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CASE SERIES

Heterogeneous clinical spectrum of severe methanol poisoning in the intensive care unit: a case series from full recovery to brain death

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ABSTRACT

Methanol, a toxic substance, has been identified as a causative agent for lethal poisoning. The present case series includes nine patients admitted to the intensive care unit (ICU) due to methanol poisoning between 25th November 2024 and 13th January 2025. The median age of the patients was 49 (43-54) years, with eight of them being male. All patients exhibited high anion gap metabolic acidosis and underwent renal replacement therapy. The majority of the patients received a combination of ethanol and folinic acid therapies. It is noteworthy that a third of patients experienced a cardiopulmonary arrest either in their own homes or in the immediate period following their admission to the hospital. Six patients survived to ICU discharge: four were transferred to hospital wards or discharged from the hospital, and two were transferred to palliative care units. One patient died shortly after ICU admission, another died during prolonged ICU follow-up, and one was diagnosed with brain death and subsequently underwent organ procurement surgery. In conclusion, a deteriorated neurological state, receipt of cardiopulmonary resuscitation, and the presence of pathological findings in radiological imaging of the central nervous system, cardiac injury, vasopressor requirement, sepsis, and ventilator-associated pneumonia were likely to be associated with an increased risk of poor outcomes.

Keywords: acidosis, alcohols, methanol, poisoning, toxicity

Introduction

Methanol, a chemical compound with a wide range of industrial applications as a solvent (1), has been identified as a causative agent of lethal poisoning (2). Even relatively minor ingestions have the potential to induce severe toxicity (3). Documented cases of inhalational and dermal toxicity have also been reported (2). However, consumption of unlabeled or home-made alcohol-based beverages has also been identified as a risk factor for methanol poisoning (1).

The underlying mechanism of methanol poisoning is associated with its acid metabolites, particularly formate. This association has been linked to a range of serious health consequences, including retinal damage with optic disc edema and/or hyperemia, ischemic or hemorrhagic damage in the basal ganglia, particularly in the putamen, and white matter of the insular subcortex and renal toxicity (1,4,5). These changes manifest with a range of complaints, including central nervous system (CNS) suppression, blurred vision, blindness, seizures, coma and acute kidney failure (1,3-6). The diagnosis of patients with methanol poisoning is based on strong suspicion (1-3). The presence of a persistent metabolic acidosis with a high anion gap, a large osmolal gap and impaired kidney function are the additional clinical conditions (3). Despite the implementation of

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interventions such as ethanol or fomepizole to inhibit alcohol dehydrogenase (ADH), and extracorporeal treatments, the prognosis for patients with methanol poisoning remains uncertain (3). The mortality rate associated with methanol poisoning ranges from 16% to 48% in the literature (7,8).

In this case series, we present the clinical characteristics of nine patients with methanol poisoning. The patients exhibited comparable clinical scenarios, yet their outcomes were influenced by distinct factors.

Case series

This case series examined a cohort of nine patients admitted to our center's intensive care units (ICUs) with methanol intoxication between November 25, 2024, and January 13, 2025. The median age of the patients was 49 (43–54) years, and the majority were male (88.9%), as detailed in Table 1. The median Charlson comorbidity index and the acute physiology and chronic health evaluation II scores were 0 (0–1) and 26 (15–36), respectively. The median Glasgow coma scale (GCS) score of the patients was 9 (3–15). The median duration from the onset of symptoms to hospital admission was 9 (2–39) hours. The median length of hospital and ICU stay was determined to be 8 (3–63) days and 3 (2–61) days, respectively.

Patient 1

A 53-year-old male patient was discovered unresponsive in his home environment by his family members and was administered cardiopulmonary resuscitation. The patient was intubated by the ambulance crew and transferred to the emergency department. On admission, ventricular fibrillation was detected. Subsequent to the defibrillation, the cardiac massage was continued for a further duration of 30 minutes. Following the return of spontaneous circulation, the infusion of noradrenaline was initiated in order to treat refractory hypotension. Coronary angiography was performed with a preliminary diagnosis of acute coronary syndrome, but coronary perfusion was found to be normal. The results of the blood gas analysis indicated the presence of high anion gap metabolic acidosis (Table 2). In addition to the findings from the laboratory, the patient was primarily suspected of methanol poisoning, as family members reported that the patient had consumed alcoholic-based homemade beverages. Subsequently, a loading dose of 4 mL/kg of 20% ethanol was administered orally, followed by a maintenance infusion at a rate of 0.8 mL/kg/hour for a period of 72 hours. Additionally, intravenous sodium bicarbonate infusion initiated a rate of 1 mEq/kg. Following a two-hour session of conventional

Table 1. Patient characteristics.

