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The primary aim of *World Journal of Clinical Cases (WJCC, World J Clin Cases)* is to provide scholars and readers from various fields of clinical medicine with a platform to publish high-quality clinical research articles and communicate their research findings online.

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INDEXING/ABSTRACTING

The *WJCC* is now indexed in PubMed, PubMed Central, Science Citation Index Expanded (also known as SciSearch®), and Journal Citation Reports/Science Edition. The 2019 Edition of Journal Citation Reports cites the 2018 impact factor for *WJCC* as 1.153 (5-year impact factor: N/A), ranking *WJCC* as 99 among 160 journals in Medicine, General and Internal (quartile in category Q3).

RESPONSIBLE EDITORS FOR THIS ISSUE

Responsible Electronic Editor: *Ji-Hong Liu*
 Proofing Production Department Director: *Xiang Li*

NAME OF JOURNAL

World Journal of Clinical Cases

ISSN

ISSN 2307-8960 (online)

LAUNCH DATE

April 16, 2013

FREQUENCY

Semimonthly

EDITORS-IN-CHIEF

Dennis A Bloomfield, Bao-Gan Peng, Sandro Vento

EDITORIAL BOARD MEMBERS

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

EDITORIAL OFFICE

Jin-Lei Wang, Director

PUBLICATION DATE

January 26, 2020

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INSTRUCTIONS TO AUTHORS

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

GUIDELINES FOR ETHICS DOCUMENTS

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PUBLICATION MISCONDUCT

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

ARTICLE PROCESSING CHARGE

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

STEPS FOR SUBMITTING MANUSCRIPTS

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

ONLINE SUBMISSION

<https://www.f6publishing.com>

Multiple organ dysfunction and rhabdomyolysis associated with moonwort poisoning: Report of four cases

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Author contributions: Li F was a major contributor to the writing of the manuscript; Duan TC and Liao R assisted in the preparation of the manuscript and revised it critically for valuable intellectual content; Li F, Chen AB, Duan YC, and Liao R collected and analyzed the data of the cases; Chen AB, Xu YW, and Tao LL interpreted the data; all authors critically reviewed and approved the final manuscript.

Supported by the Talent Development Program of The Second Affiliated Hospital of Kunming Medical University, China, No. RCPYXM2017-3-04.

Informed consent statement: Written informed consent was obtained from the patient for publication of this report and any accompanying images.

Conflict-of-interest statement: The authors declare that they have 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).

Open-Access: This article is an open-access article which was selected by an in-house editor and fully peer-reviewed by external

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Abstract

BACKGROUND

Moonwort is a widely used Chinese herbal medicine. It has various pharmacological effects, such as relieving cough and preventing asthma. To date, multiple organ dysfunction and rhabdomyolysis caused by moonwort poisoning have not been reported.

CASE SUMMARY

Here we report four cases of moonwort poisoning that presented with multiple organ dysfunction and rhabdomyolysis accompanied by vomiting, fatigue, and muscle aches. One patient was an adult male, two were adult females, and one was a boy, with an age range of 7–64 years. The adults were treated with hemoperfusion and symptomatic therapies, while the child was treated with plasma exchange and symptomatic therapies. All four patients recovered.

CONCLUSION

Blood purification combined with symptomatic treatment may be an effective method for managing multiple organ dysfunction and rhabdomyolysis caused by acute moonwort poisoning.

Key words: Moonwort poisoning; Multiple organ dysfunction; Rhabdomyolysis; Hemoperfusion; Plasma exchange; Case report

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Core tip: Moonwort is a widely used Chinese herbal medicine. To date, multiple organ dysfunction and rhabdomyolysis caused by moonwort poisoning have not been reported. In this case report, we describe the effects of blood purification combined with symptomatic treatment in patients with multiple organ dysfunction and rhabdomyolysis caused by moonwort poisoning. Use of this treatment was effective and safe in these patients.

