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CASE REPORT

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Delayed death of a child from chlorfenapyr poisoning: A case report and literature review

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Key Clinical Message

This was the first article reported a fatal case of chlorfenapyr poisoning in a child, and the typical symptoms before death include high fever, severe sweating, coma, and limb stiffness, and elevation of myocardial enzymes and myoglobin; neurological symptoms tend to appear earlier in children than in adults.

KEYWORDS

children, Chlorfenapyr, delayed death, poisoning

1 | INTRODUCTION

As a widely used insecticide, chlorfenapyr has a broad insecticidal spectrum and strong, quick-acting, and long-lasting effects.[1] Since chlorfenapyr was commercialized, there have been continuous reports of fatal cases of chlorfenapyr poisoning. Due to the high fatality rate of chlorfenapyr poisoning and certain delayed toxic reactions, clinicians do not have a clear grasp of the treatment plan for poisoned patients, thereby often leading to sudden deterioration of the patient's condition, and eventually death in most cases. Until recently, there had been no reported cases of death related to chlorfenapyr poisoining in children. This article reports a fatal case of chlorfenapyr poisoning in a child, and through literature review, analyzes its lethal clinical characteristics, and discusses the treatment strategies. Written informed consent was obtained from patient's father.

2 | CASE HISTORY

A child patient, female, 13 years and 5 months old, was admitted to the hospital on May 29, 2022, due to "accidental ingestion of chlorfenapyr for 9 days and being unconscious for 4 days". This girl took the insecticide chlorfenapyr after it was incorrectly placed and mistakenly thought to be milk. The child took a small (unknown) amount of chlorfenapyr on May 21st but showed no symptoms of vomiting, convulsions, fever, and headache. The local hospital gave her gastric lavage treatment 1 h after admission. After treatment, the patient complained of abdominal pain and dizziness. After 3 days of hospitalization (involving rehydration, stomach protection, etc.), the patient improved and was discharged. On May 25th, the patient developed a headache and sore throat. On the 26th, she developed sweating, drowsiness, and showed poor spirit. She had a low-grade fever of 37.8°C and vomited stomach contents

once. Following that, the child began speaking gibberish, and in the evening, she appeared unconscious and unresponsive but did not have convulsions. She was hospitalized again, the body temperature reached 39.7°C and she remained unconscious, without convulsions or vomiting. Considering her critical condition, the patient was transferred to the Pediatric Intensive Care Unit (PICU) of our hospital at 13:25 on May 29th. The patient was previously in good health with no history of drug or food allergy.

The patient underwent a physical examination upon admission. Her body temperature, pulse, respiration and blood pressure were 38.9°C, 102 cpm, 33 cpm, and 123/52 mmHg respectively. She appeared normally developed, with moderate nutrition, but unconscious with a Glasgow Coma Scale (GCS) of 6 points and moist skin. Her eye sockets were not sunken, and her eyes and face showed no puffiness. Her bilateral pupils were equal and round, with the left side being 2.5 mm and the right side being 2.5 mm, both showing slow response to light. Upon gastrointestinal decompression, brown fluid was drained from the patient's stomach. Her neck was soft, but no dry or moist rales were heard in either lungs. There was no precordial bulge, the heart rate was uniform with 102 beats/min, the heart sounds were low and dull, the abdomen was flat, the abdominal wall was soft, and the bowel sounds were normal. The muscle tone of the limbs was normal, and the bilateral Barthel's, Klinefelter's and Brinell's signs were all positive. Auxiliary examination at the local hospital indicated WBC: 7.29*109/L, NEUT%: 67.90%, ALT: 108 U/L, AST: 65 U/L, LDH: 217.6 U/L, CK: 1529 U/L, CK-MB: 46 U/L, Mb:2283 ug/L. Examination at our hospital indicated blood ammonia: 100umol/L; WBC: 12.80*109 /L, NEUT%: 95.20%, ALT: 54 U/L, AST: 360 U/L, LDH: 552 U/L, CK: 12376 U/L, CK-MB: 97 U/L, Mb:407.50 ug/L, blood gas analysis: PH: 7.417, LAC: 1.3 mmol/L. A CT scan of the brain showed that the bilateral cerebral hemispheres were plump, the ventricles and cisterns were smaller, suggesting possible brain swelling, and reduced densities of the bilateral frontotemporal lobe and brainstem. The remaining results, including cerebrospinal fluid examination, were normal.

