

World Journal of *Clinical Cases*

World J Clin Cases 2022 July 6; 10(19): 6341-6758



Contents

Thrice Monthly Volume 10 Number 19 July 6, 2022

MINIREVIEWS

- 6341** Review of clinical characteristics, immune responses and regulatory mechanisms of hepatitis E-associated liver failure
Chen C, Zhang SY, Chen L
- 6349** Current guidelines for *Helicobacter pylori* treatment in East Asia 2022: Differences among China, Japan, and South Korea
Cho JH, Jin SY
- 6360** Review of epidermal growth factor receptor-tyrosine kinase inhibitors administration to non-small-cell lung cancer patients undergoing hemodialysis
Lan CC, Hsieh PC, Huang CY, Yang MC, Su WL, Wu CW, Wu YK

ORIGINAL ARTICLE

Case Control Study

- 6370** Pregnancy-related psychopathology: A comparison between pre-COVID-19 and COVID-19-related social restriction periods
Chieffo D, Avallone C, Serio A, Kotzalidis GD, Balocchi M, De Luca I, Hirsch D, Gonzalez del Castillo A, Lanzotti P, Marano G, Rinaldi L, Lanzone A, Mercuri E, Mazza M, Sani G
- 6385** Intestinal mucosal barrier in functional constipation: Does it change?
Wang JK, Wei W, Zhao DY, Wang HF, Zhang YL, Lei JP, Yao SK

Retrospective Cohort Study

- 6399** Identification of risk factors for surgical site infection after type II and type III tibial pilon fracture surgery
Hu H, Zhang J, Xie XG, Dai YK, Huang X

Retrospective Study

- 6406** Total knee arthroplasty in Ranawat II valgus deformity with enlarged femoral valgus cut angle: A new technique to achieve balanced gap
Lv SJ, Wang XJ, Huang JF, Mao Q, He BJ, Tong PJ
- 6417** Preliminary evidence in treatment of eosinophilic gastroenteritis in children: A case series
Chen Y, Sun M
- 6428** Self-made wire loop snare successfully treats gastric persimmon stone under endoscopy
Xu W, Liu XB, Li SB, Deng WP, Tong Q
- 6437** Neoadjuvant transcatheter arterial chemoembolization and systemic chemotherapy for the treatment of undifferentiated embryonal sarcoma of the liver in children
He M, Cai JB, Lai C, Mao JQ, Xiong JN, Guan ZH, Li LJ, Shu Q, Ying MD, Wang JH

- 6446** Effect of cold snare polypectomy for small colorectal polyps

Meng QQ, Rao M, Gao PJ

- 6456** Field evaluation of COVID-19 rapid antigen test: Are rapid antigen tests less reliable among the elderly?

Tabain I, Cucevic D, Skreb N, Mrzljak A, Ferencak I, Hruskar Z, Misic A, Kuzle J, Skoda AM, Jankovic H, Vilibic-Cavlek T

Observational Study

- 6464** Tracheobronchial intubation using flexible bronchoscopy in children with Pierre Robin sequence: Nursing considerations for complications

Ye YL, Zhang CF, Xu LZ, Fan HF, Peng JZ, Lu G, Hu XY

- 6472** Family relationship of nurses in COVID-19 pandemic: A qualitative study

Çelik MY, Kiliç M

META-ANALYSIS

- 6483** Diagnostic accuracy of ≥ 16 -slice spiral computed tomography for local staging of colon cancer: A systematic review and meta-analysis

Liu D, Sun LM, Liang JH, Song L, Liu XP

CASE REPORT

- 6496** Delayed-onset endophthalmitis associated with *Achromobacter* species developed in acute form several months after cataract surgery: Three case reports

Kim TH, Lee SJ, Nam KY

- 6501** Sustained dialysis with misplaced peritoneal dialysis catheter outside peritoneum: A case report

Shen QQ, Behera TR, Chen LL, Attia D, Han F

- 6507** Arteriovenous thrombotic events in a patient with advanced lung cancer following bevacizumab plus chemotherapy: A case report

Kong Y, Xu XC, Hong L

- 6514** Endoscopic ultrasound radiofrequency ablation of pancreatic insulinoma in elderly patients: Three case reports

Rossi G, Petrone MC, Capurso G, Partelli S, Falconi M, Arcidiacono PG

- 6520** Acute choroidal involvement in lupus nephritis: A case report and review of literature

Yao Y, Wang HX, Liu LW, Ding YL, Sheng JE, Deng XH, Liu B

- 6529** Triple A syndrome-related achalasia treated by per-oral endoscopic myotomy: Three case reports

