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CASE REPORT

Brain damage caused by chlorfenapyr poisoning: a case report and literature review

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Abstract

Introduction: Chlorfenapyr poisoning is uncommon, but fatal, and is often ignored. Chlorfenapyr inhibits ATP production in the mitochondrial of lipid-rich organs such as the brain. The initial symptoms of chlorfenapyr poisoning are not serious and are usually ignored; fever and unconsciousness are the main signs. Patients often die of brain damage, and survivors often present toxic leukoencephalopathy.

Case report: We report a case of a 15-year-old female who swallowed 10 mL of 10% chlorfenapyr, and was subjected to gastric lavage one hour after ingestion. The patient felt no discomfort on the first and second day after lavage and went to school. On the third day, the patient complained of a headache and rested at home. On the fourth day, the patient still complained of headache, and the condition progressed to confusion and fever; therefore, the patient was admitted to the emergency room and underwent hemoperfusion. Cerebral CT revealed diffuse brain edema. The patient died on the fourth day because of central fever, brain hernia, and brain dysfunction.

Conclusion: Chlorfenapyr poisoning is fatal, even in small doses. Patients suspected of chlorfenapyr poisoning should be closely observed and promptly treated by hemoperfusion.

Keywords

Poisoning; Chlorfenapyr; Leukoencephalopathy; Hemoperfusion

1. Introduction

Chlorfenapyr is widely used as a pesticide to eliminate cotton worms, insects, and mites [1]. It is highly lipophilic, and its mechanism of action involves the uncoupling of oxidative phosphorylation and respiration in mitochondria, thus preventing ATP production [2]. This leads to anoxic necrosis of cells [2] in lipid-rich organs and other organs that require a high supply of ATP such as the brain, heart, and muscle. We report a case of a 15-year-old female who swallowed chlorfenapyr and died of brain damage and hernia.

2. Case report

A 15-year-old female student presented to our emergency room with a headache and confusion due to the ingestion of 10 mL of 10% chlorfenapyr four days previously, after a quarrel with her mother. The patient bought chlorfenapyr on the internet to kill mites on a dog. She had no history of other diseases.

One hour after chlorfenapyr ingestion, the patient was subjected to gastric lavage, and was placed under observation for approximately 12 hours before being discharged. The next day, the patient did not report any discomfort and went to school. On the third day, she complained of headache and weakness and rested at home.

On the fourth day, the headache did not improve and the patient demonstrated altered levels of consciousness. Therefore, the patient was admitted to the emergency department of our hospital.

The patient was diagnosed with chlorfenapyr poisoning based on the information provided in the anamnesis, and brain damage and symptoms of headache and confusion. The toxicological serum analysis to evaluate toxic agents were not conducted for practical reasons. The vital signs when the patient arrived at the emergency room were as follows: temperature, 36.0 °C; pulse rate, 95 beats/min; respiratory rate, 23 breaths/min; blood pressure, 116/83 mmHg; and oxygen saturation, 97% in room air. The only physical sign was confusion. The results of the initial arterial blood gas analysis, blood cell analysis, indices of liver and kidney function, coagulation cascade, procalcitonin, myocardial enzymes, troponin T, NT-pro brain natriuretic peptide, and electrolytes are presented in Table 1. The electrocardiogram and the chest CT were normal. Cerebral CT revealed diffuse edema in both hemispheres (Fig. 1).

Hemoperfusion (HP) was conducted using two resin perfusion columns as soon as the patient arrived at the emergency room. Mannitol and vitamin B were administered and the patient was adequately rehydrated via the intravenous route. Eleven hours later, the patient progressed to light coma, with

TABLE 1. The investigations conducted after admission.

Items	On admission	11 hours later	14 hours later	Normal range
Arterial blood gas value				
PH	7.467	7.455	6.883	7.35–7.45
PO2 (mmHg)	139	52	44	80–105
PCO2 (mmHg)	32.7	30.6	98	35–45
HCO3-(mmol/L)	23.1	21	18	22–27
Lactate (mmol/L)	0.95	2.11	>the high limit	0.5–1.7
Blood cell analysis value				
WBC $\times 10^9$/L	6.23		8.93	5–12
NEUT%	77		90.7	40–75
LYMPH%	15.8		8.4	20–50
Blood biochemical value				
AST (U/L)	14			13–45
ALT (U/L)	17			7–40
BUN (mmol/L)	4.17			2.6–7.5
CREA (μmol/L)	42			41–73
CK (U/L)	66			40–200
PCT (ng/mL)	0.03			0–0.5
NT-proBNP (pg/mL)	87.26			0–125
hs-TNT (ng/mL)	<0.003			0–0.014
Na (mmol/L)	140.2			137–147
K (mmol/L)	3.86			3.5–5.3
CL (mmol/L)	108.4			96–108
Coagulation				
PT (s)	14.9			11–14
APTT (s)	35.2			28–43.5
TT (s)	14.8			14–21

Abbreviations: WBC, white blood cell; AST, asparate transaminase; ALT, alanine transaminase; BUN, blood urea nitrogen; CREA, creatinine; CK, creatine Kinase; PCT, procalcitonin; hs-TNT, hypersensitivity troponin T; NT-proBNP, NT-pro brain natriuretic peptide; PT, prothrombin time; APTT, activated partial thromboplastin time; TT, thrombin time.

