INTRODUCTION

In December 2019, an outbreak of unknown acute respiratory tract infection originated in Wuhan, China. Shortly after first lower respiratory tract infection cases, the World Health Organization (WHO) named this newly emerged coronavirus as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and its resultant disease as coronavirus disease 2019 (COVID-19). Coronaviruses are a large class of viruses that often cause mild-to-moderate upper respiratory tract infection.
However, three of them have arisen from animal sources in the past two decades, resulted in severe acute respiratory syndrome (SARS) and Middle East Respiratory Syndrome (MERS) epidemics as well as the novel COVID-19 pandemic.4-6

There is evidence of increased incidence, rapid progression, and poor prognosis of COVID-19 in patients with underlying comorbidities.7,8 Diabetes is a significant risk factor for hospitalization and adverse outcome in these patients,9-11 as similar evidence was reported for two previous CoV infections, SARS and MERS.4,5,12 Furthermore, diabetes is linked with an increased risk of thromboembolism among COVID patients, and D-dimer levels have been confirmed to be directly proportionate to the severity of the disease.13

Moreover, several studies have suggested epilepsy as a potential risk factor to morbidity and mortality in COVID-19 patients; nevertheless, the association as of yet is unclear.14-16

Herein, we report a confirmed case of COVID-19 patient with a history of multiple underlying diseases, including diabetes, epilepsy, and gout. The patient died a week after intensive care unit (ICU) admission. We desire to highlight recesses in the knowledge that require further studies related to COVID-19 in patients with underlying comorbidities.

2 | CASE PRESENTATION

On 18 February 2020, a 47-year-old with acute respiratory syndrome, high fever (39.6 °C), and severe shortness of breath was admitted to ICU at Ghaem Hospital in Karaj, Iran. Regarding strong suspicion of the COVID-19 infection, complete laboratory tests and spiral chest-computed tomography (CT) scan without contrast were performed for the patient. CT scan findings were consisted of bilateral ground-glass opacities predominantly in lower zones, multiple atelectasis bilaterally in same regions, and elevation of right hemidiaphragm. No evidence of pericardial effusion, pleural fluid collection/thickening was reported. Imaging findings were characteristics of pneumonia attributed to SARS-CoV-2 (Figure 1).18-20

The patient had a history of epilepsy, gout (uric acid 9.0 mg/dL) and diabetes. Initial laboratory findings were described as follows: low white blood cells (WBC, 3400/µL), high blood sugar (BS, 179 mg/dL), impaired liver function tests (SGOT/AST, 65 U/L and SGPT/ALT, 41 U/L), and low potassium (K, 3.2 mEq/L) (Table 1). On the first day of admission, the patient received 200 mg of hydroxychloroquine tablets twice daily (bd), 200 mg of lopinavir/ritonavir tablets twice daily (bd), and oseltamivir tablets 75 mg daily. On the second day, patient body temperature was reported 38.8 °C and the Wright test was found negative. Moreover, liver enzymes SGOT/AST and SGPT/ALT were elevated to 85 and 56 U/L, respectively. Due to patient’s history and an episode of seizure during hospitalization, levetiracetam 500 mg intravenous (IV) daily and levofloxacin tablets 750 mg daily were added to his treatment. On 3rd postadmission day, he had a 38°C fever.

**FIGURE 1** Chest CT images of the patient. Ground-glass opacities are seen in both lungs, predominantly in lower zones. Multiple atelectasis is also seen in both lower zones
Sputum culture was performed, which came back negative 48 hours later. Naproxen tablet 500 mg daily was added to his medication. On the 7th postadmission day, the patient expired due to reduced oxygen pressure and asystole myocardial infarction.

### 3 | DISCUSSION

Underlying comorbidities are major risk factors for progression to critical illness and death in COVID-19 patients. Among them, our patient had history of diabetes and epilepsy, which are associated with altered immune system response and potential poor prognosis in COVID-19 patients. Weakened immune response might be related to mechanism in which these comorbidities modulate host-viral or host-immune system interactions. Specifically, it has been demonstrated that diabetes is a major risk factor for in-hospital mortality. Diabetes is also associated with an increased risk of thromboembolism in COVID-19 patients, increasing disease severity and mortality. Moreover, patient’s urinary tract infection was assumed to be due to underlying comorbidities, especially diabetes and gout. Development of acute kidney injury (AKI) in hospitalized COVID-19 patients is a negative prognostic factor and ranges between 3% and 29%. Patient’s proteinuria might be due to developed AKI during hospitalization which is highly associated with in-hospital mortality.

The presented case developed multiple organ failure induced by COVID-19. It is confirmed that COVID-19 can cause cytokine storm, in which high pro-inflammatory cytokines, as in the presented case, can lead to heart failure (CPK 340 U/L), liver failure (elevated SGOT, SGPT, and bilirubin), AKI, and respiratory failure (low PO2). Multiple organ failure is the most common cause of death in these patients.

It is crucial to develop effective treatment regimens for COVID-19 patients with multiple comorbidities, as patients’ past medical history is an essential contributor to possible organ injuries in these patients. Further observations will be required to comprehensively understand the full spectrum of COVID-19 clinical features in patients with multiple comorbidities.

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### CONFLICT OF INTEREST

None declared.

### AUTHOR CONTRIBUTION

ARS, KM, FK, PF, and NR: Contributed to following criteria as described by the International Committee of Medical Journal Editors (ICMJE).

### ETHICAL APPROVAL

Ethical approval is not necessary for retrospective studies and case presentations in our institutional policies.

### CONSENT FOR PUBLICATION

Written informed consent was obtained from the patient’s next of kin for publication of this case report and any accompanying images.

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**TABLE 1** Laboratory findings

| Date    | 02/18 | 02/19 | 02/21 | 02/22 | 02/23 |
|---------|-------|-------|-------|-------|-------|
| WBC     | 3400  | 4400  | 4800  | 7800  |       |
| PaCO2   | 47 (40-52) | 42    | 30.9  |       |       |
| PaO2    | 77.3 (80-100) | 43    | 64    |       |       |
| Blood sugar | 179   |       |       |       |       |
| SGOT (AST) | 65    | 85    | 138   | 144   | 90    |
| SGPT (ALT) | 41    | 56    | 90    | 101   | 93    |
| Total bilirubin | 1.5 mg/dL (0.1-1.2) | 2.1 | 4.6   |       |
| CPK     |       | 340 U/L (24-195) |       |       |       |
| Troponin I |       | 7.8 ng/L (negative) |       |       |       |
| Prothrombin time (PT) |       | 14.7 s (12-14) |       |       |       |
| HBs Ag  |       | Negative |       |       |       |
| HCV Ab  |       | Negative |       |       |       |
| HIV Ab  |       | Negative |       |       |       |
| Urine protein | 3+ |       |       |       |       |
| Urine bacteria | Moderate |       |       |       |       |
DATA AVAILABILITY STATEMENT

All data underlying the results are available as part of the article, and no additional source data are required.

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