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Manuscript source: Unsolicited manuscript

Received: October 30, 2019

Peer-review started: October 30, 2019

First decision: November 13, 2019

Revised: December 13, 2019

Accepted: December 22, 2019

Article in press: December 22, 2019

Published online: January 26, 2020

P-Reviewer: Coban YK

S-Editor: Zhang L

L-Editor: Wang TQ

E-Editor: Xing YX



Citation: Li F, Chen AB, Duan YC, Liao R, Xu YW, Tao LL. Multiple organ dysfunction and rhabdomyolysis associated with moonwort poisoning: Report of four cases. *World J Clin Cases* 2020; 8(2): 479-486

URL: <https://www.wjnet.com/2307-8960/full/v8/i2/479.htm>

DOI: <https://dx.doi.org/10.12998/wjcc.v8.i2.479>

INTRODUCTION

Moonwort is a plant of the genus *Pteridaceae* and the genus *Pteridium*. Moonwort is widely distributed in Yunnan, Hubei, Zhejiang, and other provinces in China. It is a widely used Chinese herbal medicine, which has numerous pharmacological effects, such as relieving cough and preventing asthma^[1,2]. In some parts of China, residents have the habit of eating moonwort. However, the chemical composition of moonwort is complex and diverse. Its effective dose and toxic dose are unknown. To date, few studies have investigated moonwort poisoning^[3-5]. The present report describes four cases of multiple organ dysfunction and rhabdomyolysis caused by moonwort poisoning.

CASE PRESENTATION

Chief complaints

Among the four patients, one was an adult male, two were adult females, and one was a boy. The age range of the patients was 7–64 years. On October 22, 2018, the four patients developed symptoms such as vomiting, fatigue, and muscle aches and pain after eating a mixture of “herbal medicine” and herbal liquid.

History of past illness

These patients are members of the same family (Table 1). Case 2 had a history of hypertension. The other cases had no particular medical history.

Physical examination

The patients visited the local hospital on the same day and were diagnosed with acute herbal poisoning. After receiving gastric lavage, laxative drugs, and other treatments, the symptoms were not relieved. The patients were subsequently transferred to our hospital on October 23. The four patients had typical vital signs. There were no abnormal signs on physical examination (Table 2).

Laboratory examinations

Admission-related investigations revealed that all four patients had liver dysfunction and elevated cardiac enzymes. However, all four patients showed marked elevations in serum levels of cardiac troponin I, myoglobin, and creatine kinase (Table 3). There were also mild increases in white blood cell count and neutrophil count (Table 3). However, electrolyte analysis did not demonstrate hyperkalemia in any of the four patients (Table 3).

Imaging examinations

Pulmonary computed tomography for the four patients showed different degrees of exudation. There were no abnormalities in renal function tests, routine urine tests, or the electrocardiogram in any of the four patients.

FINAL DIAGNOSIS

Based on the clinical history, symptom pattern, and elevated levels of creatine kinase and myoglobin, the admission diagnosis was acute herbal poisoning, multiple organ dysfunction, and rhabdomyolysis^[6,7]. During treatment at our department, the family provided the fresh and complete plants of the “herbal medicine” consumed by the patients (Figure 1). After consulting the literature and relevant Chinese herbal medicine experts, the poison was identified as moonwort. Hence, the patients were diagnosed with acute moonwort poisoning, multiple organ dysfunction (heart, liver, and lung), and rhabdomyolysis.

Table 1 Patients' characteristics

Case	Age (yr)	Weight (kg)	Past medical history	Time since poisoning to symptoms, h	Dose of herbal liquid and medicine
Case 1	64	65	No	6	200 mL + 20 g
Case 2	60	51	Hypertension	6	150 mL + 10 g
Case 3	36	53	No	2	450 mL + 40 g
Case 4	7	28	No	4	100 mL + 10 g

TREATMENT

From the day of admission, all patients were treated with oxygen therapy, liver protecting agents, cardiac nutritional treatments, anti-oxidants, anti-inflammatory drugs, and methylprednisolone. The therapeutic dose of methylprednisolone was calculated based on the patient's body weight. After consent of the patient and their family members, cases 1, 2, and 3 underwent two sessions of hemoperfusion for 2 hours on the day of admission and the second day after admission. Due to the absence of a pediatric hemoperfusion device, case 4 received two sessions of plasma exchanges on the day of admission and the second day after admission (Table 4). The methylprednisolone dose was gradually reduced on October 29. During the blood purification treatment, the patients did not develop complications such as hemorrhage or shock. After treatment, the patients' symptoms were gradually relieved, and there was no other discomfort. Blood test indicators slowly returned to normal (Figure 2, Table 3). On October 30, all patients had recovered and were discharged.

