A COVID-19 Young Girl Patient with Developing Pneumonia and Septic Shock Passing the Death

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Abstract

Rationale: Coronavirus disease is a current worldwide pandemic serious infection. Septic shock is a very common cause of death due to COVID-19 infection. There is a strong relationship between the severity of COVID-19 infection and death. COVID-19 infection is a well-known trigger for severe inflammatory changes and sepsis. Up till now, there is no well-known treatment for COVID-19.

Patient concerns: A young girl COVID-19 patient admitted to the critical care unit with severe shock and developing pneumonia.

Diagnosis: COVID-19 patient admitted to the critical care unit with COVID-19 young girl patient with septic shock and developing pneumonia.

Interventions: Electrocardiography, arterial blood gas test, chest CT scan, central venous catheterization, and intravenous vasopressors infusion.

Outcomes: Progressive dramatic clinical electrocardiography and radiological improvement had happened.

Lessons: It denotes the role of anticoagulants, vasopressors, steroids, and antibiotics in the management of COVID-19 pneumonic infection and septic shock. C- reactive protein, lymphocytic count, liver enzymes, and CT chest may be used as a good **laboratory** guide for follow up for the inflammatory activity of COVID-19 patient. Blood pressure, respiratory rate, and O₂ saturation are another strong guide for **clinical** for follow up in COVID-19 patients.

Keywords: COVID-19, Coronavirus, Extensive pneumonia, shock, Death

Abbreviations

ABG: Arterial blood gas

COVID-19: Coronavirus disease 2019

ECG: Electrocardiography

ICU: Intensive care unit

IV: Intravenous

IVB: Intravenous bolus

VR: Ventricular rate

Introduction

The novel coronavirus (COVID-19) associated with a severe acute respiratory

infection may complicate with septic shock and thromboembolism [1]. Organ dysfunction eg, shock, acute respiratory distress syndrome (ARDS), and death can

occur in severe cases [2]. Major complications during hospitalization included ARDS, arrhythmia, and shock. The most common presenting symptoms of COVID-19 infection indicating hospital admission are fever, fatigue, and a dry cough [3]. The current agreeable definition of sepsis is a life-threatening organ dysfunction caused by a dysregulated host response to infection. A small percentage of COVID-19 infections can yield in organ failure and death [4]. Septic shock is considered in coronavirus infection when the blood pressure declines to critically low levels post-infection. The condition can be caused by either a bacterial or viral infection [5]. Anyhow, autopsy in postmortem studies in the dead patients of respiratory failure had evidence of exudative extensive alveolar damage, enormous capillary congestion, and often accompanied by microthrombi. About 40% of the cases showed overlapped bronchopneumonia. The acute pulmonary embolisms, alveolar hemorrhage, and vasculitis are considered serious associations. Patients may have signs of thrombotic microangiopathy [6,7]. As reported earlier, severe acute respiratory syndrome coronavirus (SARS-CoV) is a milder version of coronavirus with a 10% mortality rate whereas Middle East respiratory syndrome coronavirus (MERS-CoV) has a mortality rate of 37%. Acute respiratory distress syndrome with cytokine storm may be the reason for the increased mortality in Novel Wuhan Coronavirus COVID-19 [8].

Case presentation

A 16-year-old single, student, Egyptian young girl patient presented to the emergency department (ED) with acute tachypnea, fever, and cough. Vomiting, sweaty, and dizziness were initial associated symptoms. She gave a history of fever for 12 hours. Her mother denies a history of contact with COVID-19 patients in the past 14 days. Upon general physical examination; generally, the patient was anxious, centrally cyanosed, profuse sweaty, with a regular tachycardia of VR 130 bpm, blood pressure of 70/40 mmHg, respiratory rate of 38 bpm, the temperature of 39.6 °C, and pulse oximeter of O₂ saturation of 87 %. No more relevant clinical data were noted during the clinical examination. He was admitted to the intensive care unit