Liu FC, Feng YL, Yang AM, Guo T

- 6536** Choroidal thickening with serous retinal detachment in BRAF/MEK inhibitor-induced uveitis: A case report

Kiraly P, Groznik AL, Valentinčič NV, Mekjavić PJ, Urbančič M, Ocvirk J, Mesti T

- 6543** Esophageal granular cell tumor: A case report

Chen YL, Zhou J, Yu HL

- 6548** Hem-o-lok clip migration to the common bile duct after laparoscopic common bile duct exploration: A case report
Liu DR, Wu JH, Shi JT, Zhu HB, Li C
- 6555** Chidamide and sintilimab combination in diffuse large B-cell lymphoma progressing after chimeric antigen receptor T therapy
Hao YY, Chen PP, Yuan XG, Zhao AQ, Liang Y, Liu H, Qian WB
- 6563** Relapsing polychondritis with isolated tracheobronchial involvement complicated with Sjogren's syndrome: A case report
Chen JY, Li XY, Zong C
- 6571** Acute methanol poisoning with bilateral diffuse cerebral hemorrhage: A case report
Li J, Feng ZJ, Liu L, Ma YJ
- 6580** Immunoabsorption therapy for Klinefelter syndrome with antiphospholipid syndrome in a patient: A case report
Song Y, Xiao YZ, Wang C, Du R
- 6587** Roxadustat for treatment of anemia in a cancer patient with end-stage renal disease: A case report
Zhou QQ, Li J, Liu B, Wang CL
- 6595** Imaging-based diagnosis for extraskeletal Ewing sarcoma in pediatrics: A case report
Chen ZH, Guo HQ, Chen JJ, Zhang Y, Zhao L
- 6602** Unusual course of congenital complete heart block in an adult: A case report
Su LN, Wu MY, Cui YX, Lee CY, Song JX, Chen H
- 6609** Penile metastasis from rectal carcinoma: A case report
Sun JJ, Zhang SY, Tian JJ, Jin BY
- 6617** Isolated cryptococcal osteomyelitis of the ulna in an immunocompetent patient: A case report
Ma JL, Liao L, Wan T, Yang FC
- 6626** Magnetic resonance imaging features of intrahepatic extramedullary hematopoiesis: Three case reports
Luo M, Chen JW, Xie CM
- 6636** Giant retroperitoneal liposarcoma treated with radical conservative surgery: A case report and review of literature
Lieto E, Cardella F, Erario S, Del Sorbo G, Reginelli A, Galizia G, Urraro F, Panarese I, Auricchio A
- 6647** Transplanted kidney loss during colorectal cancer chemotherapy: A case report
Pośpiech M, Kolonko A, Nieszporek T, Kozak S, Kozaczka A, Karkoszka H, Winder M, Chudek J
- 6656** Massive gastrointestinal bleeding after endoscopic rubber band ligation of internal hemorrhoids: A case report
Jiang YD, Liu Y, Wu JD, Li GP, Liu J, Hou XH, Song J

- 6664** Mills' syndrome is a unique entity of upper motor neuron disease with N-shaped progression: Three case reports
Zhang ZY, Ouyang ZY, Zhao GH, Fang JJ
- 6672** Entire process of electrocardiogram recording of Wellens syndrome: A case report
Tang N, Li YH, Kang L, Li R, Chu QM
- 6679** Retroperitoneal tumor finally diagnosed as a bronchogenic cyst: A case report and review of literature
Gong YY, Qian X, Liang B, Jiang MD, Liu J, Tao X, Luo J, Liu HJ, Feng YG
- 6688** Successful treatment of Morbihan disease with total glucosides of paeony: A case report
Zhou LF, Lu R
- 6695** Ant sting-induced whole-body pustules in an inebriated male: A case report
Chen SQ, Yang T, Lan LF, Chen XM, Huang DB, Zeng ZL, Ye XY, Wan CL, Li LN
- 6702** Plastic surgery for giant metastatic endometrioid adenocarcinoma in the abdominal wall: A case report and review of literature
Wang JY, Wang ZQ, Liang SC, Li GX, Shi JL, Wang JL
- 6710** Delayed-release oral mesalamine tablet mimicking a small jejunal gastrointestinal stromal tumor: A case report
Frosio F, Rausa E, Marra P, Boutron-Ruault MC, Lucianetti A
- 6716** Concurrent alcoholic cirrhosis and malignant peritoneal mesothelioma in a patient: A case report
Liu L, Zhu XY, Zong WJ, Chu CL, Zhu JY, Shen XJ
- 6722** Two smoking-related lesions in the same pulmonary lobe of squamous cell carcinoma and pulmonary Langerhans cell histiocytosis: A case report
Gencer A, Ozcibik G, Karakas FG, Sarbay I, Batur S, Borekci S, Turna A
- 6728** Proprotein convertase subtilisin/kexin type 9 inhibitor non responses in an adult with a history of coronary revascularization: A case report
Yang L, Xiao YY, Shao L, Ouyang CS, Hu Y, Li B, Lei LF, Wang H
- 6736** Multimodal imaging study of lipemia retinalis with diabetic retinopathy: A case report
Zhang SJ, Yan ZY, Yuan LF, Wang YH, Wang LF
- 6744** Primary squamous cell carcinoma of the liver: A case report
Kang LM, Yu DP, Zheng Y, Zhou YH
- 6750** Tumor-to-tumor metastasis of clear cell renal cell carcinoma to contralateral synchronous pheochromocytoma: A case report
Wen HY, Hou J, Zeng H, Zhou Q, Chen N