OUTCOME AND FOLLOW-UP

The four patients were followed at 30 d after discharge, and no further symptoms were reported. The treatment process and timeline are shown in Figure 3.

DISCUSSION

Here, we have reported four cases of acute moonwort poisoning with multiple organ dysfunction (heart, liver, and lung) and rhabdomyolysis. The mechanism of toxic fern poisoning remains unclear. A previous study^[8] found that moonwort contains flavonoids, plant proteins, cardiac glycosides, saponins, and phenols, but not alkaloids or oils. However, since no previous studies have identified which components of moonwort have the ability to induce rhabdomyolysis, it is not possible to draw any conclusions regarding the causative compound(s) in the four cases described here. At present, the identification of moonwort is mainly based on trait and microscopy. Zhou *et al*^[9] reported a detailed analysis and description of the morphology of the moonwort plant. After a comparative review, the four patients were diagnosed with acute moonwort poisoning. The time to symptom onset was 2–6 h, and the disease course was 8 d. The clinical condition of the patients was worse than that described previously in the literature^[3–5]. In these reports, acute moonwort poisoning caused damage to the liver, kidneys, and some other organs, but no lung injury or rhabdomyolysis occurred. The time to symptom onset was 2–8 h, and the patients recovered after symptomatic treatment within 72 h. The severity of our cases in comparison to those in previous reports may be related to factors such as a higher dose of moonwort, consumption during a different season, and different origin of the moonwort. The fertility of moonwort in different habitats is not the same^[8], and the efficacy of moonwort has noticeable seasonal differences^[10].

Rhabdomyolysis is an acquired or inherited clinical syndrome characterized by the destruction of skeletal muscle and release of intracellular constituents (such as myoglobin, enzymes, and electrolytes) that lead to a variety of systemic complications^[6]. The causes of acquired rhabdomyolysis are varied and include trauma, intense exertion, ischemia, thermal injury, drugs, and toxins^[6,11]. Regardless of the underlying cause, direct injury to the skeletal muscle membrane or energy depletion results in an increase in intracellular calcium that activates proteases and apoptotic pathways, leading to the generation of oxygen free radicals, mitochondrial dysfunction, and cell death^[12,13]. Rhabdomyolysis typically presents with myalgia, weakness, and/or myoglobinuria, but an accurate diagnosis is facilitated by the

Table 2 Vital signs on physical examination at admission to our emergency room

Case	Blood pressure (mmHg)	Pulse (beats/min)	Respiratory rate (breaths/min)	Temperature (°C)	SpO ₂ (%)
Case 1	114/69	93	19	36	96
Case 2	167/77	92	26	37	99
Case 3	138/97	110	28	36.2	95
Case 4	124/80	109	24	37.2	98

SpO₂: Peripheral capillary oxygen saturation.

detection of an elevated creatine kinase level^[6,7,11-13]. Acute kidney injury, the most frequent systemic complication of rhabdomyolysis, occurs with an incidence of 10%–40% and is associated with a poor prognosis, especially if multiple organ failure is also present^[6,13]. Other complications include electrolyte disturbances (such as hyperkalemia), hypovolemia, compartment syndrome, and disseminated intravascular coagulation^[13]. The management of rhabdomyolysis includes treatment of the underlying cause, infusion of fluids to correct hypovolemia and electrolyte disturbances, alkalization of the urine with sodium bicarbonate, and decompression of muscle compartments^[13,14].

Rhabdomyolysis may be associated with hyperkalemia due to the loss of large quantities of intracellular K⁺ from the damaged skeletal muscle and the development of acute kidney injury^[15]. It is essential that hyperkalemia is rapidly corrected because it can result in potentially life-threatening arrhythmia^[6]. However, it was notable that none of the four patients in our study exhibited an elevated level of plasma K⁺ before treatment. This may have been due to the rapid initiation of treatment and the absence of renal injury in these patients, which allowed the kidneys to compensate for the loss of K⁺ from skeletal muscle.

