ACUTE COVID-19 CARDIOVASCULAR SYNDROME: A CASE REPORT

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ACUTE COVID-19 CARDIOVASCULAR SYNDROME: A CASE REPORT

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ABSTRACT

A man 44 years old with metabolic syndrome and chronic kidney disease was presenting acute COVID-19 cardiovascular syndrome. The condition was aggravated by presence of ureterolithiasis and gout. After treatment, hemodialysis and ureteroscopic lithotripsy-double J ureteral stent, the patient was recovered from his condition.

INTRODUCTION

COVID-19 is known for its magical infectivity, it could be present as severe respiratory illness as well as acute COVID-19 cardiovascular syndrome i.e. acute coronary syndrome (STEMI or NSTEMI), acute myocardial injury without obstructive CAD, arrhythmias, heart failure with or no cardiogenic shock, pericardial effusion, thromboembolic complications.[1-5]

Patients with metabolic disorders like obesity, diabetes, cardiovascular and liver disease may face a higher risk of infection of COVID-19, greatly affecting the progression and prognosis of the disease, being associated with significantly worse outcome in these patients. The proposed drugs that are used for COVID-19 treatment must be carefully considered for clinical use, especially in patients with metabolic syndrome.[6,7]

CASE REPORT

A 44 year old man was referred from Dr Soetrasno hospital, Rembang to the Dr Kariadi hospital, Semarang because of confirmed COVID-19, STEMI inferior, azotemia, diabetes mellitus. Ten days before admission to Dr Kariadi hospital, patient had left chest pain continuously, he felt uncomfortable, chest tightness on exertion, he couldn't point to the pain, the pain didn't radiate to the neck, shoulder, back or down his arm, skipping of the heartbeat sometimes. He also felt feverish, no shivering, no reddish spot on skin, no yellow eye, no nosebleed, no bleeding gum, the urine colour was normal, no diarrhea. He also had shortness of breath which was disappear and arise, no effect on shortness of breath with changing position, no episodes of sudden shortness of breath that awaken patient from sleep. No weight loss, no night sweat, no seizure, no hair loss, no swelling, no redness on cheek, no sniffles, no odynophagia, no vomitus, there were fatique, cough sometimes, nausea, pain on his feet especially left feet, pin and needle sensation on his hand and feet, continuously, not relieved by rest or activity.

Patient had history of hypertension and diabetes mellitus for 10 years, smoking for 10 years 1,5 pack/day, kidney stone 7 years ago (the urine was redness), uric acid disease for 5 years, took pain killer drugs for 5 years to relieve pain (NSAIDs, steroid), cholesterol disease for 3 years. His father and mother had diabetes mellitus, his father had uric acid disease. The patient and his wife are self-employed as bread maker, living with two children. His wife and children didn't develop respiratory symptoms or chest pain.

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Patient had weight 82 kg, height 172 centimeters, with body-mass index 27.7 kg/m2 (obesity), abdominal circumference 109 cm. On 15th September 2020, 10 days from onset, his blood pressure 200/110 mmHg, heart rate 98 beats per minute, respiratory rate 36 breaths per minute, body temperature 37°C and SO2 98% NRM. He had shortness of breath, compos mentis. Examination on the neck showed distension jugular venous pressure R+3 cm. Heart examination were apex of the heart shift to the laterocaudal, no murmur, no pericardial friction rub, no muffled heart sound. Examination on the lung showed dullness on the bottom right lung, vesicular breath sound and rales in both bottom lung field. There was pitting edema of the dorsum of the feet, tophi on his feet, paresthesia on his hand and feet, flat feet.

On 9th September 2020, 4 days from onset, leucocytes 19,000/μL shift to the left neutrophil 85.4% lymphocyte 8.6% ratio 9.9, glucose 144 mg/dL, ureum 113 mg/dL creatinine 7.0 mg/dL potassium 5.3 mmol/L, SGOT 215 U/L SGPT 61 U/L, HbsAg non reactive antiHIV non reactive, troponin I positive, total cholesterol 138 mg/dL HDL 24 mg/dL LDL 56 mg/dL triglyceride 265 mg/dL. Urine analysis showed protein (++), reduction normal, ketone negative. BGA showed pH(T) 7.404 PCO2(T) 28.6 mmHg PO2(T) 156 mmHg HCO3-18.0 mmol/L BEecf -7.1 mmol/L SO2c 99% A-aDO2 382 mmHg RI 2.3, metabolic acidosis mixed respiratory alkalosis, normal anion gap, V/Q mismatch. SARS-CoV-2 IgM antibody and SARS-CoV-2 IgG antibody were non reactive.