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RESPONSIBLE EDITORS FOR THIS ISSUE

Production Editor: *Xu Guo*; Production Department Director: *Xiang Li*; Editorial Office Director: *Jin-Lei Wang*.

NAME OF JOURNAL

World Journal of Clinical Cases

ISSN

ISSN 2307-8960 (online)

LAUNCH DATE

April 16, 2013

FREQUENCY

Thrice Monthly

EDITORS-IN-CHIEF

Bao-Gan Peng, Maurizio Serati, George Kontogeorgos, Jerzy Tadeusz Chudek, Ja Hyeon Ku

EDITORIAL BOARD MEMBERS

<https://www.wjgnet.com/2307-8960/editorialboard.htm>

PUBLICATION DATE

July 6, 2022

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INSTRUCTIONS TO AUTHORS

<https://www.wjgnet.com/bpg/gerinfo/204>

GUIDELINES FOR ETHICS DOCUMENTS

<https://www.wjgnet.com/bpg/GerInfo/287>

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<https://www.wjgnet.com/bpg/gerinfo/240>

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<https://www.wjgnet.com/bpg/GerInfo/288>

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<https://www.wjgnet.com/bpg/gerinfo/208>

ARTICLE PROCESSING CHARGE

<https://www.wjgnet.com/bpg/gerinfo/242>

STEPS FOR SUBMITTING MANUSCRIPTS

<https://www.wjgnet.com/bpg/GerInfo/239>

ONLINE SUBMISSION

<https://www.f6publishing.com>



Acute methanol poisoning with bilateral diffuse cerebral hemorrhage: A case report

Jin Li, Zhi-Juan Feng, Lei Liu, Yu-Jie Ma

Specialty type: Critical care medicine

Provenance and peer review: Unsolicited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's scientific quality classification

Grade A (Excellent): 0
Grade B (Very good): B
Grade C (Good): C, C
Grade D (Fair): 0
Grade E (Poor): 0

P-Reviewer: Eizadi-Mood N, Iran; Gokce E, Turkey; Vlachopoulos G, Greece

Received: October 29, 2021

Peer-review started: October 29, 2021

First decision: March 23, 2022

Revised: April 4, 2022

Accepted: May 5, 2022

Article in press: May 5, 2022

Published online: July 6, 2022



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Abstract

BACKGROUND

Acute methanol poisoning (AMP) is a systemic disease that mainly affects the central nervous system and is characterized by ocular damage and metabolic acidosis. If appropriate treatments are inadequate or delayed, the mortality can exceed 40%. As the most serious complication, cerebral hemorrhage is rare with reported prevalence of 7%-19%.

CASE SUMMARY

A 62-year-old man drank liquor mixed with 45% methanol and 35% alcohol. His vision blurred 10 h later and he fell into coma in another 9 h. Serum toxicological tests were performed immediately, and continuous renal replacement therapy (CRRT) was carried out as the lactic acid exceeded 15 mmol/L and blood pH was 6.78. In addition, the toxicological report revealed 1300.5 µg/mL of methanol in serum and 1500.2 µg/mL in urine. After 59 h of CRRT, the methanol level decreased to 126.0 µg/mL in serum and 151.0 µg/mL in urine. However, the patient was still unconscious and his pupillary light reflex was slow. Computed tomography showed hemorrhage in the left putamen. After 16 d of life support treatment, putamen hemorrhage developed into diffuse symmetric intracerebral hemorrhage. In the end, his family gave up and the patient was discharged, and died in a local hospital.