(ICU) as a septic shock of unknown cause. Initially, the patient was treated with O₂ inhalation (100%, by nasal cannula, 5L/min). IVB of normal saline (0.9 % 500 ml over 30 minutes), Ringer solution (500 ml over 30 minutes), and heparin sulfate (5000 IU IVB amp) were immediately given. There was no improvement in blood pressure. Norepinephrine was added with an initial IV infusion rating dose: 10 mcg/min and titrated for maintenance 3 mcg/min IV infusion for about 48 hours. The patient was treated with SC enoxaparin 80 mg twice daily), aspirin tablet (75 mg, once daily), clopidogrel tablet (75 mg, once daily), imipenem; (500 mg IV every 12 hours), azithromycin (500 mg PO single daily dose on the day one, followed by 250 mg PO daily on for 5 days), oseltamivir (75 mg PO twice daily for 5 days), hydrocortisone sod succinate (100 mg IV every 12 hours for 7 days), and paracetamol (500 mg IV every 8 hours). The patient was monitored by central venous pressure (CVP) placement and urinary output. The initial ECG on ICU admission showing sinus tachycardia with VR of 130. There was widespread flat T- wave (I, aVL, II, III, aVR, aVF V4,5, and 6) (**Figure 1A**). Within 6 hours clinical deterioration occurred. The initial complete blood count (CBC); Hb was 10.6 g/dl, RBCs; $4.82 \times 10^3/\text{mm}^3$, WBCs; $21.4 \times 10^3/\text{mm}^3$ (Neutrophils; 95 %, Lymphocytes: 2%, Monocytes; 2%, Eosinophils; 1%, and Basophils; 0%), Platelets; $205 \times 10^3/\text{mm}^3$. Measured random blood sugar was 170 mg/dl. S. Ferritin was; 78 ng/ml. The troponin test was normal (0.078 ng/ml). CK-MB was high (45.8 U/L). D-dimer was high (1200 ng/ml). The initial CRP was; 157.1 g/dl. SGPT; 52 U/L, SGOT; 38 U/L, serum creatinine; 1.26 mg/dl, blood urea; 35 mg/dl. ABG showed compensated respiratory alkalosis (PH; 7.428 mmHg, PCO₂; 29.9 mmHg, HCO₃; 19.5 mmHg, and PaO₂; 67.1 mmHg). Plasma sodium was normal (145 mmol/L). Serum potassium was normal (3.9 mmol/L). Total Calcium showing mild decreasing; 9.2 mg/dl and ionized Calcium; 1.24 mmol/L. S amylase was 225 U/L and S lipase was 29.24 U/L. The S. lactate level was 9 mmol/L. In the on the second day of admission; complete blood count (CBC); Hb was 8.9 g/dl, RBCs; $3.98 \times 10^3/\text{mm}^3$, WBCs; $25.4 \times 10^3/\text{mm}^3$ (Neutrophils; 87.2 %, Lymphocytes: 10.7%,

Monocytes; 2.1%, Eosinophils; 2.1%, and Basophils; 0%), Platelets; $217 \times 10^3/\text{mm}^3$. CRP was; 157.1 g/dl. SGPT; 122 U/L, SGOT; 111 U/L, serum creatinine; 0.9 mg/dl, and blood urea; 44 mg/dl. In the on the sixth day of admission; complete blood count (CBC); Hb was 9.3 g/dl, RBCs; $4.23 \times 10^3/\text{mm}^3$, WBCs; $19.3 \times 10^3/\text{mm}^3$ (Neutrophils; 74 %, Lymphocytes: 18 %, Monocytes; 7%, Eosinophils; 1%, and Basophils; 0%), Platelets; $546 \times 10^3/\text{mm}^3$. CRP was; 12.51g/dl. S. creatinine was; 0.77 mg/dl and blood urea; 32 mg/dl. In the on the eighth day of admission; ; complete blood count (CBC); Hb was 10.1 g/dl, RBCs; $5.27 \times 10^3/\text{mm}^3$, WBCs; $13.3 \times 10^3/\text{mm}^3$ (Neutrophils; 40 %, Lymphocytes: 55 %, Monocytes; 4%, Eosinophils; 1%, and Basophils; 0%), Platelets; $637 \times 10^3/\text{mm}^3$. CRP was; 7.57g/dl. The platelets 5 days post-discharge was; $433 \times 10^3/\text{mm}^3$. The pharyngeal swab for COVID-19 was positive. The first radiological film was done on the day of admission: Plain Chest imaging (**Figure 2A1**) and Chest CT scan (**Figure 2A2**) showing no abnormality detected. The second radiological film was done on the second day of admission; Plain