3 | METHODS

The patient was treated following the process of: low-flow oxygen after admission, cefotaxime sodium (75 mg/kg, q6h) for anti-infection, acyclovir (10 mg/kg, q8h) for antivirus (central nervous system infection could not be excluded), cooling, omeprazole (0.6 mg/kg, qd) for acid suppression, mannitol (1 g/kg, q6h) for lowering intracranial pressure, various vitamins for nourishing nerves, rehydration and symptomatic and supportive treatments. At 18:20, the patient's percutaneous oxygen saturation dropped to 90% under low-flow oxygen. She experienced shortness of breath, irregular breathing and extremely poor mental response. Immediately after endotracheal intubation and ventilator-assisted ventilation were provided, the transcutaneous oxygen saturation rose to 98%. At 19:25, the patient experienced convulsions under assisted ventilation, manifested as whole body shaking, followed by body stiffness, limb stiffness, stoppage of spontaneous breathing, decreasing of heart rate to 0 cpm, and complexion and lips cyanosis. A review of blood gas analysis found PH: 6.808, type II respiratory failure, mixed acidosis, serum potassium 10.2 mmol/L, LAC: 18 mmol/L. Blood gas analysis suggests acidosis and respiratory arrest caused by epileptic seizures. cardiopulmonary resuscitation (CPR) was provided immediately to the patient. However, after repeated use of epinephrine bolus and intratracheal instillation, her heart rate remained 0 cpm. At 20:15, the patient's pupils became fixed at about 5 mm, and showed no response to light. The Electrocardiograph (ECG) showed a straight line, and clinical death was declared.

4 | CONCLUSION AND RESULTS

In conclusion, chlorfenapyr is highly toxic, has a high fatality rate, atypical early symptoms, and the characteristics of delayed poisoning reaction. The neurological manifestations in children tend to happen faster than in adults. Brain CT/MRI, CK, and Mb examinations were used as auxiliary means for examination. In addition to routine gastric lavage, catharsis, fluid replacement, etc., blood purification treatment should be actively performed regardless of the dose of poisoning and the severity of early symptoms. Early PE and HP therapy may also be considered. In addition, it is suggested that the observation period for hospitalized patients should be extended to 2 weeks or even longer.

5 | DISCUSSION

Pubmed, Springer, and Medline databases were searched using “chlorfenapyr” as the keyword for relevant case reports, and 16 articles were found, including a total of 21 cases of chlorfenapyr poisoning.[2–17] The clinical data of all patients were collected, including gender, age, clinical manifestations, laboratory tests, prognosis, etc. The results are as follows (Table 1).

In general, a total of 21 cases, including 10 in China, two in Japan, seven in South Korea, one in the United States, and one in India were found; the earliest reported

case was a case in Japan in 2004,[18] but the full text was not available, and hence was excluded; another case from South Korea was reported as pancreatitis caused by chlorfenapyr poisoning,[19] but the patient had a long history of alcoholism and alcoholism was present at the time of poisoning. We believed that the pancreatitis could not be completely attributed to chlorfenapyr poisoning and was therefore also excluded. The 21 cases found were all adults with chlorfenapyr poisoning, the youngest was 21 years old and the oldest was 74 years old. There were 13 males and eight females; 16 cases of poisoning occurred orally, of which 13 cases were suicidal oral administration, and three cases were accidental administration. The minimum amount ingested was 5 mL, and the maximum was 300 mL. Among the cases, one patient who did not die spit out all the liquid immediately after taking it by mistake, but still suffered from the sequelae of lower limb paralysis. Three of the 21 cases were poisoned through inhalation, all of which were inhalation of chlorfenapyr powder or steam by workers in factories. There were also 1 case of suicide intraperitoneal injection and one case of extensive skin contact.