Characteristics	All (n = 9)	Patients								
		I	II	III	IV	V	VI	VII	VIII	IX
Age, years	49 (43–54)	53	39	55	48	49	43	50	42	57
Sex, male	8 (88.9)	Male	Male	Male	Male	Female	Male	Male	Male	Male
APACHE II score[a]	26 (15–36)	36	3	9	25	20	26	42	35	27
CCI	0 (0–1)	1	0	1	0	0	0	1	0	1
GCS[a]	9 (3–15)	3	14	15	14	15	3	3	4	9
Length of duration										
First symptom to hospital admission, hours	9 (2–39)	N/A	48	6	1	48	12	1	4	12
Hospital stay, days	8 (3–63)	263	2	66	3	8	4	59[b]	1	28
ICU stay, days	3 (2–61)	263	2	62	2	2	4	59	1	3

All values are expressed as numbers (percentages) or median (interquartile range).
APACHE II, Acute Physiology and Chronic Health Evaluation II; CCI, Charlson Comorbidity Index; GCS, Glasgow Coma Scale; N/A, not applicable.
[a]Calculated on the day of hospital admission.
[b]The patient was transferred to a palliative care unit in another center.

Table 2. Laboratory data of the patients.

Laboratory test	Alla (n = 9)	Patients								
		I	II	III	IV	V	VI	VII	VIII	IX
BUN, mg/dL	14.4 (12.1–16.7)	13.5	14.4	14.4	18.0	6.0	10.7	25.2	15.4	13.5
Creatinine, mg/dL	1.21 (1.02–1.52)	1.10	1.09	0.95	1.21	0.74	1.42	2.00	1.60	1.44
Na, mmol/L	140.0 (138.8–142.0)	139.0	141.0	139.0	140.0	144.0	143.0	137.6	140.0	138.5
Cl, mmol/L	110 (108–112)	110	109	113	105	108	108	111	111	114
K, mmol/L	4.87 (3.87–5.50)	3.46	4.87	5.53	4.37	5.35	3.75	7.79	3.98	5.47
Serum Ethanol, mg/dL	N/A	58.4	< 10	< 10	< 10	< 10	< 10	< 10	12.2	< 10
Blood gas analysis										
pH	7.04 (6.74–7.12)	7.04	7.15	7.15	7.05	7.08	6.78	6.7	6.94	6.7
PCO_2, mmHg	30.2 (26.7–34.9)	35.8	27.3	23.7	33.9	26.2	27.1	30.2	34.0	41.3
PaO_2, mmHg	159 (112–269)	191	144	198	159	82	339	482	101	122
HCO_3, mmol/L	9.4 (4.6–10.6)	9.7	10.9	11.1	10.3	9.4	3.9	4.2	7.5	5.0
BE	-21.5 (-31.1–-20.7)	-20.8	-19.7	-20.6	-21.2	-21.5	-31.0	-32.4	-26.5	-31.2
Lactate, mmol/L	6.9 (1.3–8.5)	11.1	0.9	0.2	7.5	1.7	9.4	7.3	2.8	6.9
SO_2, %	98 (96–99)	98	98	98	99	96	99	100	96	94
PaO_2 / FiO_2	328 (310–361)	202	342	301	318	380	335	441	318	328
Anion gap	29.1 (23.9–32.3)	22.8	26.0	20.4	29.1	32.0	34.9	30.2	32.5	25.0

All values are presented as median and interquartile range or numbers.
BUN, blood urea nitrogen; N/A, not applicable; PCO_2, partial pressure of carbon dioxide; PaO_2, partial pressure of oxygen; BE, base excess; SO_2, oxygen saturation; FiO_2, fraction of inspired oxygen.
[a]Calculated based on laboratory test results performed on hospital admission.

hemodialysis in the emergency department, the patient was transferred to the intensive care unit (ICU) to initiate continuous renal replacement therapy (CRRT), in addition to other supportive therapies. Furthermore, folinic acid (4 x 50 mg) was prescribed for three days. However, fomepizole therapy was not administered due to a lack of availability. The metabolic abnormalities were resolved after several days. However, CRRT was administered for a duration of 360 hours due to acute renal failure. The patient's unconsciousness was attributed to hypoxic-ischemic encephalopathy, as evidenced by cerebral magnetic resonance imaging (MRI). The patient also suffered from ventilator-associated pneumonia (VAP), sepsis, and cardiac injury. The patient underwent a tracheostomy procedure and was provided with a home-type mechanical ventilator. A percutaneous endoscopic gastrostomy (PEG) tube was inserted for the purpose of enteral feeding. However, the patient died on the 263rd day of hospital admission due to septic shock.