There is no specific antidote for acute moonwort poisoning. Gastric lavage, purgative agents, protection of essential organ functions, scavenging of oxygen free radicals, and prevention of complications are recommended. The four patients in this report developed liver, heart, and lung dysfunction as well as rhabdomyolysis after acute moonwort poisoning. Their condition was more serious than that described in previous reports. Since moonwort contains various chemicals, we used repeated hemoperfusion or plasma exchange along with symptomatic treatment. Hemoperfusion can eliminate medium- and large-sized toxic molecules and is widely used in the treatment of acute poisoning in Asia. It has a good effect on various poisons, such as paraquat and organic phosphorus^[16]. In previous studies, some patients with severe poisoning also underwent repeated hemoperfusion, and good therapeutic results were achieved^[17]. Plasma exchange is less applicable than hemoperfusion in acute poisoning. However, the daily replacement of up to 1–2 times the patient's plasma volume until symptom relief is achieved can effectively reduce the damage caused by the poison^[18]. Acetylcysteine may also have an important role in the management of poisoning. Acetylcysteine is used as an anti-oxidant to prevent and treat cell damage, reduce apoptosis, and improve mitochondrial function^[19-21]. Acetylcysteine can also improve the patient's renal metabolism and microcirculation^[22]. These effects may be beneficial in alleviating multiple organ dysfunction and improving renal function after rhabdomyolysis.

CONCLUSION

We have reported four cases of multiple organ dysfunction and rhabdomyolysis caused by moonwort poisoning. The patients were managed using blood purification techniques and symptomatic treatments. Their symptoms completely disappeared on the ninth day after poisoning. Blood purification combined with symptomatic treatment may be an effective method for acute moonwort poisoning. However, further research is required to confirm these findings.

Table 3 Results of laboratory investigations after admission

	Case	Day ¹	Day ¹ after treatment ²	Day ² after treatment ²	Day ⁷
K ⁺ , mmol/L	Case 1	3.95	4.29	3.39	4.23
	Case 2	2.93	3.02	2.82	3.59
	Case 3	3.33	3.72	3.33	4.33
	Case 4	3.57	3.77	3.22	4.09
Ca ²⁺ , mmol/L	Case 1	2.06	2.01	1.97	2.2
	Case 2	2.11	1.96	1.98	2.19
	Case 3	2.08	2.02	2.05	2.27
	Case 4	2.27	2.17	2.19	2.39
Na ⁺ , mmol/L	Case 1	140.5	138.1	139.3	138.4
	Case 2	136.1	141	144.8	146.6
	Case 3	140.3	138.3	143.9	144.8
	Case 4	141.6	140.8	138.9	140.9
Cl ⁻ , mmol/L	Case 1	106	103.2	109.6	102.3
	Case 2	103.4	108.5	113	105
	Case 3	106	103.2	111.7	104.2
	Case 4	100	104.6	100.3	99.1
Cardiac troponin I, ng/mL	Case 1	0.26	0.14	0.06	0.03
	Case 2	0.44	0.32	0.04	0.01
	Case 3	1.26	0.62	0.1	0.04
	Case 4	0.94	0.48	0.08	0.03
Myoglobin, ng/mL	Case 1	> 1000	316.3	102	68
	Case 2	> 1000	229	85	57
	Case 3	> 1000	468.7	70	55
	Case 4	225.5	225.5	40	29
Creatine kinase isoenzyme, ng/mL	Case 1	> 500	318.8	40.8	2
	Case 2	155.8	84.1	11.2	3.4
	Case 3	> 500	171.2	41.4	2.6
	Case 4	489.5	489.5	116.6	3.07
Creatine kinase, U/L	Case 1	47643	30233	30028	69
	Case 2	7039	5101	2796	86
	Case 3	42251	28228	13446	112
	Case 4	19350	5814	1458	58
White blood cell count, 10 ⁹ /L	Case 1	11.17	12.41	15.23	9.05
	Case 2	13.14	13.03	12.93	9.38
	Case 3	15.14	13.68	14	9.11
	Case 4	11.22	10.67	11.74	6.92
Neutrophil count, 10 ⁹ /L	Case 1	9.39	11.82	13.71	6.2
	Case 2	10.56	12.14	11.51	6.26
	Case 3	13.11	12.91	13.16	6.07
	Case 4	8.54	9.43	8.85	4.69
Lymphocyte count, 10 ⁹ /L	Case 1	1.37	0.58	1.13	1.27
	Case 2	1.71	0.63	1.06	2.66
	Case 3	1.59	0.7	0.69	1.68
	Case 4	1.99	1	1.84	1.61
Monocyte count, 10 ⁹ /L	Case 1	0.37	0.01	0.38	0.39
	Case 2	0.86	0.26	0.35	0.52
	Case 3	0.4	0.07	0.14	0.58
	Case 4	0.62	0.21	1.02	0.57
Eosinophilic granulocyte count, 10 ⁹ /L	Case 1	0.03	0	0	0
	Case 2	0.01	0	0	0
	Case 3	0.04	0	0.01	0.02
	Case 4	0.04	0.01	0.01	0.03

