Article title: COVID-19 And Erythrocyte Aggregates: An Intensivists Experience When Being Affected
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COVID-19 AND ERYTHROCYTE AGGREGATES: AN INTENSIVIST'S EXPERIENCE WHEN BEING AFFECTED

ABSTRACT

COVID-19 pandemic has killed over 270,000 individuals to date. Healthcare providers are profoundly vulnerable to be contaminated as opposed to taking every single careful step. Although the respiratory tract and the lungs are the target organs some complications may develop even at the introductory phase of this sickness course. Hemoconcentration with raised serum ferritin levels is one of the dangerous conditions that might be occurred from chronic hypoxia and severe dehydration because of increased insensible loss. Recent posthumous pulmonary tissue studies revealed that the viral infective mechanism, as well as the miniaturized erythrocyte aggregates, are additionally a significant contributing phenomenon to create acute respiratory distress syndrome (ARDS). Hematological issues require to deal proactively alongside other vital organ protection protocols for better outcomes. This article will depict the disease sequence of an intensivist working in a COVID unit after being infected by COVID-19.

Key words: COVID-19; ARDS; Hemoconcentration; Erythrocyte aggregates; Thromboembolism

INTRODUCTION

A 35-year-old male intensivist, youthful and lively, having no comorbidity, no absence of confidence and inspiration. He was at the frontline of the fight against the deadly COVID-19 not long after the discovery and authority revelation of the primary case in Bangladesh on 8 March 2020.[1] He was dynamic, light-footed and vigor in the Critical Care Centre of a Level IV tertiary military hospital where a corona unit is initiated recently. A portion of the staffs of this unit additionally associated with the Emergency and Casualty division. They carefully followed the prudent orders and utilized individual defensive gear while on their obligations.

This case report will portray the ailment course of a fully complied intensivist working in a corona unit after being tainted by COVID-19 out of a propelled medical focal setting of Bangladesh.

THE CASE

Despite his adherence to the principles and conventions of contamination control in the hospital, he had a gentle dry hack and runny nose on 3 April 2020. There was tiredness that was somewhat uncommon in his everyday life. He additionally felt light myalgia and bone torments for days. However, he thought those might be because of ongoing additional overwhelming obligations and physical depletion. He took simple analgesics (paracetamol) and antihistamine orally. It diminished muscle torment and rhinorrhea as well. However, he felt abnormal tiredness and light cough by even nominal physical activities like attending the patients. On that night, in the wake of coming back from the hospital to his dormitory, he felt hot and he noted his body temperature at 102 degrees Fahrenheit estimated by a clinical thermometer. He became very disappointed and informed it to his senior in-control. His in-charge exhorted him to report to the fever clinic of the emergency division for affirmation. He got conceded in the segregation unit at that night on 6 April 2020 and was given the following introductory treatment:

- Tab. Paracetamol 500 mg for fever
- Tab. Fexofenadine 120 mg 1+0+1 is an antihistamine
- Tab. Montelukast 10 mg 0+0+1
- Oral rehydration saline (ORS) and water as much as he drinks to keep up haematocrit level typical
- Tab. Vitamin C 1+1+1 to increase immunity
- Tab. Zinc 1+0+1 to increase immunity

Relevant investigations were in progress. His nasopharyngeal sample was sent sharp for real-time polymerase chain reaction (RT-PCR). Colleagues were guaranteeing him that it will be negative, nothing to be stressed and it's a simple viral fever. In any case, following a couple of hours, he turned out to be seriously stunned after realizing that his sample was certain for COVID-19. He got a condition of thought block assuming the upcoming consequences of this novel coronavirus disease. He was moved to a cubicle of the corona unit and following administration was included:

- Tab. Hydroxychloroquine 200 mg 2+0+2 on the first day and then 1+1+1 for subsequent 10 days
- Tab. Favipiravir 200 mg 8+0+8 on the first day then 3+3+3 on subsequent days
- Cap. Doxycycline 100 mg 1+0+1 for forestalling atypical pneumonia

High-resolution computed tomographic (HRCT) scan of his chest was done on the next morning and the report was: acute respiratory distress syndrome (ARDS) with bilateral ground-glass opacity with pneumonia. Then, the accompanying parenteral meds were added:

- Inj. Meropenem 1 gm 1+1+1 IV
- Inj. Moxifloxacin 400 mg 0+1+0 IV

Instead of above-mentioned management along with the highest dose of intravenous paracetamol his body temperature never touched the benchmark for seven days. Therefore, he turned out to be increasingly feeble and delicate. His liver proteins, hemoglobin and serum ferritin levels were discovered increasing step by step. The vital laboratory findings were:

- Hb%: 19 mg/dL
- Haematocrit: 49%
- WBC count: 18,000/µl
- Neutrophil: 90%

- Serum Ferritin: more than 9,000 ng/mL
- ALT and LDH: were elevating bit by bit
- D-dimer: Negative
- S. Creatinine: 0.9 mg/dL
- CRP: Negative
- S. Procalcitonin: Normal