Patient 2

A 39-year-old male patient was admitted to hospital with complaints of nausea, vomiting and blurred vision. On admission, the Glasgow Coma Scale (GCS) score was 14. It was reported by the patient's family that his mental state had deteriorated since the onset of symptoms, and that he had consumed large quantities of alcoholic, homemade beverages over the previous two days. The results of the laboratory tests indicated the presence of two key pathologies: high anion gap metabolic acidosis and acute kidney injury. Following a two-hour session of conventional hemodialysis in the emergency department, the patient was transferred to the ICU. Furthermore, sodium bicarbonate infusion was administered. In the intensive care unit, metabolic abnormalities were

resolved after the initiation of several hours of CRRT. The patient was discharged from hospital on the second day of ICU admission.

Patient 3

A 55-year-old male patient diagnosed with hypertension was admitted to the emergency department with complaints of nausea, vomiting, confusion, and blurred vision. It was reported that the patient consumed homemade alcohol-based beverages six hours prior to admission. On admission, the GCS score was 15. The laboratory tests revealed a high anion gap metabolic acidosis reflecting methanol poisoning. The patient was subsequently transferred to the ICU. On ICU admission, the GCS score was documented as 11. Intravenous 10% ethanol was initiated with a loading dose of 10 mL/kg over 60-minute period, followed by a maintenance dose of 1 mL/kg per hour for a duration of 72 hours. Furthermore, the patient received a sodium bicarbonate infusion at a dose of 1 mEq/kg for a duration of 10 hours. Following the administration of CRRT, the patient required noradrenaline infusion due to hypotension. Following a period of several hours, there was a deterioration in the patient's mental state, which resulted in a decrease in the GCS score to 7. The patient was intubated to ensure optimal airway safety. The cerebral diffusion MRI revealed restricted perfusion in bilateral supratentorial and infratentorial areas. The therapeutic approach of anti-antiaggregant therapy was initiated in the management of acute ischemic cerebrovascular disease. It was observed that by the third day of admission to the intensive care unit (ICU), metabolic abnormalities had been resolved. However, CRRT was required to be administered for a duration of 432 hours due to anuria. During the patient's hospitalization period in ICU, sepsis, cardiac injury, and VAP were observed as complications. The patient underwent a tracheostomy procedure and was provided with a home-type mechanical ventilator. A PEG tube was inserted for the purpose of enteral feeding. Following a period of 62 days in the intensive care unit, the patient was discharged to the palliative care unit.

Patient 4

A 48-year-old male patient was admitted to hospital with symptoms of chest pain, and paresthesia in the upper limbs. On admission, the GCS score was 14. The laboratory tests revealed high anion gap metabolic acidosis. As reported by the patient's family, the patient had consumed alcohol-based beverages, prepared in the home, a few hours prior to admission. In the emergency department the patient was intubated as a consequence of a loss of consciousness. Following a two-hour hemodialysis session, the patient was transferred to the ICU. The intravenous administration of 10% ethanol was initiated with a loading dose of 10 mL/kg over a 60-minute period, followed by a maintenance dose of 1 mL/kg per hour for a duration of 48 hours. Furthermore, the patient received a sodium bicarbonate infusion at a dose of 1 mEq/kg for a duration of 6 hours and folinic acid therapy (4 x 50 mg, for three days). The metabolic abnormalities were resolved with no requirement for additional CRRT. On the second day of admission to the ICU, the patient was extubated and subsequently transferred to a ward one day later.

