

Fat Embolism Syndrome or COVID-19 Pneumonia: A Diagnostic Dilemma

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The novel coronavirus, named SARS-CoV-2 has caused a global pandemic state which affects the respiratory system of patients. Given the current situation, any acute respiratory distress has to be evaluated thoroughly to arrive at a definitive diagnosis for targeted management, which is challenging. We report a patient who presented with respiratory distress with fat embolism syndrome that was suspected as COVID-19 pneumonia.

Key words: SARS-CoV-2, fat embolism syndrome, acute respiratory distress

Introduction

The novel coronavirus, named SARS-CoV-2 is a viral pneumonia which may lead to respiratory distress syndrome. It may be challenging to differentiate non-COVID 19 respiratory distress from COVID 19 pneumonia which may result in under diagnosis of other health conditions during COVID-19 pandemic. Fat embolism syndrome (FES) is a clinical condition that may manifest as severe respiratory distress following traumatic bone injuries. This case discusses of a patient with FES who was co infected with SARS-CoV-2 virus.

Case history

A 19-year-old previously healthy 80kg male presented to a base hospital following a road traffic accident. The primary survey revealed no life-threatening injuries. His GCS was 15/15. He had sustained an open hairline non-displaced fracture of right tibia. No chest injuries or other significant injuries were noted. A plaster cast was applied, and the limb was immobilized. He was transferred to a

tertiary care center the same day for orthopaedic management.

Next day, he developed shortness of breath and acute confusion. The respiratory rate (RR) was ranging from 30 to 60/min. The saturation on air was 73%. There was no haemodynamic instability, but had sinus tachycardia with a heart rate of 100-120 beats/min.

There was no evidence of pulmonary oedema. He had fever spikes but were less than 100⁰ C.

The investigations revealed a drop in platelet count, CRP of 253 mg/dl and fat globules in urine. (Table 1) The Chest X-ray showed mild bilateral diffuse alveolar involvement (Figure 1). Considering the supportive clinical evidence, a tentative diagnosis of fat embolism syndrome was made.

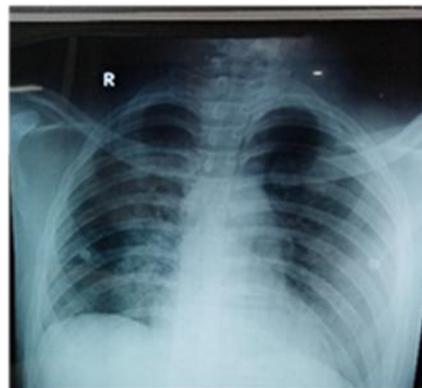


Figure 1. Chest X-ray on admission

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He was admitted to intensive care unit (ICU) and started on respiratory supports with high flow nasal oxygenation (HFNO) which required titration of FiO₂ to 90% and flow rate of 60L/min to maintain oxygen saturation of more than 94%. He was

transferred to an ICU in a multi-disciplinary COVID-19 management center.

On admission to ICU he was conscious and rational. His respiratory rate was ranging around

Table 1: Summary of investigations from admission to discharge

Date	4/4/2021	6/4/2021	7/4/2021	9/4/2021	10/10/2021
WBC	6.2*10,000	6.7*10,000	7.9*10,000		6.07*10,000
Hb g/dl	14.6	13.6	12.6		11.0
Platelet count	115*10,000	135*10,000	174*10,000		215*10,000
PCV%	40	37	35		33
International normalized ratio (INR)	1.24				1.2
Activated partial-thromboplastin time APTT (sec)	21				35
Sodium (mmol/l)	137	137	139		138
Potassium (mmol/l)	4.0	4	4.8		5.0
SGOT u/l	22	31	27		24
SGPT u/l	43	37	34		31
CPK u/l	266				
S Cr mg/dl	1.04	1.14	1.15		1.14
CRP mg/dl	253	176			46
CBS mg/dl	92				102
FiO ₂		0.9	0.6	0.4	0.35
PH		7.4	7.4	7.46	7.45
PaO ₂ mmHg		136	81	106	120
PaCO ₂ mmHg		32	38	39	42
HCO ₃ ⁻ mmol/L		21	26	28	27
BE		-3	2	4	2.5
PaO ₂ /FiO ₂		136	135	265	342

started on ticarcillin clavulanic acid and clindamycin intravenously for the open fracture. Reverse transcription polymerase chain reaction (RT-PCR) on nasopharyngeal swab was sent with the desaturation and it came as positive for SARS-CoV-2. Following this report, the patient was

60/min but had an oxygen saturation of 100% on non-rebreathing mask with 15L/min on admission to ICU. There was sinus tachycardia ranging between 100-120/min. The blood pressure was stable. Pain score on Numerical Rating Scale (NRS) was 2/10.

There was a documented temperature of 100°C on admission. There were no petechial rashes. The urine output was satisfactory. He also had a HRCT of lungs (Figure 2).



Figure 2. HRCT of Lungs

Tentative diagnoses for this patient were fat embolism syndrome, COVID-19 pneumonia or presence of both pathologies.

In the ICU, he was continued on High Flow Nasal Oxygen (HFNO) for respiratory support and saturation was maintained more than 94%. He was started on enoxaparin prophylactic dose at 40mg daily as the D-dimers were 217ng/ml. Intravenous Dexamethasone 6mg daily was also commenced. Same antibiotics were continued following the opinion from microbiologist. He was educated on chest physiotherapy but awake proning was not possible due to the fracture and immobilization. He was not given tocilizumab.

