a left shift up to metamyelocytes was observed in blood count. Because of deep anemia (Hb-74g/L, Erytrocytes – 2.74 $\times 10^{12}$/L) patient was transfused red blood cell (RBC) mass. Blood urea nitrogen (BUN), creatinine (Cr) and coagulation tests were in normal range. CRP and D-dimer level diminished, though remained high (CRP-172mg/L; D-dimer 13000 µg/L).

On 12.05. patient developed complete right sided spontaneous pneumothorax, confirmed by X ray. By right sided thoracentesis drainage tube was left in pleural cavity for air evacuation. Considerable amount of air was evacuated from the left pleura as well. In dynamic radioscopy showed that both lungs were expanded, but both lungs were covered totally by nonhomogeneous opacity due to GGO and consolidations. MV was continued via tracheostomy tube with parameters corrected according to ABG data maintaining SpO2 up to 96-98%, but periodically the patient was hyperventilated disturbing respiratory indices. For hemodynamic stabilization vasopressors were used, high fever was reduced by antipyretics and mechanical methods. Antibiotic treatment was held according to the bacteriology of bronchoalveolar lavage. Neurological state was also severe. The patient was still prone to drowsiness, he opened eyes in response to loud sound and pain, fixated a look, had round and moderately narrow pupils and weak motor activity of extremities without side preferences. According to TTE EF was not diminished, vegetation on the right coronary cusp was smaller (4mm), but caused sever aortic stenosis and moderate/severe regurgitation. Tricuspid valve regurgitation and PASP were comparatively increased. Still patient's state remained critical with expressed features of septic shock.

Repeated CT showed disease progress in lungs, PI score became 23 (0-24). Due to severe general condition bilateral thoracentesis and pleural drainage was considered as maximal surgical intervention and additional large-scale surgical activity had been denied.

On 20.05. microbiological investigation of bronchoalveolar lavage revealed growth of *Klebsiella pneumoniae* (10^8 CFU/ml), susceptible only to colistin. Examination by acid-fast stain and GeneXpert MTB/RIF test ruled out tuberculosis infection. Therefore colomycin was included in antibiotic treatment according to creatinine clearance. In dynamic creatinine level, CRP and PCT indices increased, patient developed acute renal failure.

Patient was consulted by nephrologist and thoracic surgeon. On 23.05 tracheostomy tube was withdrawn under general anesthesia. Observation of trachea and bronchial tree was performed. Vocal folds closure was incomplete, saliva drained into trachea by small portions, under vocal folds trachea was hyperemic, there were purulent deposits on the tracheal wall. At the site of the stoma there was a fold of mucus, also purulent. There were also obvious signs of wall damage. Excess purulent contents were observed in both main bronchi and distal bronchi. Observation in dynamic was necessary. The armored tube was inserted in trachea and held distally, the cuff was pressed at low pressure. Sanitation of tracheobronchial tree with physiological saline solution was performed. Significant amounts of air were again evacuated from both pleural cavities with purulent impurities, with more excess on the left. Left pleural drainage was changed.

High leukocytosis (24.44×10^9/L), neutrophilic granulocytosis (96.5%) with a left shift, (band neutrophils - 12%); deep anemia (Hb – 80g/L, Eryt. 2.9×10^{12}/L), lymphopenia (Lym. – 2.3%), CRP – 158 mg/L was notable among laboratory investigations. Fever was continuing. Purulent, odorous discharge from compromised areas - endobronchial space, tracheostomy wound area, drainage - remained noticeable.

In this situation, the probability of spontaneous closure of the endobronchial-lung tissue defect was almost non-existent. Surgical intervention for the liquidation of bilateral pneumothorax could not increase survival as well. According to the Hb level end erythrocyte count periodically patient was transfused RBC mass.

On 29.05. percutaneous gastrostomy was performed under endoscopic control. Feeding was continued through a gastrostomy. At all stages of treatment patient's state remained critical mainly due to complications that developed on the background of COVID-19 and was caused by this infection. Underlying chronic diseases and low reserve potential was a high risk for progression of multiple organ failure and irreversible decompensation of vital functions, hence the lethal outcome. Septic shock accompanying with cardiovascular and respiratory failure was progressing and on 14.06. cardiac arrest was stated. Attempt of cardiopulmonary resuscitation was ineffective.

Presented case once again shows that COVID-19 is a multiple system disease causing deterioration and dysfunction of multiple organs. That's why SARS-CoV-2 is called a syndrome of multiple organ dysfunction [5], [6].

III. Discussion and Conclusion

Signs of multiple organ damage, typical for sepsis, occur in approximately 2-5% of cases on the 8th to 10th day after the onset of COVID-19. On the other hand, infectious endocarditis is a rare, severe, and life-threatening disease that accounts for 30% of deaths (among patients with infectious endocarditis) per year in common [7], [8], [9]. In the presented case sepsis was complicated by infective endocarditis.

ARDS development risk increases with age (> 65), by the presence of comorbid diseases (hypertension, diabetes), neutrophilia, lymphocytopenia, increased lactate dehydrogenase, CRP, D-dimer. Mortality is high (52-53%) in patients with COVID-19 accompanied with ARDS [10], [11].

The cause of renal failure during COVID-19 is also multifactorial; Acute renal failure is a severe complication of COVID-19 with a high risk of mortality [12]. In hospitalized COVID-19 patients acute renal failure is found in 40% of cases, 20% of these patients require acute hemodialysis [13].

Abovementioned case once more underlines the possibility of the development of various organ dysfunction in patients with COVID-19 and the necessity to check not only lung function, but coagulation tests, cardiovascular and other organ systems as well for timely diagnosis and treatment of complications in case they exist. It is also important to follow and monitor the patients not only in sever and critical but in mild or common cases as well.

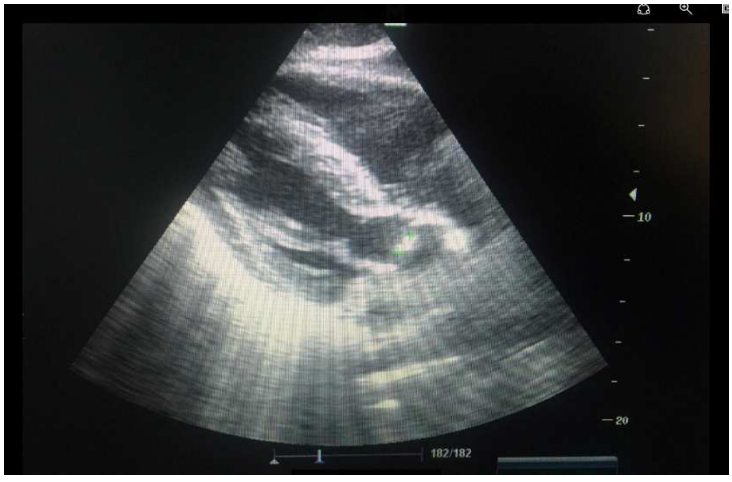

Figure 1. TTE, subcostal view. Aortic valve calcification, hyperechogenic formation (5mm) - vegetation on the right coronary cusp (RCC).

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