Patient 5

A 49-year-old female patient suffering from hypertension was admitted to the emergency department, presenting with symptoms including headache and blurred vision. The onset of these symptoms was reported to have occurred 48 hours prior, following the consumption of homemade alcohol-based beverages. At the time of admission, the GCS score was recorded at 15. The laboratory test revealed a high anion gap metabolic acidosis, indicative of methanol poisoning. The sodium bicarbonate infusion was initiated with an initial dose of 1 mEq/kg, with the infusion continuing for a duration of 6 hours. Following a two-hour session of conventional hemodialysis, the patient was transferred to the ICU. In the ICU, a loading dose of 4 mL/kg of 20% ethanol was administered orally, followed by a maintenance infusion at a rate of 0.8 mL/kg/hour for a period of 24 hours. Furthermore, the patient received folinic acid

therapy 50 mg every six hours for three days. The therapeutic interventions previously mentioned have been shown to be effective in resolving metabolic abnormalities, thereby negating the necessity for CRRT. On the second day of admission to the ICU, the patient was discharged from the ICU to the internal medicine ward.

Patient 6

A 43-year-old male patient was admitted to the hospital after presenting with symptoms including vomiting, dizziness, impaired speech, and loss of consciousness following the consumption of homemade alcohol-based beverages twelve hours prior. The patient had a medical history of hypertension, hyperlipidemia, and atrial arrhythmia. The patient was intubated in view of a deteriorated neurological state. The blood gas analysis revealed a high anion gap metabolic acidosis indicative of methanol poisoning. A sodium bicarbonate infusion was initiated with a dose of 1 mEq/kg. Concurrently, during the patient's conventional hemodialysis treatment, a generalized tonic-clonic seizure manifested. The seizure was successfully managed by the administration of antiepileptic medication, and hemodialysis therapy was interrupted during the first hour of the session. Following the conducting of a cerebral computed tomography (CT) scan, the patient was transferred to the ICU. A neurological examination was conducted on the patient upon their admission to the ICU, and it was found that the patient lacked a pupillary light reflex. The CT scan revealed a widespread cerebral oedema. The neurosurgical team determined that the patient was not suitable for surgical intervention. The enteral solution of 20% ethanol was administered at a dose of 4 ml/kg, with a subsequent maintenance dose of 0.5 ml/kg/h over a 48-hour period. In order to address metabolic acidosis, the CRRT was initiated. A neurological examination conducted in the 72nd hour of ICU follow-up revealed the absence of brain stem reflexes. A subsequent apnea test resulted in a diagnosis of brain death. A contrast-enhanced CT cerebral angiography revealed a lack of cerebral perfusion. The patient underwent a surgical procedure involving the donation of a liver, heart and kidneys on the 4th day of admission to the ICU.

Patient 7

A 50-year-old male patient suffering from hypertension was found unresponsive and was taken to hospital by his family. Following admission, the neurological examination revealed the presence of dilated pupils with no light reflex, and a GCS score of 3. The patient was intubated for the aforementioned reasons. As reported by the patient's family, the patient was engaged in the production of alcoholic beverages within the domestic environment. In addition, empty bottles of homemade alcoholic beverages were discovered in the immediate vicinity of the patient. The laboratory tests revealed a high anion gap metabolic acidosis. The infusion of sodium bicarbonate was initiated at a rate of 1 mEq/kg intravenously. The patient underwent conventional hemodialysis. However, the patient suffered from cardiopulmonary arrest during hemodialysis therapy. The patient was transferred to the ICU after the ROSC within a period of 9 minutes cardiopulmonary resuscitation. After ROSC the patient also required vasopressor support. In the ICU, the CRRT was initiated. Furthermore, intravenous 10% ethanol therapy was initiated, with a loading dose of 10 mL/kg over a 60-minute period, followed by a maintenance dose of 1 mL/kg per hour for a duration of 48 hours. Additionally, folinic acid therapy was administered for 50 mg every six hours for three days. On the 2nd day of ICU admission, the patient exhibited myoclonic jerks that were successfully controlled by antiepileptic medications. However, subsequent MRI scan results revealed restricted diffusion in both cerebral hemispheres, in all lobes, in cortico-subcortical regions and basal ganglia, in the posterior fossa, in both cerebellar regions, and microbleeds in bilateral basal ganglia. Subsequent to the passage of several days, the metabolic abnormalities were resolved. However, the patient received CRRT for a period of ten days due to anuria. During the patient's stay in the ICU, additional complications were

observed, including cardiac injury, sepsis, and VAP. Consequently, the patient underwent tracheostomy and PEG procedures due to a poor neurological state and was discharged to the palliative care unit with home type MV support on the 59th day of hospital admission.

