Case Report

Ongoing Challenges of Extubation of COVID-Pneumonic Patient in Multi-Organ Failure: a Case Report

Md Zulfiker Riad Ibn Aziz[1], Md Masum Hossain Arif[2], Abdullah Al Mamun[2], A. K. M Abdun Noor[3], A. K. M Jakaria[3], Mahfuz Ahmed Chowdhury[4], Khondoker Hasan Al Hudaibi[4], Md Mahbub Noor[5], Md. Sadiqul Islam[6]

1. Specialist, MICU, Critical Care Unit, Evercare hospital Dhaka.
2. Specialist, MICU, Critical Care Unit, Evercare hospital Dhaka.
3. Senior Specialist, MICU, Critical Care Unit, Evercare hospital Dhaka.
4. Associate Consultant, MICU, Critical Care Unit, Evercare hospital Dhaka.
5. Senior Consultant and Coordinator, MICU, Critical Care Unit, Internal Medicine, Evercare hospital Dhaka.
6. Senior Consultant Internal Medicine, Evercare hospital Dhaka.

Address for Correspondence:
Dr. Md Zulfiker Riad Ibn Aziz
Registrar
MICU, Critical Care Unit
Evercare Hospital Dhaka.
zkariad@gmail.com

ABSTRACT

In this report, a case of a patient with COVID-19 pneumonia is presented who developed multi-organ failure and required mechanical ventilation. Despite multiple attempts at extubation, the patient remained intubated for an extended period due to respiratory failure. The challenges encountered during the process of extubation are discussed and the strategies employed to overcome them. This case highlights the importance of careful decision and management in patients with COVID-19 pneumonia for extubation who develop multi-organ failure.

Key words: *Covid-pneumonia, Extubation, Multiorgan Failure*

INTRODUCTION

The coronavirus disease 2019 (COVID-19) is an acute infectious disease caused by infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The World Health Organization (WHO) has labeled COVID-19 as a global infectious disease pandemic. COVID-19 is the third major outbreak caused by coronavirus in this century, with the earlier ones being severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS).

Physicians and care providers are familiar with the management of acute respiratory distress syndrome (ARDS), however when it occurs as a sequela of COVID-19, it has different features and there remains uncertainty on the consensus of management especially in i) acute hypoxemic respiratory failure, ii) presentation within 1 week of worsening respiratory symptoms and iii) bilateral airspace disease on chest X-ray, computed tomography (CT), or ultrasound that is not fully explained by effusions, lobar or lung collapse, or nodules. Clear information will provide insight into the pattern of patient response and challenges faced by the ICU teams and give a comprehensive multispecialty recommendation for circumnavigating these difficulties.

CASE REPORT

A 68 years old gentleman of a known case of hypertension for 10 yrs, chronic kidney disease for 8 yrs, old myocardial infarction, congestive cardiac failure (EF-25%), S/P-CABG (2013), AICD (Dual Chamber) was admitted to Evercare Hospital Dhaka through the emergency department on 6.9.22 at 11pm with the complaints of sore throat and running nose for 2 days, shortness of breath for 1 day. The patient had no history of fever. On admission, his pulse was 80 beats/min, BP 140/80 mm of hg, temperature 98oF, respiratory rate 18 breath/min, SPO2 96% in room air, RBS 10.9 mmol/l. heart S1+S2+0, lungs: bilateral creeps present. CXR showed bilateral ground glass opacities and features of pneumonia, which is a sign of lung involvement of COVID1(Figure-1).

He was shifted from the ward to the medical ICU in the evening on 7.9.22 due to the onset of sudden

severe respiratory distress followed by desaturation with high oxygen demand. The patient was intubated immediately at 9:15 pm on the same day due to impending respiratory arrest and severe metabolic acidosis. Then he was put on mechanical ventilation in AC mode, FiO2-100%, with sedation, muscle relaxant and inotropes.

