A Fatal Case of Rhabdomyolysis in a Sri Lankan Patient with COVID-19 Pneumonia

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Abstract

The complications of Corona Virus Disease – 2019 (COVID-19) are multiple and virtually can affect most of the organ systems in human. The renal injury in COVID-19 is a topic for current discussions and rhabdomyolysis is considered as a possible aetiopathogenesis, though the information is minimal. Here we present a Sri Lankan male patient, who developed fatal rhabdomyolyisis during his recovery phase of COVID-19 pneumonia.

Keywords: *Rhabdomyolysis, COVID-19 Pneumonia*

Introduction

The pandemic of COVID-19 has been the mostly discussed health topic in this era and it has numerous consequences toward the health of the patient. The spectrum of clinical features can be varied from asymptomatic to fetal. To date, pulmonary and thrombo-embolic complications are recognized as the leading causes of death in COVID-19. Acute kidney injury (AKI) has been a topic of focus and rhabdomyolysis could be contributory. Global and local literature in rhabdomyolysis of COVID-19 are few in number.

Case Presentation

A 64 year old Sri Lankan male was diagnosed with COVID-19 pneumonia according to the RT- PCR (real time polymerase chain reaction) test performed, following his presentation with shortness of breath, myalgia and fever to a local hospital, which lasted for 3 days. His comorbidities were well controlled type-2 diabetes mellitus and ischaemic heart diseases for which he had undergone coronary artery bypass surgery 3 years ago and he was taking metformin, gliclazide, atorvastatin, aspirin, carvedilol and enalapril. Initial clinical evaluation was not suggestive of other differential diagnosis for his complaint such as cardiac failure, pulmonary embolism, pericardial effusion or pneumothorax.

On admission, he was conscious and dypnoeic with a respiratory rate of 40 breathes per minute. He was tachycardic, normotensive and hypoxic with an oxygen saturation of 85% on room air. Following the initial correction of hypoxemia with face mask oxygen, he was transferred to COVID-19 intensive care unit in our institution. He demonstrated hypoxia and was started on high flow nasal oxygen with 40 L/minute with a FiO_2 of 75%. His blood pressure was maintained without inotropes though there was a persistent tachycardia.

Full blood count showed lymphopenia with normal other cellular parameters. Electro-cardiogram showed only sinus tachycardia. On admission, baseline liver function tests and renal function tests were within normal limits while his CRP (C-reactive protein) was 132 mg/l and procalcitonin was 0.66 ng/ml. Troponin I, D-Dimer and ferritin levels were elevated as 1.2 ng/ml, 1200 ng/ml and 1400 µg/l respectively. His Brain Natriuretic Peptide (BNP) and baseline Creatinine Phosphokinase (CPK) levels were within normal limits. The chest X-ray showed inflammatory shadows involving bilateral lower lobes. (Figure 1)

The routine nursing care was continued along with intravenous meropenem, oral levofloxacin, intravenous dexamethasone 6 mg daily and subcutaneous enoxaparin 40 mg twice daily. Aspirin, enalapril and carvedilol were continued while withholding atorvastatin due to mild elevation of transaminases and presumed risk of acute hepatitis. Glycaemic control was achieved with soluble insulin.

Over a period of one week, he had signs of improvement with reduced oxygen demand. After 8th day, the renal functions were started to deteriorate gradually, maximum serum creatinine of 6.8 mg/dl reached on 11th day, with reduced urine output. Moreover, he complained of significant myalgia. Liver functions, inflammatory markers including CRP and procalcitonin were maintained within normal limits except moderate elevation of Aspartate Aminotransferase (AST – 143 U/L). Serum potassium and CPK were elevated to a level of 5.6 mmol/l and 1400 U/l respectively, with positive urine myoglobin. Urinary full report was acellular and did not show proteinuria. Sonography of abdomen and pelvis did not reveal significant anatomical abnormalities.

The diagnosis of rhabdomyolysis was made and liaised with nephrology team for the management. On 12th day, he was electively intubated and underwent haemodialysis due to worsening of AKI and metabolic acidosis. Four hours after the haemodialysis, sudden cardiac arrest was witnessed and appropriate management was carried out. However, it was not successful leading to death. Pathological post-mortem was not done due to non-consenting.

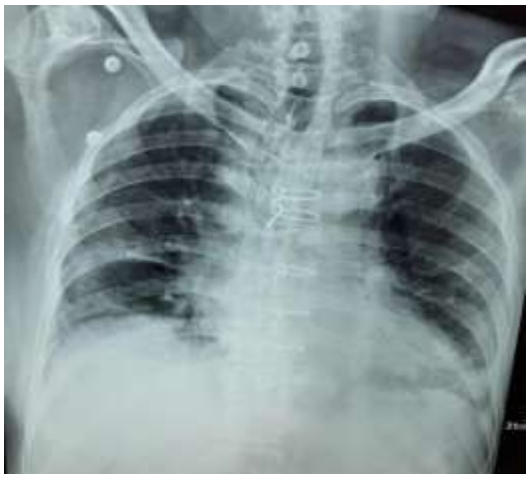

Figure 1: Chest X-ray shows evidence of bilateral lower zone predominant inflammatory shadows and evidence of bypass cardiac surgery

Discussion

Rhabdomyolysis is the breakage of skeletal muscle myocytes and release of intracellular contents [1]. This will lead to further consequences including hyperkalaemia, AKI, metabolic acidosis, disseminated intravascular coagulopathy, arrhythmia and cardiac arrest [2]. Common causes for rhabdomyolysis are trauma, alcohol, medications, seizures, neuroleptic malignant syndrome, viral infections and auto immune diseases [1,2]. The pathophysiology of AKI in rhabdomyolysis is due to the precipitation of myoglobin in the glomeruli when the protein binding capacity is exceeded due to the increased release [2,3]

The COVID-19 is a corona viral infection and there is a tendency to cause rhabdomyolysis as the primary aetiology. In addition, the drugs which used to manage COVID-19 earlier like hydroxychloroquine and oseltamivir can cause rhabdomyolysis [2,4]. The atorvastatin is a well- known to cause rhabdomyolysis however, it was omitted in our patient.

Rhabdomyolysis due to COVID-19 has been reported in scientific literature as early or late complication (4,5). The possible mechanisms were proposed in relation to direct viral invasion, autoimmune process and cytokine induced muscle damage [5,6]. There is no local published data of COVID-19 induced rhabdomyolysis.

Conclusion

This case would illustrate the importance of knowing the occurrence of rhabdomyolysis in COVID-19. Vigilant monitoring and early interventions would improve the outcome. A higher level of clinical

suspicion of rhabdomyolysis in COVID-19 patients, especially, when there is an AKI or related complications would improve the survival. However, more information from future studies on aetiology and the management should fill the gaps. We suggest to do CPK levels and urine myoglobin as initial investigations in appropriate clinical setting.

Consent

Written informed consent was obtained from the patient relatives for publication of this case report and accompanying images. A copy of the written consent is available for review with the corresponding author.

Authors' contributions

HB drafted the manuscript. DM supervised and involve the management and manuscript, SD and PW involve the management.

Author's information

HB is senior registrars in respiratory medicine. DM (MD, FRCP) is a senior consultant respiratory physician.PW and SD Resident respiratory physician,

Conflicts of interest:

All the authors have declared that they have no conflicts of interest.

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