At that point, the following meds were included:

- L-Omithine+L-Aspartate (converts toxic ammonia to non-toxic urea)
- Ursodeoxycholic acid (suppresses synthesis and secretion of cholesterol from the liver and reduces cholesterol absorption from intestines)
- Prednisolone 40 mg 1+0+1 (for ARDS)
- Target-controlled hydration

His weakness increased on the next day and he began to feel initial breathlessness on mild activities, then even on rest. His oxygen saturation was estimated 83-84% by pulse oximetry. At that point, he was given high stream humidified oxygen by ventimask at a rate of 6-8 L/min. It increased his saturation a little (92-93%). He developed bilateral crepitation in both lung fields that gave him feelings of suffocations and chest tightness. At that moment, he was about to be intubated and went on mechanical ventilation. That made the intensivist exceptionally tense and anguished, indeed.

However, the invasive episodes of interventions could be avoided as the condition didn't deteriorated further. At that point, he adopted a periodical change of his posture at his own to take advantages of ventilation in prone and lateral positions. Then, he got management of negative fluid balance just to maintain a normal or near-normal range of hematocrit value. He was given the following to prevent the thromboembolic phenomenon:

- Deferoxamine (an iron-chelating agent) 1 gm slow IV infusion BD for 3 days
- Prophylactic enoxaparin (low molecular weight heparin) 40 mg SC for 5 days

Three to four days after the fact, his clinical conditions began to improve and the lung fields were seen little well on the accompanying chest Computed Tomographic films. At that point, he rehearsed chest physiotherapy at his own and he found the breath-holding exercise the most productive. His oxygen saturation raised to 95-96% with oxygen at a rate of 2 L/min. He was feeling better step by step and announced relieved after discovering two consecutive RT-PCR negatives on 22nd day (29 April 2020) and released from the hospital.

DISCUSSION

This novel disease course teaches us some important issues. A study on 7,015 confirmed cases demonstrated that it is rapidly transmitting and has a short and widely variable incubation period (5-14 days). The onset chiefly occurs among young to middle-aged adults (average age of all cases was 44.24 years old).[2] Healthcare personnel are the most vulnerable group of people to be infected by COVID-19.[3] The inadequacy of proper testing facilities at the mass level is predominant as like as many developed countries. Standard personal protective equipment (PPE) supply and competent compliance to the protocols are also in lacking.[4] Working in the emergency and outpatient department is the most susceptible area of contamination.[5] Sadly, it is reported that some patients are carrier without any symptoms, while many of them ignore or deliberately hide their complaints mimicking the initial signs and recent exposure of COVID patients to their primary physicians.[6] Then again, doctors and paramedics working in the emergency department must be wary and should attempt to maintain a strategic distance from aerosol-generating procedures (AGP) as much as possible.[7]

Hyperpyrexia doesn't relief for about a week instead of utilizing the highest dose of analgesics. This causes excessive perspiration and insensible loss of volume which may contribute to

developing severe dehydration.[8] Dry cough, throat and chest pain produces irritability and discomfort which instigate to spit more. On top of these, hypoxia initiates excessive red blood cell production and an increased level of serum ferritin.[9] In this way, hypoxia and increased hematocrit are a vicious cycle. These entire phenomena may ignite the risk of thromboembolism because of hemoconcentration. It is also detailed that, among subjects not treated with heparin, mortality raised agreeing with D-dimer levels.[10] The management may remain stay occupied with the hyposaturation and the chest films and this aspect of complication may be unintentionally ignored while treating the COVID-19 patients. Rather, microthrombus, stroke and pulmonary embolism are the potential reasons for the sudden death of these patients. It warrants early detection and appropriate management for better results. Recent studies indicated that erythrocyte aggregates maybe one of the potential causes of ARDS (Figure 1)[11] and acute tubular necrosis (ATN)[12].

Although the lung is the target organ of COVID-19 however other vital organs functions are to be monitored proactively. Liver enzymes and serum creatinine are to be checked routinely.[13] Respiratory invasive interventions are tried the most to be avoided. Self-adopted chest physiotherapy, incentive spirometer and lateral and/or prone decubitus have shown effective fruitfulness.[14] Patients must be consoled to forestall psychological injury as it's an obscure and unique disease process.[15] They might be encouraged by an audio-visual display to perform these useful exercises in the wards and at home.

CONCLUSION

Intelligent suspicion, inquisitiveness; caregivers safety; early detection, isolation and proactive concise management are the key instruments to win the battle against COVID-19. The issue of erythrocyte aggregates shouldn't be missed anyway. Take hold of the ailment before it assumes control over you. The final victory could be accomplished only after the innovation of an effective vaccine against this lethal virus which is in the pipeline.