Clinical symptoms: we found that during the incubation period from ingestion of chlorfenapyr to the onset of initial symptoms, 13 cases showed symptoms within 1 day, and the remaining cases showed symptoms within 10 days. The symptoms reported included fatigue, sweating, nausea, vomiting, dizziness, shortness of breath, blurred vision, and even difficulty urinating, and abdominal pain in those who take a large dose. Among the two survivors, one developed visual loss within 14 days, and the other developed progressive paralysis of the lower extremities and calf within 19 days. Most adult patients entered a coma (GCS ≤8 points) within 24 h before death. The child patient in this report however showed neurological symptoms 4 days before death, much earlier than that in the adults. The symptoms at the time of death were coma, high fever, and severe sweating. Five of the cases described the same peculiar manifestations of muscle stiffness found in the child's case.

Auxiliary examination: 21 cases showed abnormal elevation of CK and Mb, and some cases showed an elevation of AST, ALT, NEUT%, CK-MB, LAC, and LDH, but the increase rate was not as remarkable as those of CK and Mb; 10 cases described imaging findings from MRI or CT, all of which suggested implication of white matter, demyelinating lesions, and diffuse edema.

Treatment and prognosis: 21 cases were treated with conventional gastric lavage, catharsis, fluid infusion, liver protection, stomach protection. Among them, 7 cases received hemodialysis treatment, but all seven cases died. Of the 21 cases, 19 died. The shortest case of death within 24 h was the patient who took 300 mL of chlorfenapyr, and the longest case of 21 days was the patient who took only 5 mL. The mean time to death was 8.6 days. The other two survivors developed irreversible sequelae of optic atrophy and lower limb paralysis, respectively.

Chlorfenapyr is a new type of pyrrole compound that acts on the mitochondria of insect cells, mainly inhibiting the conversion of adenosine diphosphate (ADP) to adenosine triphosphate (ATP), thereby hindering the production of intracellular energy, leading to cell exhaustion and achieving insecticidal effect.[20] Judging from the general situation of the 21 human poisoning cases, most of them came from China, South Korea, Japan, and other Asian countries. The management of agricultural pesticides in these countries should therefore be strengthened. In addition, 13 patients were suicidal. Psychological counseling for patients with depression or mental illness should therefore be strengthened to avoid the occurrence of similar situations. The child patient reported in this study ingested chlorfenapyr by accident. Awareness of the danger and storage guidance of household insecticides should therefore be strengthened to prevent children from taking them by mistake.

Delayed toxic reaction is the major characteristic of chlorfenapyr poisoning. In the case of the child patient, she was only given gastric lavage and other routine treatments at the early stage of poisoning and was discharged when the symptoms were relieved. On the 6th day of poisoning, the patient's condition recurred, with fever and neurological symptoms. The child developed cardiac arrest on the 9th day of poisoning, and was unsuccessfully rescued. Most of the 21 adult cases reviewed were discharged from the hospital after the initial symptoms improved, and were readmitted after a few days. The specific mechanism of delayed toxic reaction of chlorfenapyr poisoning is still unclear. Some studies believe that chlorfenapyr is a fat-soluble compound that is easily absorbed by organs. After the toxins in the blood are removed, the poisons absorbed by the organs can be slowly released into the blood again, causing delayed toxic reactions.[21]

Chlorfenapyr is defined as a moderately dangerous pesticide by the World Health Organization.[22] Combined with this case report and literature review, the fatality rate of chlorfenapyr poisoning is extremely high. The typical symptoms of death include high fever, coma, severe sweating, and general stiffness. Mitochondrial dysfunction and insufficient energy supply after poisoning lead first to the damage of tissues and organs with high energy demand,[23] such as the brain and muscles. Insufficient energy supply to the brain will lead to hypoxia changes, such manifested as dizziness, lack of muscle energy, and general weakness. With increased metabolic consumption, shortness of breath can occur, and the energy released by

TABLE 1 Clinical data of 21 patents with chlorfenapyr poisoning in the retrieved case reports.