In ICU on 3rdday, he developed atrial fibrillation with a fast ventricular rate. Therefore, bolus intravenous (IV) amiodarone was given followed by maintenance doses for 24 hrs. Then he developed transient ventricular tachycardia which was resolved spontaneously. So, Inj. amiodarone was continued for the next three days as per the advice of the cardiologist. On 5th day chest x-ray was improving and ABG was acceptable, so sedation was stopped, and he was put on SIMV mode. On 6th day the patient's ventilatory effort was acceptable but the patient developed further 2 episodes of transient ventricular tachycardia which resolved spontaneously and IV amiodarone was continued. The patient's GCS was $E_3V_TM_6$. He responded with vocal commands and was arousable, taking spontaneous breaths with adequate tidal volume. Inotrope was stopped. On 7th day IV amiodarone was switched to oral and atrial flutter was controlled. On 8th day he was put on spontaneous mode. All vitals were stable. ABG was acceptable (Table-1). Chest x-ray also improved (Figure-2). On 9th day extubation was done as all the clinical parameters were favorable. Following extubation the patient was put on high flow nasal cannula (HFNC) with 40 L air flow and 5 L oxygen. On 12th day HFNC was gradually stopped and put on a face mask with 5 L oxygen. On 13th day face mask was switched to a nasal cannula with 3L Oxygen. Post-extubation, the patient developed ICU psychosis which was managed conservatively.

Throughout the course the patient received inj. Remdisivir 100 mg iv 24 hourly for 2 days and then 50 mg iv 24 hourly for 3 day, inj. Meropenem 500 mg iv 12 hourly for 13 days, inj. Moxifloxacin 400 mg iv 24 hourly for 4 days, inj. Ticoplanin 400 mg iv 12 hourly for 2 days and then 200 mg iv 24 hourly for 11 days, inj. Pentaglobin 100 ml iv 24 hourly for 5 days, inj. Clexane 40 mg S/C 24 hourly for 22 days. Other drugs given were, tab. Amiodarone 200 mg 8 hourly, tab. Clopid 75 mg 24 hourly, tab. Xinc 20 mg 12 hourly and cap. Dicaltrol 0.25 mg 24 hourly.

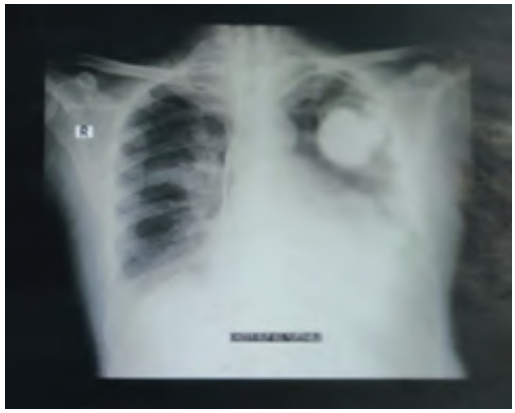

Figure 1: Chest X-ray before intubation

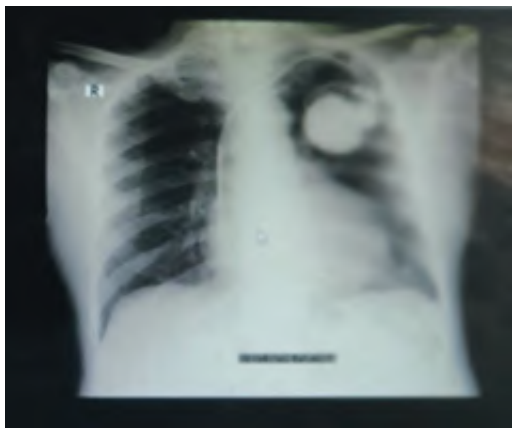

Figure 2: Chest X-ray after intubation

Table 1: Arterial Blood Gas (ABG) Analysis

Parameters	Before intubation	After extubation
pH	7.13	7.49
PCO_2	43	42.5
PO_2	110	119
HCO_3	11	24

Table 2: Laboratory Data during the ICU course

Parameter	Values
Sodium (mmol/L)	138
Potassium (mmol/L)	3.6
Creatinine (mg/dl)	2.50 > 3.70 < 2.48
Urea (mg/dl)	74<158
Ammonia (**µ g d l**	52
CRP (mg/dl)	2.32 > 14.50 < 0.3
Procalcitonin (ng/ml)	6.79 > 22 < 0.78
LDH (U/L)	354
Lactate (mmol/L)	1.1 > 1.4
S. Calcium (mg/dl)	8.2
S. Albumin (g/dl)	3.5
S. Phosphate (mg/dl)	2.7 > 4.7
S. Uric Acid (mg/dl)	8 > 10.3
D-Dimer (**µ g l**	1503>4302 <2459
Pro-BNP (pg/ml)	14956>21055>25000
IL-6 (pg/ml)	204>350<65
High Sensitive Trop.-l (ng/L)	204>506>881>1076>662

Table 3: Coagulation Profile

Coagulation Profile	Values
PT	15.6
INR	1.33
aPTT	22

DISCUSSION

The patient initially had COVID myocarditis, with a low ejection fraction of 20-25%, chest x-ray showed moderate bilateral pulmonary opacities (more on the left). He was hemodynamically unstable with inotrope support, all inflammatory markers were high. He had arrhythmia which was managed according to the institution's ICU protocol and cardiology advice.