Patient 8

A 42-year-old male patient was transferred to the emergency department by his family, exhibiting symptoms that included a loss of consciousness subsequent to the ingestion of domestically prepared alcoholic beverages. Upon admission, the GCS was recorded as 4, and the patient underwent cardiopulmonary resuscitation for a duration of 5 minutes. After ROSC, the patient required vasopressor support with noradrenaline infusion at a rate of 0.4 mcg/kg/min. The laboratory tests revealed a high anion gap metabolic acidosis. In the emergency department, intravenous sodium bicarbonate infusion was initiated at a rate of 2 mEq/kg. Following a two-hour session of conventional hemodialysis, the patient was transferred to the ICU. The CRRT therapy was initiated in the ICU. In addition, folinic acid therapy was prescribed 50 mg every six hours. Despite the interventions undertaken, the patient died several hours after being admitted to the ICU due to refractory hypotension and metabolic acidosis that did not improve despite all supportive treatments, prior to the administration of intravenous or enteral ethanol therapy.

Patient 9

A 57-year-old male patient was transferred to the emergency department by ambulance, reporting a complaint of confusion and vision loss. On admission, the GCS score was documented as 9. However, the patient was intubated due to the neurological deterioration half an hour later. The ambulance crew reported finding empty bottles in the patient's environment that could potentially be linked to homemade alcohol-based beverages. The laboratory test revealed a high anion gap metabolic acidosis, indicating methanol poisoning. An infusion of sodium bicarbonate was initiated at a rate of 1 mEq/kg. Following a two-hour session of conventional hemodialysis, the patient was transferred to the ICU. In the ICU, the intravenous administration of 10% ethanol was initiated with a loading dose of 10 mL/kg, followed by a maintenance dose of 1 mL/kg/hour for a period of three days. Furthermore, folinic acid was prescribed for a period of three days (4 x 50 mg). Following a 24-hour period of ICU admission, the patient was successfully weaned from mechanical ventilation. Furthermore, the CRRT was terminated on the third day of ICU admission. Nevertheless, the patient continued to exhibit symptoms of oliguria. The patient was transferred to the nephrology ward two days after weaning, with a GCS score of 15.

Written informed consent for publication of this case series was obtained from the patients or their next of kin.

Discussion

The present case series, which analyzes a cohort of nine patients treated in the ICU due to methanol poisoning, reveals that four of the survivors were discharged from the ICU with normal neurological status and kidney function. However, two of the patients exhibited poor neurological outcomes, and one patient died immediately after ICU admission without achieving further treatment modalities and radiological analysis, while another in the late period of ICU follow-up. Notably, one patient was diagnosed with brain death and underwent kidney, heart, and liver donation surgery.

In patients admitted to the hospital with methanol poisoning, Kussmaul breathing (6) and gastrointestinal symptoms, including nausea, vomiting, and abdominal pain, are often the initial manifestations (1,8). Furthermore, patients may present with symptoms of CNS suppression, including confusion, drowsiness, seizures, coma, and visual abnormalities, such as diplopia, photophobia, blurred vision (1-3,6,8), early or late blindness, and nystagmus (1-3).

Pupillary mydriasis with delayed or no response to light is another manifestation (1,8). The most prevalent presentations in this case series were those of CNS suppression and visual abnormalities, which were associated with severe intoxication (3).

The diagnosis of patients with methanol poisoning is largely dependent upon a high degree of suspicion or clear evidence of ingestion (1-3). Laboratory findings, specifically blood gas analysis indicating acidosis with a high anion gap, enlighten to the diagnosis (1,2). Increased levels of lactate may also contribute to the extension of anion gap (2). Impaired kidney function tests or elevated osmolal gap levels may also support the diagnosis (1-3). However, the direct measurement of serum methanol or formate levels, which is based on gas chromatographic analysis, is important for rapid clinical decision-making. Unfortunately, these measures are not widely available in many healthcare facilities (1,9). In the present case series, the ingestion of home-made alcohol-based beverages was confirmed by patients, family members, or patient's friends. Blood gas analysis indicated a high anion gap metabolic acidosis for all patients on hospital admission and acute kidney injury was present for all patients on admission or during their hospital stay. The direct measurement of serum methanol level is only available as a "send out-test" in our center, and it is not suitable for the rapid clinical decision-making. Furthermore, at the time of admission of these patients in this case series, clinicians had a clear perception of a multi-victim methanol poisoning outbreak. This perception also facilitated the diagnosis of methanol poisoning.