On 10th September 2020, 5 days from onset, ECG showed STEMI inferior. There were ST elevation at II, III, aVF, Q wave at III, aVF, biphasic T wave at V_2, reciprocal changes at I, aVL (Figure 1).

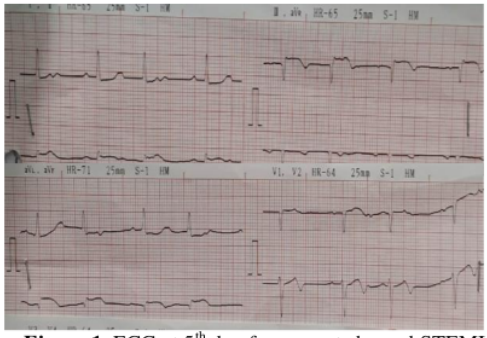

Figure 1. ECG at 5th day from onset showed STEMI inferior

On 11th September 2020, 6 days from onset, D-dimer 4,727.3 ng/mL, nasopharyngeal/ oropharyngeal swab for SARS-CoV-2 positive. On 15th September 2020, 10 days from onset, Hb 10.4 g/dL MCV 92 fL leucocytes 10,500/μL thrombocytes 341,000/μL shift to the left neutrophil 72% lymphocyte 18% ratio 4.0 prothrombin time 16.6 second/15.6 second partial thromboplastin time 28.7 second/29.5 second, D-dimer 19,720 ng/mL fibrinogen 774 mg/dL, glucose 124 mg/dL HbA1c 7.8%, ureum 210 mg/dL creatinine 6.2 mg/dL potassium 4.3 mmol/L, SGOT 17 U/L SGPT 20 U/L albumin 2.8 g/dL, CKMB 30 U/L troponin 26.984 ng/mL, procalcitonin 5.1 ng/mL. Urine analysis showed protein 30 mg/dL, reduction 250 mg/dL, ketone negative, leucocytes 45/μL (pyuria), bacteria (++).

On 16th September 2020, 11 days from onset, blood culture was sterile, nasopharyngeal/ oropharyngeal swab for SARS-CoV-2 was negative. On 17th September 2020, 12 days from onset, troponin 11.437 ng/mL, urine culture showed urinary tract infection E.cloacae > 100.000 cfu/ml urine, nasopharyngeal/ oropharyngeal swab for SARS-CoV-2 was negative. ECG showed sinus rhythm, Q wave at III, aVF, poor R wave progression. Chest radiograph showed pulmonary consolidation with air bronchogram in the right lung field, left perihilar, right pleural effusion, cardiomegaly and pulmonary edema (Figure 2).

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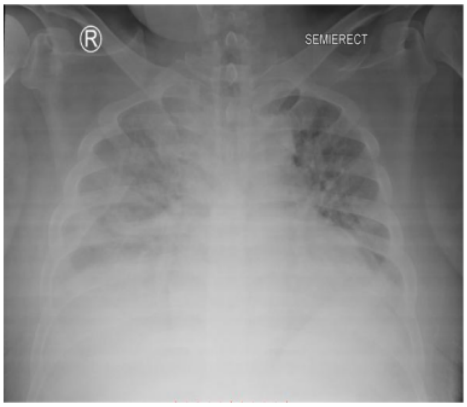

Figure 2. Chest radiograph at 11th day from onset showed pulmonary consolidation, cardiomegaly and pulmonary edema