CONCLUSION

Cerebral hemorrhage requires constant vigilance during the full course of treatment for severe cases of AMP.

Key Words: Acute methanol poisoning; Cerebral hemorrhage; Toxicity; Hemodialysis; Case report

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Core Tip: We describe a case of a 62-year-old man who drank liquor mixed with 45% methanol and 35% alcohol, and the serum methanol level was almost 2.6 times that of the recommended indication for hemodialysis even at 24 h after drinking. It was encouraging that his vital signs tended to be stable and methanol level dropped sharply at 48 h after hemodialysis and necessary life support treatment. Unfortunately, putamen hemorrhage occurred 16 d after the treatments and progressed to bilateral symmetric diffuse cerebral hemorrhage. His family gave up further treatment, and the patient died eventually.

Citation: Li J, Feng ZJ, Liu L, Ma YJ. Acute methanol poisoning with bilateral diffuse cerebral hemorrhage: A case report. *World J Clin Cases* 2022; 10(19): 6571-6579

URL: <https://www.wjgnet.com/2307-8960/full/v10/i19/6571.htm>

DOI: <https://dx.doi.org/10.12998/wjcc.v10.i19.6571>

INTRODUCTION

Methanol is a colorless volatile liquid with an alcohol odor at room temperature, and is easily soluble in water and body fluids. Its molecular weight is 32 g/mol. Methanol is mainly metabolized in the liver and can be converted to formaldehyde with the participation of alcohol dehydrogenase. Formaldehyde, with a half-life of 1-2 min, is rapidly converted to formic acid by formaldehyde dehydrogenase. Finally, formic acid is decomposed to CO₂ and water, and the half-life of formic acid has been 20 h in human body (Figure 1)[1].

Although methanol itself is not highly poisonous, its metabolites are highly toxic. Hence, the accumulation of formic acid is mainly responsible for the pathological changes of methanol poisoning. Clinical findings usually evolve over 6-24 h but can be delayed as long as 72-96 h if ethanol is co-ingested[2]. Manifestations include central nervous system (CNS) disease, ocular damage and metabolic acidosis. The lethal dose of pure methanol is estimated to be 1-2 mL/kg, but permanent blindness and death have been reported with as little as 0.1 mL/kg (6-10 mL in adults)[3]. The poisoning effects of formaldehyde and its metabolites are as follows[1,4-6]: (1) Formic acid can inhibit cytochrome oxidase and block the mitochondrial respiratory chain, which leads to histotoxic hypoxia and metabolic acidosis; (2) The accumulation of formic acid and methanol in ocular aqueous humor and ocular tissue causes selective damage to the retina and optic nerve cells, and acidosis may enhance the toxicity; and (3) The histotoxic hypoxia and metabolic acidosis also cause edema and necrotic damage to the putamen and white matter.

In the first few hours after drinking methanol, gastric lavage is recommended since methanol is rapidly absorbed with a half-life of 5 min in the gastrointestinal tract, but there is no solid evidence or studies that have examined the efficacy[1]. Sodium bicarbonate should be given intravenously. Antidotes such as ethanol or fomepizole suppress methanol metabolism by blocking ethanol dehydrogenase and folic acid accelerates the decomposition of formic acid to CO₂ and water[1,2,7-9]. However, fomepizole, which has an affinity for alcohol dehydrogenase 8000 times that of ethanol[2], is not available in China. For severe cases, the indications for hemodialysis are: significant metabolic acidosis (pH < 7.25-7.30), visual abnormalities, deterioration of vital signs despite intensive supportive care, electrolyte imbalance unresponsive to conventional therapy, or a serum methanol concentration 415.6 mmol/L (50 mg/dL). Intermittent hemodialysis (with a large-surface area dialyzer and high-flux membrane) removes toxic alcohols more rapidly than continuous renal replacement therapy (CCRT)[10, 11].

CASE PRESENTATION

Chief complaints

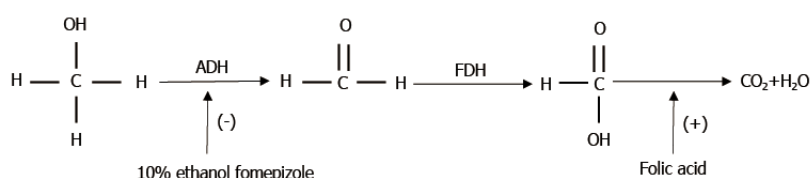
A 62-year-old man with blurred vision for 13 h and felt chest tightness and breathlessness for 5 h was sent to the emergency department of our institution by ambulance.