The initial treatment of patients with methanol poisoning should prioritize airway safety (1,2). Ensuring a brief apneic phase during rapid sequence intubation, particularly in patients with severe metabolic acidosis, may reduce the risk of complications related to intubation (10). The treatment of hypotension involves the administration of crystalloids (1,2) and/or vasopressors (2). It is noteworthy that two-thirds of the patients in the present case series required invasive mechanical ventilation and almost as many vasopressor requirements. The primary treatment strategy for patients with methanol poisoning involves the inhibition of ADH by fomepizole or ethanol to prevent further metabolism of methanol to formate (1,2,8). However, due to unavailability of fomepizole, this treatment option could not be provided to the patients in the present case series. Nevertheless, ethanol treatment was administered intravenously or enterally to the patients. However, a study analyzing risk factors for the poor outcomes in patients with methanol poisoning revealed that fomepizole therapy is more effective than ethanol therapy among these patients (10). Notably, *patient II* did not require ethanol therapy after conventional HD, while *patient VIII* died immediately after admission without receiving ethanol therapy. Consequently, healthcare facilities should be adequately prepared to administer ethanol or fomepizole therapies at the earliest stages of patient admissions, particularly during periods of multi-victim poisoning outbreaks. In patients suffering from methanol poisoning, bicarbonate facilitates the process of formate dehydronation, thereby enabling excretion in the urine rather than penetration into end-organ tissues (1,2). In a similar manner, folinic acid administration has been shown to enhance formate metabolism to carbon dioxide and water (1,2,8). In the present case series, all patients received bicarbonate therapy, while folinic acid therapy could not be administered to four of them. We hypothesized that the efficacy of folinic acid therapy would be limited due to the critical condition of *patient VIII*, who did not receive this treatment. However, *patient VI*, another patient who did not receive folinic acid therapy and was diagnosed with brain death, provides an exception to this observation. Continuous renal replacement or conventional HD therapies are the optimal treatment options for the elimination of methanol and its metabolites. The administration of extracorporeal therapies is recommended in cases of severe poisoning, characterized by symptoms such as coma, seizures, visual abnormalities, and metabolic acidosis with a pH <7.15, an anion gap

>24 mmol/L, or persistent acidosis despite adequate therapy or antidotes. Furthermore, in the context of fomepizole and ethanol therapies, serum methanol concentrations of >700 mg/L (21.8 mmol/L) and >600 mg/L (18.7 mmol/L) are considered decision thresholds for the initiation of HD administration, respectively. However, it is important to note that plasma exchange, hemoperfusion, and peritoneal dialysis are not recommended since these treatment modalities have been demonstrated to be ineffective for the clearance of methanol and its metabolites. Furthermore, clinical improvement or methanol concentration <200 mg/L (6.2 mmol/L) are considered decision points for discontinuing extracorporeal therapies (3).

A comprehensive review of the studies analyzing patients with methanol poisoning reveals that high osmolal or anion gap (11), low levels of pH (10,11), inadequate respiratory compensation of metabolic acidosis (10), respiratory arrest (12), low levels of GCS score (13), presence of coma (10), hypothermia (13), hyperglycemia (12), increased levels of serum creatinine (13), and length of duration from methanol intake to hospital admission (12) have been identified as risk factors associated with poor outcomes or mortality. In the present case series, a deteriorated neurological state, and/or low GCS score on admission, undergoing cardiopulmonary resuscitation prior or after admission, and the presence of pathological findings in radiological imaging of the CNS are likely to be associated with an increased risk of mortality and poor neurological outcome. Furthermore, major events such as cardiac injury, vasopressor requirement, sepsis, and ventilator-associated pneumonia might be additional risk factors.

Ethical approval

Written informed consent for publication of this case series was obtained from the patients or their legal representatives.

Author contribution

Study conception and design: MNY, OD, MA; data collection: FOD, BB, ÖA, BK, CY, OA; analysis and interpretation of results: MNY, OD, MA; draft manuscript preparation: FOD, MNY; critical review of the manuscript for important intellectual content: FOD, BB, ÖA, OD, BK, CY, OA, MA. All authors approved the final version for publication and agreed to be accountable for all aspects of the work, including ensuring that any questions regarding its accuracy or integrity are appropriately investigated and resolved.

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Conflict of interest

The authors declare that there is no conflict of interest.

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