Basophilic granulocyte count, 10 ⁹ /L	Case 1	0.01	0	0.01	0.01
	Case 2	0	0	0.01	0.01
	Case 3	0	0	0	0.01
	Case 4	0.03	0.02	0.02	0.02
Red blood cell count, 10 ⁹ /L	Case 1	5.3	4.97	4.71	4.66
	Case 2	5.06	4.85	4.58	4.04
	Case 3	4.88	4.75	4.66	4
	Case 4	4.94	4.99	4.57	4.7
Blood platelet count, 10 ⁹ /L	Case 1	264	185	111	127
	Case 2	178	135	108	132
	Case 3	404	320	258	273
	Case 4	364	335	313	266
Hemoglobin, g/L	Case 1	163	154	145	141
	Case 2	148	145	136	121
	Case 3	116	113	111	117
	Case 4	135	135	125	137

¹The day in our hospital.

²Two hours after hemoperfusion/plasma exchange.

Table 4 Type and time of blood purification

Date	Case	Type	Time (h)
2018.10.23 ¹	Case 1	Hemoperfusion	2
	Case 2	Hemoperfusion	2
	Case 3	Hemoperfusion	2
	Case 4	Plasma exchange	2
2018.10.24 ²	Case 1	Hemoperfusion	2
	Case 2	Hemoperfusion	2
	Case 3	Hemoperfusion	2
	Case 4	Plasma exchange	2

¹The second day after poisoning.

²The third day after poisoning.



Figure 1 The entire moonwort plant.