History of present illness

His vision blurred 10 h after drinking and he fell into coma in another 9 h. No examination was performed before he was sent to the emergency department.

History of past illness

The patient had been suffering from hypertension for > 10 years. He took nifedipine and metoprolol tartrate orally to control his blood pressure (BP) to 130/70 mmHg.



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Figure 1 Schematic diagram of methanol metabolism. ADH: Alcohol dehydrogenase; FDH: Formaldehyde dehydrogenase.

Personal and family history

The patient smoked and drank for more than 50 years. In recent years, he smoked 10 cigarettes per day and drank 100 g alcohol per day. He had no family history.

Physical examination

Basic physical examination showed that BP was 150/112 mmHg when admitted to the emergency department; his pupils were dilated and the reflection of light disappeared and neuropathological reflexes, such as the Babinski sign, were negative. Cardiac auscultation was sinus rhythm at 108 bpm and no murmur was heard. The vital signs during hospitalization are presented in [Table 1](#).

Laboratory examinations

Blood tests for myocardial enzymes, thyroid hormones, and liver and kidney function indicators were normal or slightly abnormal, which indicated hyperglycemia, hyperlipemia and renal insufficiency with serum creatinine of 139 mmol/L (normal range, 57-111 mmol/L). Coagulation function was normal, but D-dimer was 2125 ng/mL (normal range, 0-255 ng/mL). The blood gas analysis showed severe metabolic acidosis. The results of laboratory tests in the Emergency Department are presented in [Table 2](#), and the blood gas analysis until the end of the second CRRT is shown in [Table 3](#).

Imaging examinations

Computed tomography (CT) of the chest ([Figure 2A](#)) and head ([Figure 3A](#)) showed diffuse exudation in the lungs and no sign of fresh cerebral infarction or hemorrhage approximately 1 h after admission.

DISEASE PROGRESSION AND CORRESPONDING TREATMENT

During the examination in the emergency department, the patient's BP continued to drop from 155/103 mmHg to 120/85 mmHg. The emergency department physician treated him with antibiotics (ertapenem 1g + 0.9%NS 250mL IV) empirically and temporarily since CT showed diffuse exudation in the lungs. At 19:00 h, the patient got seizure with BP dropping to 58/32 mmHg and SpO₂ dropping to 50% in 2 min. Vasoactive agents (epinephrine 1 mg, atropine 0.5 mg and dopamine 6 µg/kg min) and endotracheal intubation were administered immediately and urgent consultations with physicians from neurology, nephrology and intensive care units were requested. The vital signs were stabilized. After consultation, it was agreed that the possibility of poisoning was the likeliest scenario, but CT angiography (CTA) of the pulmonary artery and aorta should be improved to exclude pulmonary embolism and aortic dissection. The patient was transferred to the intensive care unit (ICU). Meanwhile, the Toxicology Center was contacted for serological testing. CTA showed no embolism or organic change, but the exudation in lungs was significantly less than on the previous chest CT ([Figure 2B](#)). The possible explanation was neurogenic pulmonary edema, which was characterized by acute respiratory distress triggered by acute, severe compromise of the central nervous system.

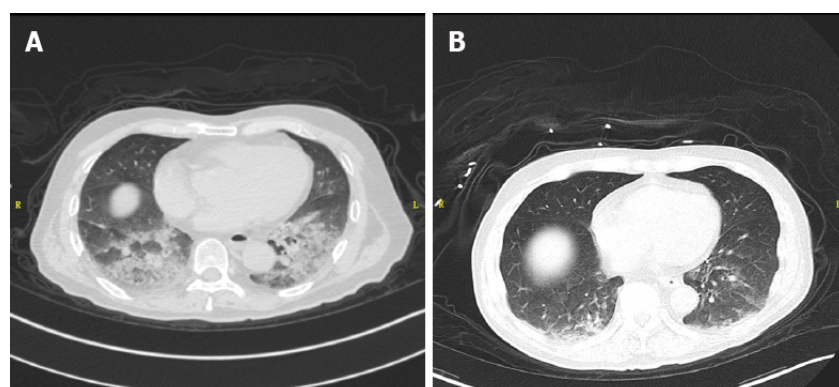
FINAL DIAGNOSIS

The Toxicology Center reported a methanol level of 1300.5 µg/mL in the serum and 1500.2 µg/mL in the urine. The patient's family recollected that the day before hospitalization, the patient drank a "medicinal liquor", a self-made mixture with Chinese herbs and liquor from an unknown source. Finally, acute methanol poisoning (AMP) was diagnosed.