Fluid therapy and nutritional supports were maintained. Orthopaedic team decided that surgical intervention was not needed and planned for conservative management.

During the stay, his oxygen requirement was static on first 3 days and he was in moderate ARDS. He neither could be weaned off nor needed increased oxygen support to maintain the target saturations.

He continued to be tachypnoeic with the respiratory rate ranging between 30-60/min. He continued to be tachycardic at 100-120 beats /min in sinus rhythm with stable blood pressure. He was afebrile after the admission and the pain score on NRS was 2/10 managed only on paracetamol.

From day 3 onwards his respiratory symptoms improved. The RR went down, and the oxygen requirement decreased. On day 5 of ICU stay he was weaned to face mask oxygen. Antibodies for SARS-CoV-2 was detected in his blood sample.

A diagnosis of fat embolism syndrome was made in the light of clinical and laboratory findings. This was further supported by the findings on HRCT. On day 5 he was discharged from ICU to a ward, and discharged from the hospital with a plaster cast and a plan to review in 2 weeks as an outpatient.

Discussion

Fat embolism syndrome occur following trauma and commonly seen in long bone fractures. Its pathophysiology is poorly understood and is usually described based on the mechanical and biochemical theory.¹ FES usually presents 12 to 72 hours after the initial insult. This patient started to develop symptoms 24 hours after the initial insult.

FES usually has a classical triad, respiratory insufficiency, petechial rash, and neurologic manifestations. Our patient had respiratory manifestations which usually occur early and in 75% of patients.¹ Neurological manifestations are seen in 86% of patients.¹ Acute confusion was noted in our patient. This could be attributable to acute hypoxia and/or as a result of pathophysiology of fat embolism. Dermatologic manifestation is usually seen within 24 to 36 hours but was not seen in this patient.

FES is usually a clinical diagnosis. The usual criteria used are those of Gurd, Schonfeld, and Lindeque.^{1,2,3} Gurd's criteria are most widely used, and requires at least one major criterion and four minor criteria for diagnosis. As per Lindeque's criteria, FES can be diagnosed using respiratory

parameters alone. In this patient, the FES was diagnosed in the presence of two major criteria as per Gurd, respiratory and central nervous system involvement. He also had raised heart rate, pyrexia, fat globules in urine, a sudden drop in platelets and raised ESR (92mm/1st hour). Further, according to recently introduced Schonfeld's criteria, the patient fulfilled more than 5 points^{3,4}

In the current context SARS-CoV-2 infection had become a global pandemic and Sri Lanka was fighting against second wave of the disease. Therefore, his respiratory symptoms also warranted a probable diagnosis for underlying COVID-19 pneumonia. He underwent an early SARS-CoV-2 RNA test which became positive.^{4,5} This virus is known to cause severe bilateral pneumonia and acute respiratory distress syndrome (ARDS) which can lead to difficulty in breathing requiring supportive ventilation and intensive care management with significant morbidity and mortality.⁶

FES usually resolves spontaneously but may rarely lead to lethal consequences.¹ Differentiating FES from COVID-19 pneumonia is difficult as respiratory manifestations (which will have a respiratory distress leading to hypoxic respiratory insufficiency), laboratory results (raised CRP) and radiological findings (CXR and HRCT) of both conditions tend to overlap.⁷

Even though our patient had criteria favouring a tentative diagnosis of FES he was also diagnosed to have underlying SARS-CoV-2 infection. Further, there was an argument that the pattern of patient's fracture was very unlikely to cause a clinically significant FES.

His clinical respiratory parameters and laboratory reports favoured both FES and COVID-19 pneumonia. Further his CXR was inconclusive for a definitive diagnosis. As there were arguments in favour of both diagnoses, dexamethasone and enoxaparin were commenced as standard evidence based management protocol for COVID-19 pneumonia.⁶

In this situation differentiating the cause was important as if the patient progressed into severe ARDS category of COVID-19 pneumonia he would be an ideal candidate for early use of tocilizumab. He had higher oxygen requirements, was in the first week of respiratory symptoms manifestation and had CRP values of >75mg/dl.^{6,8} But if his diagnosis was more in favour of FES, giving tocilizumab (with high side effect profile) would have been unwarranted and costly.

HRCT of lungs was done to make a definitive diagnosis. There was extensive bilateral air space shadowing in all 5 lobes, patchy consolidations and ground glass appearance with a severity score of 25/25. However, the radiologist reported the HRCT more in favour of FES or infective pneumonia as the pattern of consolidation and ground glass appearance were not typical of COVID-19 pneumonia.

In the absence of continued fever and a high WBC supportive of bacterial infection, the multi-disciplinary team decided to carefully monitor the patient closely and to consider treatment with tocilizumab in the event of clinical deterioration. Tocilizumab was not given as the respiratory parameters were not shown a downward trend.

The antibody levels against SARS-CoV-2 virus became positive on day 5 since admission suggesting a possible early infection with the virus. PCR positivity could have been explained by the late shedding of remaining RNA particles, favouring the diagnosis of FES.

He was given ICU care for 5 days and was discharged to ward with complete clinical recovery of respiratory functions.

Conclusion

FES and COVID 19 pneumonia may have similar clinical, laboratory and radiological findings. FES is usually self-limiting and rarely may lead to lethal complications. On the other hand, COVID 19 pneumonia is associated with increased morbidity and mortality among the affected. FES usually

needs supportive management whereas COVID-19 pneumonia requires additional medications and measures.⁶ In a situation where a diagnostic dilemma exists, careful monitoring, timely decision making and intervention plays a pivotal role to improve patient outcomes.

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