On 8th September-14th September 2020 at Dr Soetrasno hospital Rembang, patient was treated with intravenous ceftriaxone 2 gram once daily, azithromycin 500 mg once daily, acetylcysteine 5 gram once daily, fast-acting insulin 4 IU subcutaneous injection three times daily, lopinavir 100 mg ritonavir 25 mg combination 2 tablet twice daily, ISDN 5 mg three times daily, atorvastatin 40 mg once daily. At Dr Kariadi hospital, patient was treated with intravenous azithromycin 500 mg once daily, vitamin C 1 gram three times daily, acetylcysteine 5 gram three times daily, nicardipine 3 mg/hour, fast-acting insulin (max dose 2 IU/hour), heparin 1000 IU/ hour, lopinavir 100 mg ritonavir 25 mg combination 2 tablet twice daily, zinc 20 mg twice daily, sodium bicarbonate 500 mg three times daily, folic acid 1 mg once daily, furosemide 40 mg once daily, ramipril 5 mg once daily.

On 22th September 2020, 17 days from onset, Hb 7.8 g/dL MCV 97 fL reticulocyte 1.4% transferrin saturation 4.0% according to iron deficiency anemia (anemia on chronic disease), ureum 133 mg/dL creatinine 4.4 mg/dL uric acid 11.2 mg/dL potassium 5.5 mmol/L, antiHCV negative. Echocardiography showed dilation left atrium, left ventricle, right atrium, eccentric LVH, LVEF 46.5%, MR (+) moderate, TR mild-moderate, low probability for PH, RWMA – IHD. Abdominal ultrasound showed congestive liver, severe left hydronephrosis and hydroureter, increase echogenicity cortex of the right kidney, no fatty liver. Abdominal radiograph showed no urinary lithiasis. The patient was treated with fast-acting insulin 6 IU subcutaneous three times daily, long-acting insulin 14 IU subcutaneous daily, ramipril 10 mg once daily, amlodipine 10 mg once daily, bisoprolol 2,5 mg once daily, furosemide 20 mg once daily, nitroglycerine 2,5 mg once daily, sodium bicarbonate 500 mg three times daily, folic acid 1 mg once daily, ferrous sulfate 300 mg once daily, atorvastatin 20 mg once daily, allopurinol 100 mg once daily, colchicine 0,5 mg once daily.

On 26th September 2020, 21 days from onset, he felt shortness of breath, uncomfortable on his chest like heaviness, no cold sweating since yesterday evening. His blood pressure 95/60 mmHg, heart rate 50 beats per minute irregular, respiratory rate 28 breaths per minute, body temperature 36.2°C and SO_2 96% nasal cannula. Laboratorium results showed ureum 163 mg/dL creatinine 6.2 mg/dL (GFR 10 ml/min/1.73 m^2), natrium 126 mmol/L potassium 6.1 mmol/L chloride 92 mmol/L, troponin 0.9 ng/mL, D-dimer 4,380 ng/mL fibrinogen 735 mg/dL, procalcitonin 0.5 ng/mL, NT-pro BNP 18,898 pg/mL. ECG showed sinus bradycardia, Q wave at III, aVF and poor R wave progression, no S_I Q_{III} T_{III} pattern, no RAD, no RBBB. Chest radiograph showed pulmonary consolidation with air bronchogram in the right lung field, left perihilar, right pleural effusion (reduced), cardiomegaly and pulmonary edema. Abdominal CT Scan showed severe left hydronephrosis and hydroureter e.c. left ureterolithiasis approximately 2.6 x 1.3 cm (Figure 3). Patient was treated with intravenous atropine sulfate 0.5 mg, acetylsalicylic acid 80 mg once daily, salbutamol 2 mg twice daily, 10 IU fast-acting insulin in 40% dextrose 50 cc, Ca polystyrene sulfonate 5 g three times daily, 1 packed red cell transfusion. Ramipril, amlodipine, furosemide, nitroglycerine and bisoprolol were stopped.