Table 1 Time line and vital signs

Days (since ingestion)	Time	Events	BP (mmHg)	HR (bpm)	SpO ₂ (%)	Glasgow Coma Scale
Day 1	8:00	Blur vision				
	15:30	Chest tightness and breathless	140/100	120	99	E4V5M6 (15)
	17:00	Admission to emergency department	150/112	108	85	E2V2M4 (8)
	18:00	CT scan of chest (Figure 2A) and head (Figure 3A)	130/88	78	88	E2V2M4 (8)
	19:00	Seizure, endotracheal intubation	58/32	43	60	E1V1M1 (3)
	19:30	Toxicology test				
	21:15	CTA of pulmonary artery and aorta (Figure 2B)	92/49	117	99	E1VTM1
	21:30	Admitted to ICU	53/33	110	100	E1VTM1
	22:30	First course of CRRT (59 h)	172/90	95	98	E1VTM1
Day 2		Toxicology report confirmed AMP	140-152/70-78	60-65	96-100	E1VTM1
Day 4		Hemorrhage at left putamen (Figure 3B)	134-153/75-85	75-110	96-100	E4VTM1
		Second course of CRRT (62 h)				
Day 10		Extubation	98-123/55-78	65-125	96-100	E4V2M1
Day 19		Hemorrhage aggravated (Figure 3C)	90-130/58-80	61-75	95-99	E1V1T1(3)
Day 29		Discharge	100/64	66	99	E1VTM1

BP: Blood pressure, HR: Heart rate, SpO₂: Pulse oxygen saturation; CT: Computed tomography; ICU: Intensive care unit; CRRT: Continuous renal replacement therapy; AMP: Acute methanol poisoning.



DOI: 10.12998/wjcc.v10.i19.6571 Copyright ©The Author(s) 2022.

Figure 2 Chest computed tomography. A: Diffuse exudation in the lungs; B: Exudation in the lungs was significantly cleared up. Imaging A and B were performed at an interval of approximate 3 h for excluding pulmonary embolism and aortic dissection.

TREATMENT

After admission to ICU, the patient was still in shock, with an APACHE II score, a scoring system for estimating the risk of death for patients admitted to ICU[12], of 34 and an estimated mortality risk of 80.95%. Under this circumstance, CRRT was administered immediately to correct acidosis and electrolyte disturbance. Oral folic acid (5 mg tid for 27 d) was prescribed after AMP was diagnosed. In order to prevent cerebral hemorrhage, sodium citrate was applied in CRRT instead of heparin. After 59 h of CRRT, the vital signs were stable, and the methanol level decreased to 126.0 µg/mL in serum and 151.0 µg/mL in urine. However, the patient was still unconscious, and the pupillary light reflex was slow. Hence, we decided to discontinue the CRRT, and perform another CT scanning. Unfortunately, CT showed a 1.5 cm × 0.5 cm hemorrhage in the left putamen and multiple low-density shadows in bilateral brain parenchyma, which conformed to the characteristics of poisoning (Figure 3B). The Neurosurgery Department recommended non-surgical intervention as the hemorrhage area was limited

Table 2 Results of laboratory blood tests in the Emergency Department

Items	Results	Abnormality	Normal range
White cell count ($\times 10^9/L$)	15.23	↑	3.5-9.5
Proportion of neutrophils (%)	58.8	Normal	40-75
Hemoglobin (g/L)	202	↑	130-175
CRP	< 1	Normal	0-10
Glucose (mmol/L)	8.8	↑	3.6-6.1
Creatinine ($\mu\text{mol/L}$)	139	↑	57-111
Total Cholesterol (mmol/L)	6.71	↑	2.8-5.18
Triglyceride (mmol/L)	4.56	↑↑	0.51-1.7
ALT (U/L)	28	Normal	9-50
AST (U/L)	40	Normal	15-40
Myocardial enzyme			
Troponin I (ng/mL)	0.011	Normal	0-0.023
Creatine kinase MB isoenzyme (ng/mL)	3.6	Normal	0-7.2
Myoglobin (ng/mL)	131	↑	23-112
BNP (pg/mL)	34.6	Normal	< 100
Coagulation			
Prothrombin time (sec)	11.3	Normal	9.4-12.5
Prothrombin activity (%)	96	Normal	70-130
Thrombin time (sec)	16.3	Normal	10.3-18
Activated partial thromboplastin time (sec)	34	Normal	25.4-38.4
D-dimer	2125	↑↑	0-255
Arterial blood gas			
PH	6.797	↓↓	7.35-7.45
PaCO ₂	37.5	Normal	35-45
PaO ₂	82.5	Normal	80-100
SpO ₂	85.1	↓	95-100
Base excess (mmol/L)	-30.2	↓↓	(-3)-(-3)
HCO ₃ ⁻	4.4	↓↓	22-27
H ⁺	159.5	↑↑	35.5-44.7
A-aDO ₂ (mmHg)	29.4	↑	0-20
Lactic acid (mmol/L)	>15	↑↑	0.4-2.2