Chest imaging (**Figure 2B1**) and Chest CT scan (**Figure 2B2**) showing extensive ground-glass consolidation in the middle and lower right lobes. The third radiological film was done on the 8th day of admission: Plain Chest imaging (**Figure 2C1**) and Chest CT scan (**Figure 2C2**) showing dramatic improvement of above extensive ground-glass consolidation. Echocardiography was normal. Suspected acute pneumonic infection and septic shock pneumonia due to COVID-19 was the most probable diagnosis. ECG **tracing** was taken within 8 days of ICU admission showing normal sinus rhythm with VR of 72. There was non-specific T- wave inversion (I, II, aVL, and V5, 6) with upright T- wave in aVR (**Figure 1B**). ECG **tracing** was taken one week after hospital discharge showing normal sinus rhythm with VR of 76 with non-specific T-wave inversion (I and aVL) with mild ST-segment elevation (II, III, aVF, V4, and V5). Also, there was a wavy triple an electrocardiographic Sign (Yasser Sign) of hypocalcaemia (V3 and V6). (**Figure 1C**). The patient discharged within 8 days after complete clinical, radiological, and laboratory improvement.

Figure1: Serial ECG tracings; A. tracing was the initial ECG on ICU admission showing sinus tachycardia with VR of 130. There was wide-spread flat T- wave (I, aVL, II, III, aVR, aVF V4,5, and 6; lime arrows). **B. tracing** was taken within 8 days of ICU admission showing normal sinus rhythm with VR of 72. There was non-specific T- wave inversion (I, II, aVL, and V5,6; orange arrows) with upright T- wave in aVR (blue arrows). **C. tracing** was taken one week after hospital discharge showing normal sinus rhythm with VR of 76. There was non-specific T- wave inversion (I and aVL; orange arrows) with mild ST-segment elevation (II, III, aVF, V4, and V5; orange arrows). There was a wavy triple an electrocardiographic Sign (Yasser Sign) in hypocalcemia (V3 and V6; pink arrows).

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Figure2: First film: **2A1.** Plain Chest imaging and **2A2.** A chest CT scan was done on the day of admission showing no abnormality detected. Second film: **2B1.** Plain Chest imaging and **2B2.** A chest CT scan was done on the second day of admission showing extensive ground-glass consolidation in the middle and lower right lobes (lime arrows). Third film: **2C1.** Plain Chest imaging and **2C2.** A chest CT scan was done on the 8th day of admission showing dramatic improvement of above extensive ground-glass consolidation.

Discussion

• Overview:

• A young girl COVID-19 patient admitted to the critical care unit with irreversible severe shock and developing pneumonia.

• **The objective primary** for my case study was the presence of irreversible severe shock and developing pneumonia within 48 hours of fever.

• **The secondary objective** for my case study was the **question** of; How did you manage the case?

- There was an unclear history of direct contact to confirmed the COVID-19 case. Her father is a dentist.
- Despite initial normal lungs, the development of ground-glass extensive consolidation, lymphocytopenia, and positive pharyngeal swab on top of fever, tachypnea, and dry cough will be supporting the COVID-19 diagnosis.
- The severe shock, hypoxia, and elevated d-dimer are highly suggestive of associated pulmonary embolism.
- Hyperthermia with hypotension on the case presentation may be interpreted by the presence of severe septic shock (due to pneumonia).
- The conversion sinus tachycardia normal sinus rhythm in ECG after clinical improvement was marked.
- A delayed blood pressure normalization for about days despite fluid infusions was an indication for adding norepinephrine IV infusion.
- Later blood pressure normalization occurred after establishment and maintenance with the effects of anti-infective drugs, anticoagulants, and antiplatelet.
- Later blood pressure normalization occurred after the establishment and maintenance of anti-infective drugs, anticoagulants, steroids, and antiplatelet strongly implies their effects.
- The presence of non-specific ST-segment and T-wave changes indicate either; pneumonia or pulmonary embolism.
- Blood pressure, respiratory rate, and O₂ saturation are a strong guide for clinical for follow up in COVID-19 patients.
- A gradual decreasing the level of elevated CRP, lymphocytic count, liver enzymes, and CT chest may be used as another good laboratory guide for follow up in COVID-19 patients.
- Marked thrombocytosis was mostly reactive and secondary to either infection or drug inducing.
- The platelets gradually and spontaneously regressive to normal level after complete recovery and medications cession.