During this critical situation, extubation was very difficult although ABG was acceptable and all inflammatory marker was gradually declining. The fever had subsided, and oxygen requirement was reduced (fiO2-0.3), simultaneously inotrope support was stopped, and other clinical parameters were improving. and we started the weaning trial and subsequently patient was extubated after a few days.

Clinical studies on the pathogenesis of this virus show an association with coagulopathy. Our patient also showed a similar phenomenon (Table 3). This however differs from sepsis-associated disseminated intravascular coagulation (DIC) by the relatively normal levels of PT, fibrinogen, and platelets, despite markedly elevated d-dimer levels. Although the primary pathogenesis was thought of as pulmonary type II pneumocyte injury, viral pneumonia, acute respiratory distress syndrome (ARDS), or macrophage activating like syndrome complicating ARDS leading to DIC. SARS-CoV2 binds to angiotensin converting enzyme 2 (ACE2) receptors on type II pneumocytes and possibly on vascular endothelial cells and causes lysis of the cells immediately leading to direct activation of the endothelium causing procoagulant activity and activates accumulation of fibrin deposits in pulmonary microcapillary venous vessels. The fibrin deposits cause a compensatory mechanism of increased plasminogen at the beginning but as the disease progresses fails to break down the fibrin deposits reflected in increased d-dimer levels. In the lung, SARS-CoV-2 causes acute diffuse alveolar damage, pneumocyte hyperplasia, and interstitial pneumonia. Coagulation dysfunction is common in COVID-19 (detected by raised D-dimer levels)[2].

Recent studies have suggested that in addition to direct viral damage, uncontrolled inflammation contributes to disease severity in COVID-19. Consistent with this hypothesis, high levels of inflammatory markers, including C-Reactive protein (CRP), ferritin, D-dimer, and high neutrophil-to-lymphocyte ratio, increased levels of inflammatory cytokines. A high IL-6 predicted a 227% increase in the chances of death. Procalcitonin (PCT) has emerged as a crucial biomarker for the severity and prognosis of COVID19 infection[3].

The treatment for COVID-19 and its problems is mostly to help the patients breathe and keep their organs working. COVID-19 ARDS is an anticipated severe complication of COVID-19 that requires prompt recognition and comprehensive multispecialty management. The procedure of weaning from MV and extubation requires careful judgment. Extubation not only demands the improvement of breathing ability and ABG measurements. But also depends on other factors, and that is the most challenging. The case described here was under MV and gradually improved with acceptable ABG levels. But was not possible to extubate as he had ventricular tachycardia and atrial flutter, and these had to be managed first before extubation.

CONCLUSION

This case highlights the importance of careful decision and management in patients with COVID-19 pneumonia for extubation who develop multi-organ failure. Extensive research and studies are required to address the vital unanswered queries about treatment for COVID-19 pneumonia, because of the high mortality in mechanically ventilated patients of pneumonia, with associated multisystem disorder and COVID-induced multi-organ dysfunction.

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Pyoderma Gangrenosum: A Case Report with a brief review

Q. M. Mahabub Ullah[1], Jasmin Manzoor[2], Rubaiya Ali[1], Walid Hasan Khan[3]

1. Senior Consultant, Department of Dermatology & Venereology, Evercare Hospital Dhaka
2. Senior Consultant & Coordinator, Department of Dermatology & Venereology, Evercare Hospital Dhaka
3. Specialist, Department of Dermatology & Venereology, Evercare Hospital Dhaka

Address for Correspondence:
Dr. Q. M. Mahbub Ullah
Sr. consultant,
Department of Dermatology & Venereology,
Evercare Hospital Dhaka
qm.mahbub.ullah@evercarebd.com

ABSTRACT

Pyoderma Gangrenosum presents as a rapidly enlarging,very painful ulcer. It is one of a group of autoinflammatory disorders known as neutrophilic dermatoses. In this paper we describe a case of Pyoderma Gangrenosum in a 15-year-old girl along with its clinical features, differential diagnosis, histopathological findings and treatment.