CRP: C-response protein; ALT: Alanine transaminase; AST: Aspartate aminotransferase; BNP: Brain natriuretic peptide; PaCO₂: Partial pressure of carbon dioxide; PaO₂: Partial pressure of oxygen; A-aDO₂: Alveolar-arterial differences for oxygen; SpO₂: Pulse oxygen saturation.

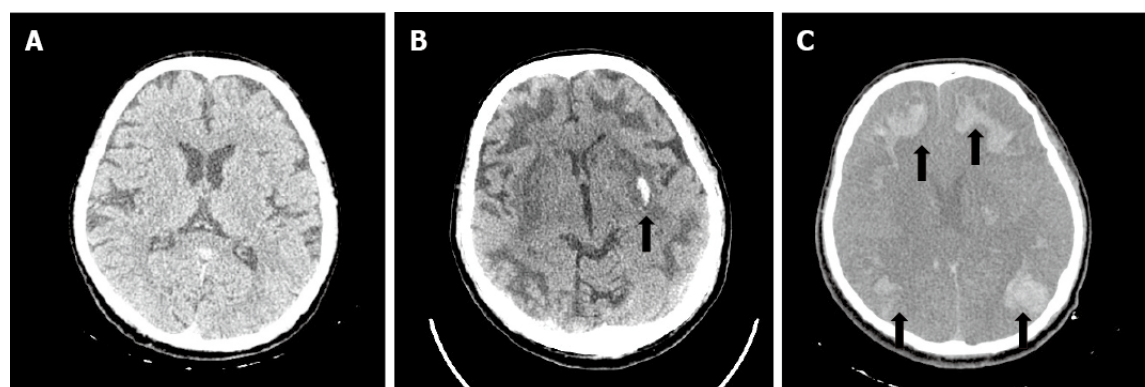
and the surgical risk was extremely high with minimal benefit. To reduce the neurotoxic effects of methanol and its metabolites, another course of CRRT was carried out, and the methanol level decreased to 2.3 $\mu\text{g/mL}$ in serum and 1.8 $\mu\text{g/mL}$ in urine.

During the subsequent treatment, the patient developed successive pancreas injury with amylase at 389 U/L (normal range, 35-135 U/L), acute liver injury with alanine transaminase (ALT) at 138 U/L (normal range, 9-50 U/L) and aspartate aminotransferase (AST) at 264 U/L (normal range, 15-40 U/L) and myocardial injury with TnI at 0.049 ng/mL (normal range, 0-0.023 ng/mL). After effective treatment, all indicators were significantly improved and the patient was able to open his eyes autonomously and respond to painful stimuli. On February 25, 2021, the endotracheal tube was removed and the patient resumed spontaneous breathing. However, on March 6, the patient fell into coma again. CT scan showed diffuse symmetric intracerebral hemorrhage (Figure 3C). The time line of

Table 3 Arterial blood gas monitoring until the end of the 2nd course of continuous renal replacement therapy

Items	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Normal range
PH	6.86	7.24	7.34	7.41	7.46	7.47	7.35-7.45
PCO ₂ (mmHg)	32	31	39	29	27	27	35-45
PO ₂ (mmHg)	101	141	121	137	109	114	80-100
SpO ₂ (%)	90	99	98	99	99	99	95-98
Base excess (mmol/L)	-27.7	-12.8	-4.8	-5.2	-3.4	-2.8	(-3) -(-3)
HCO ₃ ⁻ (mmol/L)	5.7	13.3	21	18.4	19.2	19.7	22-27
Lactic acid (mmol/L)	11.6	1.1	2.2	1.7	1.3	1.2	0.5-2.22

PaCO₂: Partial pressure of carbon dioxide, PaO₂: Partial pressure of oxygen, SpO₂: Pulse oxygen saturation.



DOI: 10.12998/wjcc.v10.i19.6571 Copyright ©The Author(s) 2022.