Author/Year/Country	Age(years)	Gender	Exposure dose	Way of poisoning	Incubation period	Initial symptoms
Zhang et al.[2] /2022/ China	49	Male	30 mL	Oral (suicide)	4d	Fatigue, sweating
Zha o et al.[3] /2022/China	31	Female	Unknown	Oral (mistake)	10d	Nausea, vomiting, blurred vision, dizziness, sweating
Zha o et al.[3] /2022/China	36	Male	300 mL	Oral (suicide)	0 h	Sweating, chest tightness, nausea, vomiting
Liao et al.[4] /2022/China	21	Male	Unknown	Oral (suicide)	1 h	Nausea, vomiting, dizziness, fatigue, sweating
Liao et al.[4] /2022/China	55	Male	60 mL	Oral (suicide)	0.5 h	Nausea, sweating, fatigue, abdominal pain
Gong et al.[5] /2021/China	50	Female	60 mL	Oral (suicide)	1 h	Sweating, nausea, vomiting, dizziness
Gong et al.[5] /2021/China	50	Male	UNKNOWn	Inhalation powder (factory)	0.5d	Sweating, fatigue, low fever
Gong et al.[5] /2021/China	38	Male	Unknown	Inhalation powder (factory)	1d	Dizziness, fatigue, sweating
Gong et al.[5] /2021/China	32	Female	5 mL	Oral (mistake)	13d	Nausea, vomiting, sweating
Luo et al.[6] /2020/China	66	Male	20 mL	Oral (suicide)	0 h	Fatigue, nausea, sweating, dizziness, shortness of breath
Kobashi et al.[7] 2020/ Japan	45	Female	100 mL	Oral (suicide)	0.5 h	Vomiting, abdominal pain
Han et al.[8] 2019/Korea	49	Male	Unknown	Skin contact	1d	Difficulty urinating, blurred vision, sweating, thirst
Chomin et al.[9] /2018/US	42	Male	300 mL	Oral (suicide)	0 h	Nausea, vomiting, abdominal pain