Key words: *Pyoderma Gangrenosum, Violaceous Ulcer,*

CASE SUMMARY

A 15-year-old female came to Dermatology OPD of Evercare Hospital Dhaka with complaints of multiple painful ulcers on her left hand for two months. Initially she developed localized pain & swelling in her left forearm. Within 2 days, the color of the swelling changed from skin color to reddish to bluish. A painful ulcer developed within the next 3 days. Gradually similar types of lesions developed on the left forearm. On examination, pain & swelling of left upper limb was present. She had no history of fever, cough, joint pain, diarrhea, or abdominal pain. No history of contact with TB patient or personal history of TB. She was found normotensive, and non-diabetic. Before presenting to us, she has taken Dapsone, Azathioprine, Prednisolone, Amoxicillin, Levofloxacin, Linezolid, Cefuroxime, Clindamycin, and Ciprofloxacin during the disease process without significant improvement.

On integumentary system examination, there are multiple deep-seated ulcers of varying size on flexor surface of left forearm (Fig 1). Margin is irregular & color is violaceous, undermined edge, crusting present & on removal of crust serosanguinous discharge is present (Fig 2). There is a well-defined bluish colored swelling with surrounding erythema on left forearm. Ulcer base was hard on palpation & extremely painful.The localized The localized temperature was increased. No regional lymphadenopathy. Peripheral pulses were normal. Pathergy test was positive. Mucous membrane, hair, nail & other systemic examination revealed no abnormality. The patient was given systemic Prednisolone, Azathioprine, and topical Mupirocin ointment and advised regular dressing of the wound. She had a gradual recovery overtime.

DISCUSSION

Pyoderma Gangrenosum (PG) is a rare disease characterized by a full-thickness ulcer with a blue or purple undermined border and by pathergy[1]. It is often associated with systemic disease. There are several rarer subtypes of pyoderma gangrenosum: Ulcerative (Classic PG), Bullous, Pustular, and Vegetative[2].

The pathogenesis of pyoderma gangrenosum is not fully understood. It is thought to involve genetic mutations, neutrophil dysfunction and immune/inflammatory dysregulation. Post-operative Pyoderma Gangrenosum has been seen reported following breast reconstructive surgery and misdiagnoses of Pyoderma Gangrenosum as necrotizing fasciitis[3,4].

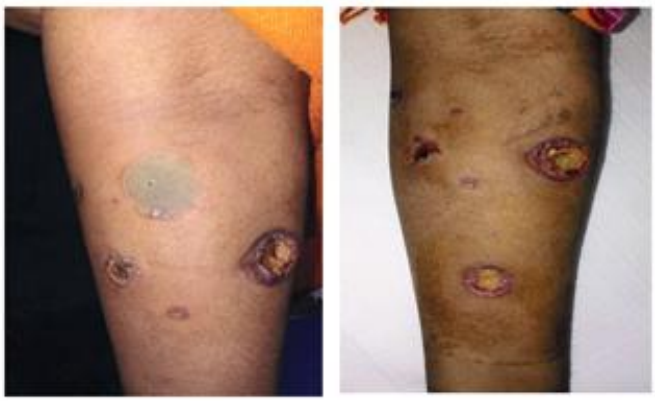

Figure 1: Multiple deep-seated ulcers of varying size on flexor surface of left forearm

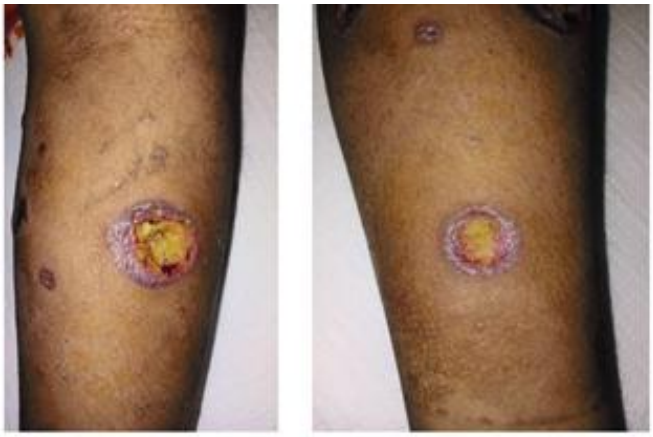

Figure 2: Ulcer showing violaceous colored irregular margin, undermined edge, with overlying crusting and serosanguinous discharge.