• The serial change of radiological changes from normal chest CT to abnormal to normal at the end will strengthen the used medications in this effective management. Also, it gives a good radiological impact for follow up.

• I can't **compare** the current case with similar conditions. There are no similar or known cases with the same management for near comparison.

• Limitations of the study:

• There are no known limitations to the study.

Conclusion and Recommendations

• It implies the role of anticoagulants, vasopressors, steroids, and antibiotics in the treatment of COVID-19 acute respiratory disease and septic shock.

• C- reactive protein, lymphocytic count, liver enzymes, and chest CT may be used as a recommended good **laboratory** guide for follow up for the inflammatory activity of COVID-19 patient.

• Blood pressure, respiratory rate, and O₂ saturation are another recommended strong guide for **clinical** for follow up in COVID-19 patients.

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References

- [1] **Coronaviridae Study Group of the International Committee on Taxonomy of Viruses** (2020). The species severe acute respiratory syndrome-related coronavirus: classifying 2019-nCoV and naming it SARS-CoV-2. *Nat Microbiol.* 5(4):536-44.
- [2] **Huang C, Wang Y, Li X, et al** (2020). Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China [published January 24, 2020]. *Lancet.* DOI:10.1016/S0140-6736(20)30183-5
- [3] **Zick M** (2020). Can COVID-19 Cause Sepsis? Explaining the Relationship Between the Coronavirus Disease and

- Sepsis. Available online: [https:// www.global-sepsis-alliance.org/news /2020/3 /4/can-covid-19-cause-sepsis-explaining-the-relationship-between-the-coronavirus-disease-and-sepsis-cvd-novel-coronavirus](https://www.global-sepsis-alliance.org/news/2020/3/4/can-covid-19-cause-sepsis-explaining-the-relationship-between-the-coronavirus-disease-and-sepsis-cvd-novel-coronavirus) (Accessed: March 4, 2020)
- [4] **Dawei Wang, Bo Hu, Chang Hu, Fangfang Zhu, Xing Liu, Jing Zhang, et al** (2020). Clinical Characteristics of 138 Hospitalized Patients With 2019 Novel Coronavirus-Infected Pneumonia in Wuhan, China. *JAMA*. 2020;323(11):1061-1069. DOI:10.1001/jama.2020.1585
- [5] **Laguipo ABB** (2020). Teens develop septic shock due to COVID-19. Available online: <https://www.news-medical.net/news/20200524/Teens-develop-septic-shock-due-to-COVID-19.aspx> (Accessed: May 24 2020).
- [6] **Menter T , Haslbauer JD, Nienhold R, et al** (2020). Post-mortem examination of COVID19 patients reveals diffuse alveolar damage with severe capillary congestion and variegated findings of lungs and other organs suggesting vascular dysfunction. *Histopathology* . 2020 May 4. PMID: 32364264 DOI: 10.1111/his.14134 [Epub ahead of print].
- [7] **Schaller T , Hirschtbühl K, Burkhardt K, et al** (2020). Postmortem examination of patients with COVID-19. *JAMA*. 2020 May 21. DOI:10.1001/jama.2020.8907 [Epub ahead of print].
- [8] **Rajan R, Jarallah MAI, Dashti R** (2020). Cardiovascular complications of novel Wuhan Coronavirus (COVID-19) – A 2020 update. *J Cardiol Curr Res*. 13(1):28. DOI: 10.15406/jccr.2020.13.00468

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