Gcs ≤8 points	Symptoms before death	Laboratory abnormal index (maximum)	Brainct/mri	Blood purification treatment	Ending
7d	Coma, high fever (41°C), severe sweating, muscle stiffness	CK: 5385 U/L; AST: 161 U/L; NT-proBNP: 334 pg/mL		HP 2 times on the 4th day	Death on day 7
11d	Coma, high fever (42.2°c), severe sweating	CK: 4130.2 U/L; Mb: 785.5 ng/mL; NEUT%: 72.4%	CT: brain and pons lesions		Death on day 11
20 h	Coma, fever (38.1°c), severe sweating	CK: 4640 U/L; CK-MB: 458.4 ng/mL; Mb: 23220.7 ng/mL ALT: 112 U/L; AST: 647 U/L; NEUT%: 75.5%		HP 2 times on the first day	Dead within 24 h
3d	Coma, high fever, heavy sweating, muscle stiffness	CK: 933.74 U/L	CT showed no abnormality	Give HP once on the second day	Death on day 3
7d	Coma, fever, heavy sweating, muscle stiffness	CK: 820.48 U/L; Mb: 358.28 ng/mL		Give HP once on the first day	Death on day 7
7d	Coma, high fever (40°C), severe sweating	CK: 1960 U/L; CK-MB: 49 ng/mL; Mb: 1810 ng/mL ALT: 47.7 U/L; AST: 95 U/L; NEUT%: 79.1%			Death on day 7
8d	Coma, high fever (41°c), severe sweating	CK: 1590 U/L; Mb: 547 ng/mL; ALT: 84 U/L; NEUT%: 84%; LDH: 333 U/L			Death on day 8
3d	coma, high fever (40°C), severe sweating	CK: 5945 U/L Mb: 1136 ng/mL; ALT: 189 U/L; NEUT%: 82.3%; LDH: 350 U/L			Death on day 4
21d	Coma, high fever (40.1°C), severe sweating	CK: 3762 U/L CK-MB: 57 ng/mL; Mb: 346 ng/mL; LDH: 266 U/L	MRI: diffuse swelling of brain tissue		Death on day 21
4d	Coma, high fever (41°C), severe sweating, muscle stiffness	CK: 4931 U/L CK-MB: 68.4 ng/mL; Mb: 1791 ng/mL ALT: 181.3 U/L; AST: 92.3 U/L		HD + HP once on the 3th day,CRRT once on the 4th day	Death on day 5
8d	Coma, high fever (41°C), severe sweating	CK 604 U/L, AST 112 U/L	MRI: white matter abnormalities, cervical and thoracic abnormalities	HP + CHDF once on the 8th day	Death on day 16
5d	Coma, high fever (41.5°C), severe sweating	CK: 4484 U/L CK-MB: 29.64 ng/mL; Mb: 857.5 ng/mL ALT: 56 U/L; AST: 170 U/L	MRI: Abnormal signal in the internal capsule, brain stem, cerebellum		Death on day 5
5d	Coma, high fever (42.2°C), severe sweating, muscle stiffness	CK: 432 U/L; LAC: 7.9 mmol/L		HD once on the 6th day	Death on day 5

(Continues)

TABLE 1 (Continued)

Author/Year/Country	Age(years)	Gender	Exposure dose	Way of poisoning	Incubation period	Initial symptoms
Su et al.[10] /2018/Korea	44	Female	10 mL ~ 20 mL	Oral (suicide)	14d	Vision loss
Baek et al.[11] /2016/Korea	44	Female	Trace	Vomiting immediately after oral administration (accidental)	19d	Progressive paralysis of the lower leg, with reduced to complete loss of sensation
Kang Kang et al.[12] /2014/Korea	41	Female	20 mL	Oral (suicide)	14d	Nausea, fatigue
Lee et al.[13] /2013/Korea	74	Male	20 mL	Intraperitoneal injection (suicide)	0 h	Abdominal pain, sweating, fever
Tharaknath et al.[14] /2013/India	28	Female	Unknown	Oral (suicide)	5d	Lower extremity weakness, lethargy, fever
Kwon et al.[15] /2012/ Korea	49	Male	200 mL	Oral (suicide)	1 h	Fatigue, sweating
Choi et al.[16] /2010/Korea	55	Male	250 mL	Oral (suicide)	2d	Sweating, fever, thirst, blurred vision
Hoshiko et al.[17] /2007/ Japan	55	Male	Unknown	Inhalation of steam (factory)	0 h	Sweating, fatigue

Abbreviations: AST, aspartate aminotransferase; ALT, glutamine aminotransferase; CHDF, continuous hemofiltration dialysis; CK, creatine kinase; CK-MB, creatine kinase isoenzyme; CT, Computed Tomography; CRRT, continuous Hemofiltration; GCS, Glasgow Coma Scale; HP, hemoperfusion; HD, hemodialysis; LAC, blood lactic acid; LDH, lactate dehydrogenase Enzyme; Mb, myoglobin; MRI, Magnetic Resonance Imaging Scan; NEUT%, percentage of neutrophils; NT-proBNP, N-terminal pro-brain natriuretic peptide.

the oxidation of substances in the body cannot be stored and can only be released in the form of heat, resulting in an increase in basal metabolism and body temperature, resulting in sweating to dissipate heat. The nervous system of children is still developing, and therefore the energy supply required by the brain is relatively large. This explains the earlier neurological manifestations in children than that in adults. If the toxins are not removed and the energy not replenished in time, the energy supply to the heart will be insufficient. This will cause CK and Mb to increase significantly, leading to myocardial hypoxia, and cardiac arrest. The insufficient skeletal muscle function will cause general stiffness, and severe energy shortage to the brain, resulting in a deep coma and brain swelling, and eventual death.