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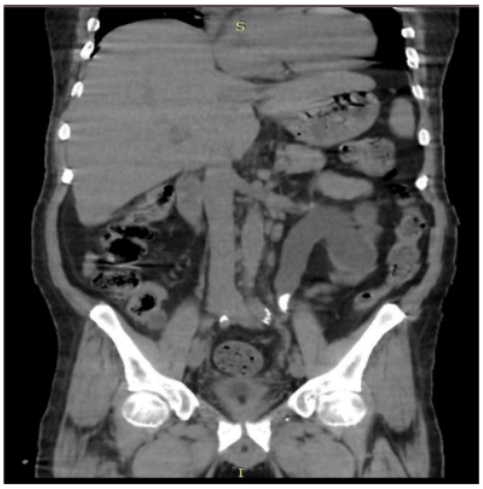

Figure 3. Abdominal CT Scan showed severe left hydronephrosis and hydroureter e.c. ureterolithiasis

On 29th September 2020, patient was restless, the communication was disgress even not always, in 1-2 days. He was somnolence. His blood pressure 110/60 mmHg, heart rate 67 beats per minute irregular, respiratory rate 28 breaths per minute, body temperature 36.5°C and SO2 97% nasal cannula. Urine output 0.6 cc/kgbodyweight/hour. Examination on the lung showed wheezing which is disappear and arise. Labs showed Hb 7.8 g/dL, ureum 180 mg/dL creatinine 6.7 mg/dL (GFR 9 ml/min/1.73 m2), natrium 123 mmol/L potassium 5.8 mmol/L chloride 92 mmol/L, procalcitonin 0.2 ng/mL. Chest radiograph showed pulmonary edema.

The patient was treated with NaCl 3% 250 ml, getting initiation of hemodialysis for 2 hours, UFG 1,500 mL UFR 750 mL/hour Qb 150 mL/min with 1 packed red cells transfusion. Two days later, the patient was getting hemodialysis for 4 hours, UFG 2000 mL UFR 500 mL/hour Qb 150 mL/min with 2 packed red cells transfusion. The patient was getting better day by day, on 2nd November 2020, underwent ureteroscopic lithotripsy and double J ureteral stent for his left ureterolithiasis. The patient underwent outpatient care. The patient was consulted to the rheumatology and planned given charchoat foot shoes. The left foot radiograph showed no lytic, no sclerotic, no destruction distal tibia fibula bone and pedis bone, no dislocation, no narrowing ankle, metatarsal, metatarsophalangeal joint.

DISCUSSION

COVID-19 is known for its magical infectivity, fast transmission and high death toll based on the large number of infected people. The vast majority of patients in the ongoing coronavirus disease 2019 (COVID-19) pandemic primarily present with severe respiratory illness, but it also could present as acute COVID-19 cardiovascular syndrome. The spectrum of acute COVID-19 cardiovascular syndrome are acute coronary syndrome (STEMI or NSTEMI), acute myocardial injury without obstructive CAD, arrhythmias, heart failure with or no cardiogenic shock, pericardial effusion, thromboembolic complications. Mechanisms for acute COVID-19 cardiovascular syndrome occurrence are hypoxemia, direct injury of SARS CoV-2 to cardiomyocytes, infection of pericardium, stress- induced cardiomyopathy, cytokine storm, microvascular/thrombotic injury as well as pre-existing cardiovascular disease.[1-5]

Patient was a man 44 years old with cardiovascular risk factors i.e. metabolic syndrome, smoking, hyperuricemia. Patient had COVID-19 infection, in stage III systemic hyperinflammation (D-dimer 19,720 ng/mL, troponin 26.984 ng/mL, NT-pro BNP 18, 898 pg/mL), which present as acute COVID-19 cardiovascular syndrome i.e. STEMI (differential diagnosis myocarditis), which is supported by history of left chest pain, troponin between 10-100 ng/mL, ECG ST elevation at II III aVF Q wave at III aVF and reciprocal changes at I, aVL, echocardiography showed RWMA.