PG affects males and females of any age but is more common in those aged over 50 years. It is rare in children. But may be associated with Irritable Bowel Disease, Leukemia, Childhood acquired immunodeficiency syndrome (AIDS). Genitals, head and neck areas are mostly affected in children[5]. For adults, it is frequently associated with an internal disease or condition i.e. Inflammatory bowel disease (ulcerative colitis and Crohn disease), Rheumatoid arthritis, Myeloid blood dyscrasias including leukemia, Monoclonal gammopathy (usually IgA), Chronic active hepatitis, Granulomatosis with polyangiitis, PAPA syndrome, Behçet disease and use of levamisole-adulterated cocaine. Then again, about half of those affected by pyoderma gangrenosum have none of the associated risk factors[1].

Pyoderma gangrenosum usually starts quite suddenly, often at the site of a minor injury. It may start as a small pustule, red bump, or blood-blister, often misinterpreted as an insect bite[6]. The skin then breaks down resulting in an ulcer. The ulcer can deepen and widen rapidly. Characteristically, the edge of the ulcer is purple and undermined. Pyoderma gangrenosum is usually very painful. Several ulcers may develop at the same time or over months to years.

In untreated cases, the ulcers may continue to enlarge, persist unchanged, or may slowly heal. Treatment is usually successful in arresting the process, but complete healing may take months. This is particularly true if there is an underlying venous disease, another reason for leg ulcers. Deep ulcers heal with scarring, and this is sometimes with a characteristic cribriform (criss-cross pattern) or atrophic appearance1. In our patient, there were multiple lesions that started suddenly and rapidly progressed to painful ulcers. The ulcer base was undermined, and the edge was typically purple in color. Some of the ulcers healed with scarring. Pathergy test was positive.

Pyoderma gangrenosum is diagnosed by its characteristic appearance and severe pain. The pathergy test is usually positive (a skin prick test causing a papule, pustule, or ulcer). The wound should be swabbed and cultured for secondary infection. A biopsy may be necessary to rule out other causes of ulceration. In most cases, blood tests are not particularly helpful. Although some patients may have a positive ANCA (antineutrophil cytoplasmic antibody)[1,7].

Histopathological features of Pyoderma gangrenosum include neutrophilic folliculitis and perifolliculitis with Intradermal neutrophilic abscess formation in early lesions. For advanced lesions, epidermal ulceration, superficial dermal necrosis, mixed inflammation with undermining at ulcer edge and abscess, (advancing edge often has subepidermal edema), leukocytoclastic vasculitis and lymphocytic vasculitis, and acanthosis in perilesional zone may be present[8]. The histopathological findings In our case were mild exocytosis in the epidermis. The dermis had moderate perivascular infiltration of lymphocytes, histiocytes & neutrophils. Karyorrhetic debris was present. Subcutaneous tissue revealed infiltration of chronic inflammatory cells.

Differential diagnosis of Pyoderma gangrenosum are Venous or arterial ulcers; Medium and large size vessel

vasculitis; Occlusive vasculopathy; Ecthyma and ecthyma gangrenosum; Ulcerating infections e.g.

Differential diagnosis of Pyoderma gangrenosum are Venous or arterial ulcers; Medium and large size vessel vasculitis; Occlusive vasculopathy; Ecthyma and ecthyma gangrenosum; Ulcerating infections e.g. Buruli ulcer, Lupus vulgaris; Sporotrichosis; Cutaneous amoebiasis; Ulcerating skin tumors and lymphomas; Drug-induced ulcers e.g. iododerma, bromoderma, hydroxycarbamide, nicorandil; Artefact1. Pyoderma Gangrenosum is oftentimes diagnosed by the exclusion of related diseases. Clinical and pathological correlations coupled with laboratory investigation to exclude alternative etiologies is essential.

Management of Pyoderma Gangrenosum is challenging. The treatment is determined by the severity of the disease and the rate of progression. Successful treatment requires both halting the inflammatory process and healing a large wound. In rapidly progressive cases, aggressive early management may reduce morbidity[2,6,-8]. The case reported here was previously treated with inadequate responses. We have given the patient a combination of systemic Prednisolone, Azathioprine, and topical Mupirocin ointment synchronized with a regular dressing of the wound. She had a steady recovery over time.

CONCLUSION

In the majority of patients, the prognosis is good, but the skin disorder does recur. In addition, most people are left with some residual scar. The skin lesions breakdown from minimal trauma. Despite the use of immunosuppressive agents, relapses are common and long-term care is required.

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