Figure 3 Head computed tomography. A: On February 16, 2021 when the patient was in the Emergency Department, there was slight symmetrical decrease in density in the bilateral putamen but no sign of hemorrhage; B: On February 19 after the first course of continuous renal replacement therapy, there was an area of hemorrhage 1.5 cm × 0.5 cm in the left putamen (black arrows) and bilateral confluent symmetrical hypodensity in bilateral brain parenchyma (white arrows); C: On March 6, there was diffuse symmetric intracerebral hemorrhage (black arrows).

the case is presented in [Table 1](#).

OUTCOME AND FOLLOW-UP

His family gave up further treatment, and he died in another hospital eventually.

DISCUSSION

AMP is a systemic disease that mainly affects the central nervous system and is characterized by ocular damage and metabolic acidosis. Studies of methanol mass poisoning in Estonia, Norway and Czechia have reported acute mortality of 18%-21%, whereas the rate of sequelae after survival ranged between 10% and 34% [13]. As the most serious complication, cerebral hemorrhage is relatively rare with reported prevalence of 7%-19% [14-16].

Reviewing this case, there are two points that need to be emphasized. The first is the endpoint of hemodialysis. The traditional endpoint is the completely removal of serum methanol or a concentration below 25 mg/dL (250 mg/mL) with the disappearance of acid-base imbalance. With high serum methanol concentration, dialysis of 18-21 h may be required to reach the endpoint [1]. However, methanol is not mainly responsible for the toxicity, so it may be inaccurate to evaluate the toxicity degree by the blood concentration of methanol. As a matter of fact, the methanol level reached the endpoint in the present case after the first course of CRRT. Considering that the patient was old and the levels of methanol and organic acid were extremely high, another course of CRRT was administered to eliminate methanol and its metabolites as soon as possible. This is the feature that we wish to promote for further studies. For patients who are old or in poor health with high level of serum methanol and

have no access to fomepizole, which may obviate the need for hemodialysis, the formic acid concentration should be considered as an important indicator for the endpoint of hemodialysis. Existing studies have confirmed the effectiveness of formic acid concentration measurement in the diagnosis of methanol poisoning[17,18], and it is theoretically feasible to determine clinical treatment. Unfortunately, formic acid was not detected in this case.

The second point is hemorrhage. Bilateral basal ganglia necrosis or hemorrhage are considered to be the most typical imaging features of methanol poisoning and may occur at almost any stage during the course of AMP[19]. Studies and case reports[20-26] have revealed signs of edema and necrotic damage of the basal ganglia and hemorrhages in the subcortical white matter, which may lead to parkinsonism in survivors. There are studies and conjectures about this complication[1]. It is speculated that putamen injury may be caused by both a high concentration of formic acid potentiated by poor venous drainage and inadequate arterial flow in the lenticular nucleus. This region is known to have higher consumption rates of oxygen and glucose than the adjacent white matter, meanwhile it is more sensitive to hypoxia. In addition, the anticoagulation strategy is worth discussing. AMP patients are often accompanied by hypotension, which increases the risk of thrombosis during hemodialysis. However, systemic anticoagulants may increase the risk of bleeding. The use of heparin during hemodialysis is thought to be the cause of hemorrhage[2], although hemorrhage has been seen in the absence of systemic anticoagulation [1]. In a retrospective study involving 46 patients, 2 of 15 cerebral hemorrhage patients did not receive systemic anticoagulant therapy which is similar with this case, and the study indicated no association between brain hemorrhages and systemic anticoagulation during dialysis[16]. In addition, other anticoagulant strategies such as aspirin, warfarin and novel oral anticoagulants have been used in intermittent hemodialysis of end-stage renal disease, but their safety in AMP patient is unknown[27]. Due to the limited number of cases, the predisposing factors for cerebral hemorrhage and anticoagulant strategy in AMP patients need further study.

CONCLUSION

Cerebral hemorrhage requires constant vigilance during the full course of treatment for severe cases of AMP as its predisposing factors are still unclear. And the formic acid concentration may contribute to determining clinical treatment, but further studies are needed.

ACKNOWLEDGEMENTS

The author expresses sincere thanks and condolences to the patient's family and wishes the deceased rest in peace. No patient's personal privacy information was disclosed in this case report.

FOOTNOTES

Author contributions: Li J contributed to case summary and wrote the paper; Feng ZJ contributed to the table and figure management; Liu L contribute to the literature search and induction; Ma YJ contribute to the core argument of this case; all authors revised the paper and approved the submitted version.

Informed consent statement: The patient's family provided informed written consent prior to the case report.

Conflict-of-interest statement: There is no conflict-of-interest.

CARE Checklist (2016) statement: The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016).

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S-Editor: Wu YXJ

L-Editor: A

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