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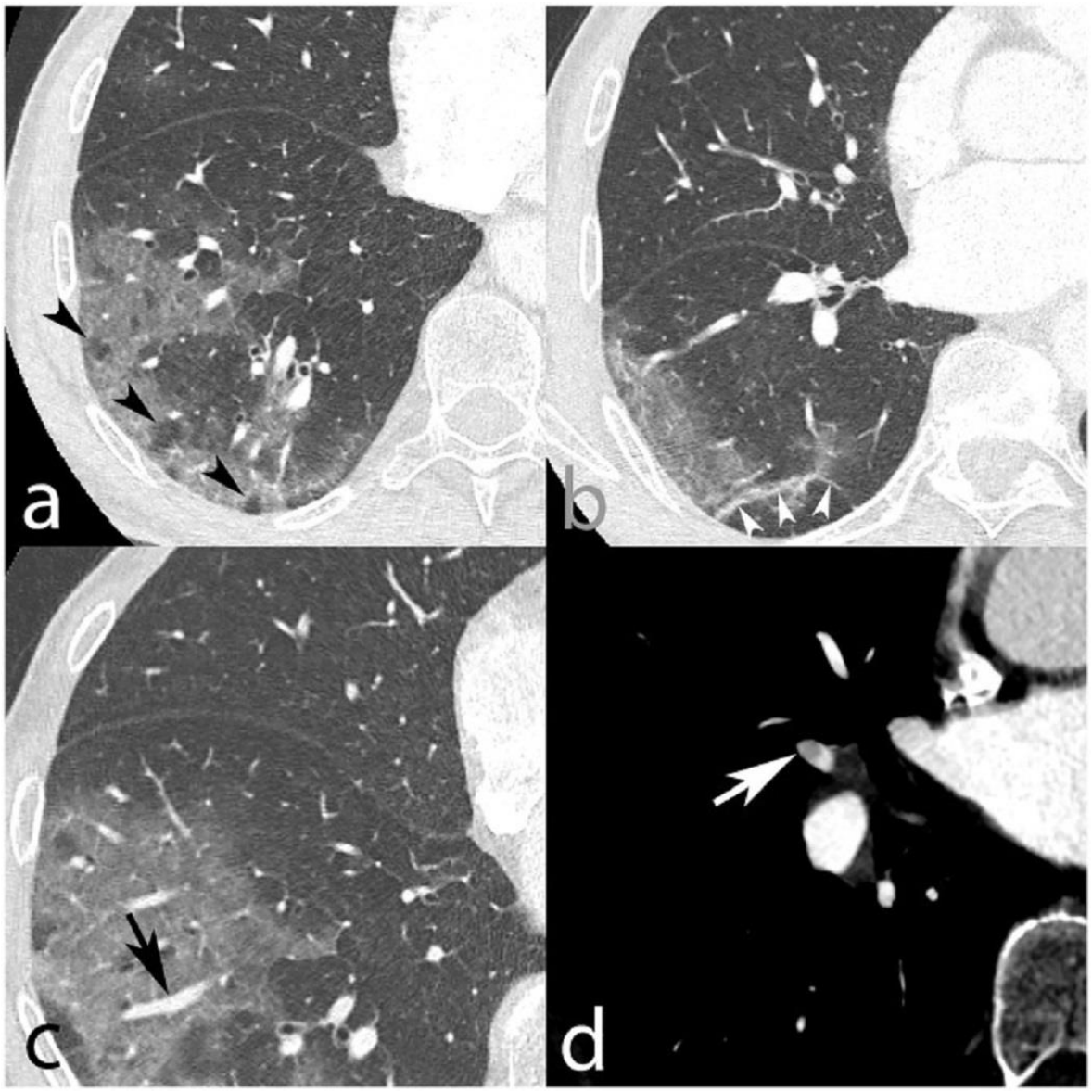

Figure 1: Axial CT pulmonary angiography in lung window, from a 75-year-old man, who was diagnosed with COVID-19. Images show multifocal predominantly peripheral ground-glass opacities in the right lung base (a–c), with associated vacuolar sign (black arrowheads, a), fibrous streaks (white arrowheads, b), and vascular dilation sign (black arrow, c) suggestive of SARS-CoV-2 infection. In the soft tissue window, a filling defect partially outlined by contrast agent was found in the lateral branch of the right middle lobar artery, indicating acute pulmonary embolism (white arrow). Acute pulmonary embolism was unlikely to be caused by in-situ thrombosis due to interstitial COVID-19 injury since the parenchyma in the right middle lobe was normal (b). *[Reproduced from: Rotzinger DC, Beigelman-Aubry C, von Garnier C, Qanadli SD. Pulmonary embolism in patients with COVID-19: Time to change the paradigm of computed tomography. Thromb Res 2020;190:58-59. doi:10.1016/j.thromres.2020.04.011]*