The clinical manifestations and death time of chlorfenapyr poisoning appear to be dependent on a dose–response relationship. The patient who took 300 mL orally died within 24 h, and the patient who took 5 mL by mistake will die after 21 days of poisoning; The other two survivors had trace doses, but both had serious sequelae. Chlorfenapyr is a fat-soluble compound that can theoretically be cleared by hemoperfusion, but the effect of hemoperfusion analyzed in this study is poor. Considering the high mortality rate of chlorfenapyr poisoning, this study believes that it is better to take intensive treatment immediately after exposure to chlorfenapyr, including combined treatment such as blood purification, plasma exchange and hemoperfusion. Even if the symptoms are mild after ingestion or the intake of chlorfenapyr is very small. On the other hand, some patients with chlorfenaprile poisoning, including the present case, were temporarily discharged because their symptoms were not obvious, indicating that doctors had insufficient knowledge of the natural course of chlorfenaprile poisoning. Through this case report, it is hoped to improve doctors' awareness of the late toxic reaction of chlorfenapyr poisoning and avoid the occurrence of delayed diagnosis and treatment. In addition, in view of this case of misuse, it is called for the society to strengthen the management of pesticide labeling to avoid

Gcs ≤8 points	Symptoms before death	Laboratory abnormal index (maximum)	Brainct/mri	Blood purification treatment	Ending
None	None		MRI: optic nerve and optic tract white matter abnormalities		Optic atrophy
None	None		MRI: suspected toxic leukoencephalopathy, diffuse swelling of the spinal cord		PParalysis of lower limbs
15d	Coma, high fever (40.7°C), severe sweating	CK: 3081 U/L; AST: 130 U/L	CT: Diffuse brain swelling		Death on day 15
12 h	Coma, high fever, heavy sweating				Death on day 12
8d	Coma, high fever, heavy sweating	CK: 10370 U/L	MRI: white matter demyelinating lesions, diffuse edema		Death on day 10
14d	Coma, high fever (40°C), severe sweating	CK 14336 U/L; CK-MB 81.1 ng/mL; ALT 116 U/L; AST 332 U/L	MRI: Symmetrical changes in bilateral white matter		Death on day 14
5d	Coma, high fever (40.9°C), severe sweating	CK: 10507u/L; Mb: 3603 U/L			Death on day 5
7d	Coma, high fever (40°C), severe sweating				Death on day 7

the recurrence of similar cases of misuse. Of course, this study also has shortcomings. The number of cases considered is small, the treatment effect is poor, and there are no cases with good treatment effects as a control, and therefore this study can only provide clinical recommendations and cannot be used as a guidance.

AUTHOR CONTRIBUTIONS

Yishan Zhan: Writing – original draft; writing – review and editing. **Jing Li:** Data curation; investigation. **Yourong Zhu:** Data curation; investigation. **Qiang Tao:** Data curation. **Shouhua Zhang:** Conceptualization; writing – original draft; writing – review and editing.

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There was no funding for this study.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

The studies involving human participants were reviewed and approved by Jiangxi Provincial Children's Hospital. Written informed consent for participation was not provided by the participants' legal guardians/next ofkin because the requirement for written informed consents was waived since no additional interventions and potential harmwere posed to these patients.

CONSENT

Written informed consent was obtained from the patient's legal guardian to publish this report, in accordance with the journal's patient consent policy.

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