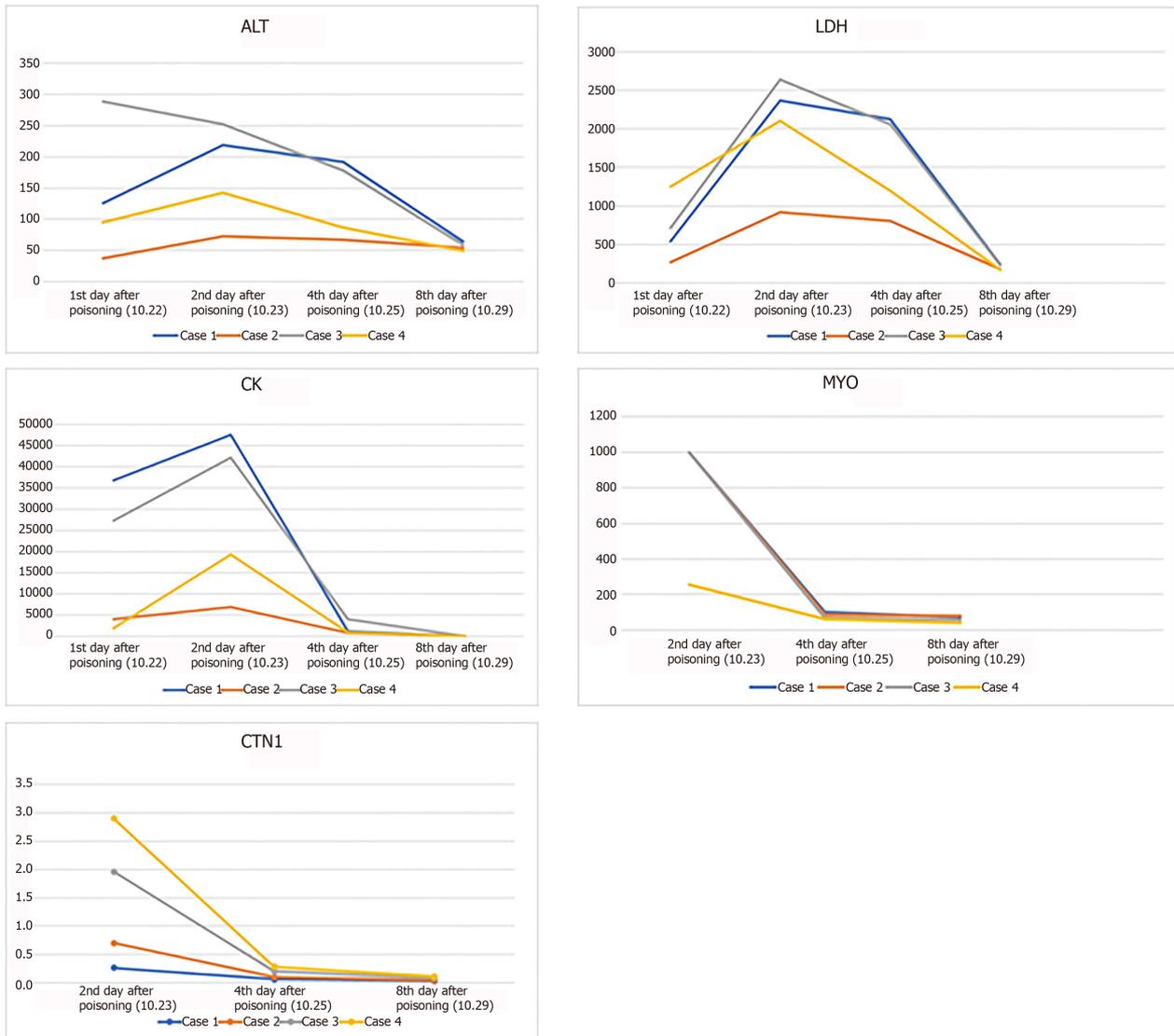


Figure 2 Laboratory results. ALT: Alanine aminotransferase (U/L); CK: Creatine kinase (U/mL); CTNI: Cardiac troponin I (ng/mL); LDH: Lactate dehydrogenase (U/mL); Myo: Myoglobin (ng/mL).

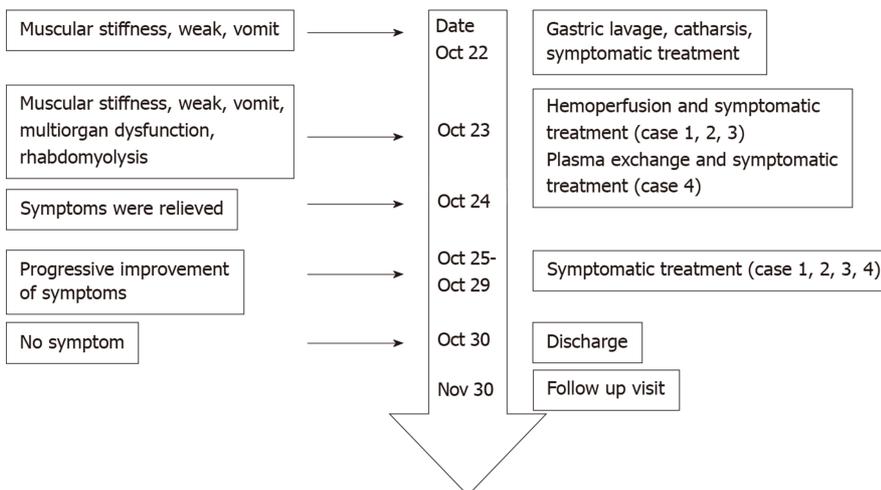


Figure 3 The treatment process and timeline for the four patients.

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