Patients with metabolic disorders like obesity, diabetes, cardiovascular and liver disease may face a higher risk of infection of COVID-19, greatly affecting the progression and prognosis of the disease, being associated with significantly worse outcome in these patients. The proposed drugs that are used for COVID-19 treatment must be carefully considered for clinical use, especially in patients with metabolic syndrome like favipiravir with adverse

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effect increased of serum uric acid, remdesivir with adverse effect anemia, acute kidney injury, increased blood creatinine.[6,7] High troponin could describe acute coronary syndrome, myocarditis, hyperinflammation phase of COVID-19 infection. NT-proBNP as biomarkers of hemodynamic myocardial stress and HF are frequently elevated among patients with severe inflammatory/respiratory illnesses, which could be seen as the combination of the presence/extent of pre-existing cardiac disease and or the acute hemodynamic stress related to COVID-19.[4,5,8-10]

Patient had chronic kidney disease, radiolucent ureterolithiasis, hyperuricemia and gout. Chronic kidney disease could be attributed by metabolic syndrome, hyperuricemia and obstructive uropathy from ureterolithiasis. Radiolucent urolithiasis i.e. uric acid stone, ammonium urate stone, xanthine stone, 2,8 dihydroxyadenine stone, drug stone.[11,12] In this patient, the most possible radiolucent ureterolithiasis is uric acid stone. Chronic kidney disease and coronary artery disease are two entities share both common etiologies and risk factors. Advanced CKD patients have both increased risks of bleeding and thrombosis. CAD management is complicated in CKD patients, due to comorbid conditions and potential side effects during interventions. Decisions on reperfusion in patients chronic kidney disease with STEMI have to be made before any assessment of renal function is available, it is important to estimate the GFR as soon as possible. The type, dose of antithrombotic agent and the amount of contrast agent should be considered based on renal function. Acute coronary syndrome patients with chronic kidney disease (CKD) receive frequently excess dosing with antithrombotics, contributing to the increased bleeding risk.[13-15] The patient had recent STEMI (> 48 hours) with chronic kidney disease stage 5, no symptoms suggestive of ischaemia, hemodynamic instability, or life threatening arrhythmias at Dr Kariadi hospital. With considering benefit and risk, coronary angiography wasn't conducted.

Viral infections are a known cause of acute arthralgia and arthritis. Oligoarticular or polyarticular involvement (either symmetric or asymmetric), good response to NSAIDs, a clinical manifestation characterised by an early onset (within the first weeks of symptomatic infection) and a self-limiting presence are the elements that orientate toward a viral arthritis. Arthralgia is one of the symptoms that occurs in patients with COVID-19 and is present in 14.9% of cases. Reactive arthritis associated with COVID-19 had been reported.[16,17] The patient had pain on his feet, which it is occured before this illness and the pain wasn't disappear until now. This condition described the pain was caused by diabetic neuropathy and gout.

Charcot foot is a condition affecting the bones, joints, and soft tissues of the foot and ankle, characterized by inflammation in the earliest phase. The interaction of several component factors (diabetes, sensory-motor neuropathy, autonomic neuropathy, trauma, and metabolic abnormalities of bone) results in an acute localized inflammatory condition that may lead to varying degrees and patterns of bone destruction, subluxation, dislocation, and deformity. X-rays may be normal or show subtle fractures and dislocations or later show more overt fractures and subluxations. In later stages, the calcaneal inclination angle is reduced and the talo-first metatarsal angle is broken. Medial calcification of the arteries is present in most charcot feet and is a frequent secondary finding on radiographs. However, radiographic changes of charcot feet are typically delayed and have low sensitivity. Magnetic resonance imaging allows detection of subtle changes in the early stages of active charcot feet when X-rays could still be normal.[18] Patient has risk factors for developing charcot feet, even the X-rays normal, but the patient had charcot feet clinically.

CONCLUSION

COVID-19 is known for its magical infectivity, it could be present as acute COVID-19 cardiovascular syndrome i.e. acute coronary syndrome (STEMI or NSTEMI), acute myocardial injury without obstructive CAD, arrhythmias, heart failure with or no cardiogenic shock, pericardial effusion, thromboembolic complications. Mechanisms for acute COVID-19 cardiovascular syndrome occurrence are hypoxemia, direct injury of SARS CoV-2 to cardiomyocytes, infection of pericardium, stress- induced cardiomyopathy,

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cytokine storm, microvascular/thrombotic injury as well as pre-existing cardiovascular disease. Patients with metabolic syndrome may face a higher risk for developing acute COVID-19 cardiovascular syndrome, greatly affecting the progression, prognosis of the disease as well as the therapy.

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