a high fever of 39.9 °C; pulse rate, 117 beats/min; respiratory rate, 33 breaths/min; blood pressure, 107/54 mmHg; and oxygen saturation, 84% with an oxygen mask. The arterial blood gas analysis revealed that hypoxia, hyperventilation, metabolic acidosis, and lactic acid value increased. Fourteen hours later, the patient's breathing became laborious and she progressed to moderate coma; therefore, endotracheal intubation and mechanical ventilation were conducted. The arterial blood gas analysis revealed severe acidosis and hypoxia, CO_2 retention, and type II respiratory failure, and that the lactate concentration exceeded the highest acceptable limit (Table 1). Fifteen hours later, the heart rate dropped rapidly, eventually leading to cardiac arrest; cardiopulmonary resuscitation (CPR) was immediately but unsuccessfully employed. After 50 minutes of CPR, the patient died. The autopsy was not performed because her father refused.

3. Discussion

In the case reported here, the patient felt no discomfort on the first and second day. Thereafter, the patient complained of headache and weakness on the third day and progressed to altered levels of consciousness on the fourth day. Although HP was conducted, the patient experienced high fever, coma, and laborious respiration, and she died on the fourth day. As we did not find any source of infection or signs of organ failure, and the brain edema was notable on CT, we speculated that she died of central fever and brain hernia and dysfunction.

We searched articles using "chlorfenapyr" and "case" as keywords in titles and abstracts in PubMed, EMBASE, Google Scholar, and Scopus databases from 01/01/1970 to 03/01/2021 and found 12 case reports with full text (Table 2) (Ref. [3–14]).

TABLE 2. The characteristics of the 12 cases of chlorfenapyr poisoning.

Author_Year	Country	Age (year)	Gender	Way of poisoning	Dosage (mL)	Gastric lavage	Fever (Da)	Disorde of consciousness (Db)	Blood purification (Dc)	Outcome (Dd)	Sequelae
Ku JE _2015 [3]	Korea	61	female	oral	10	yes	yes (d2)	yes (1 h)	no	live	pancreatitis
Baek BH_2016 [4]	Korea	44	female	oral (spat it out without swallowing)	10	no	no	no	no	live	leukoencephalopathy
Chomin J _2018 [5]	America	42	male	oral	300	no	yes (d6)	no	HD (d6)	death (d6)	-
Choi UT _2010 [6]	Korea	55	male	oral	250	yes	yes (d2)	yes (d5)	HD (d4)	death (d5)	-
Luo ZH_2020 [7]	China	66	male	oral	20	yes	yes (d2)	yes (d4)	CRRT + HP (d3)	death (d6)	-
Tharaknath VR _2013 [8]	India	28	female	oral	not mentioned	yes	yes (d10)	yes (d10)	no	death (d10)	-
Kang C _2014 [9]	Korea	41	female	oral	20	no	yes (d14)	yes (d14)	no	death (d15)	-
Lee J _2013 [10]	Korea	74	male	intraabdominal injection	20	no	no	no	no	death (d12)	-
Han S-K _2018 [11]	Korea	49	male	dermal exposure	not mentioned	no	yes (d5)	yes (d5)	no	death (d5)	-
Kim IS _2018 [12]	Korea	52	male	oral	100	not mentioned	yes (d12)	yes (d12)	HP (d5, d8)	live	leukoencephalopathy
Kim JH _2020 [13]	Korea	71	male	oral	100	not mentioned	no	no	no	death (d20)	-
Park SJ _2018 [14]	Korea	44	female	oral	20	yes	no	no	no	live	toxic optic neuropathy

Abbreviations: Da, first day with fever after poisoning; Db, first day of experiencing confusion after poisoning; Dc, day of blood purification after poisoning; Dd, day of death after poisoning; HD, hemodialysis; CRRT, continuous renal replacement treatment; HP, hemoperfusion.

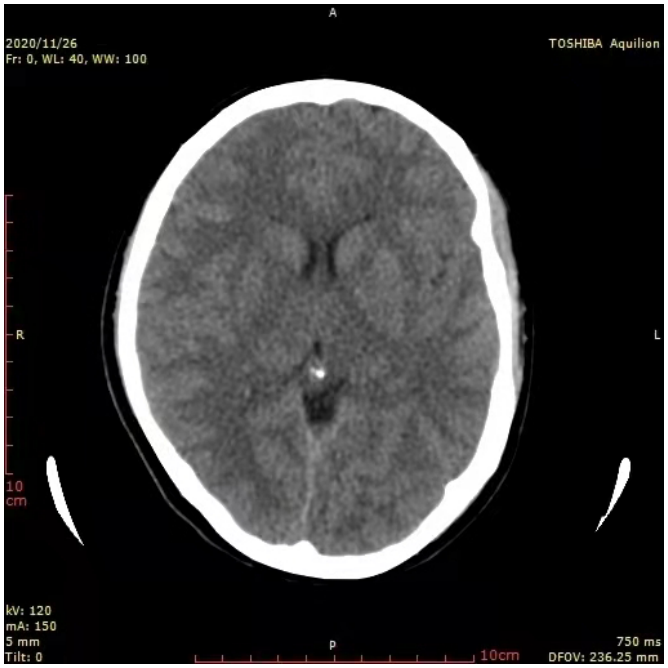

FIGURE 1. Cerebral CT showed diffuse edema in both hemispheres.

The results of the literature search are summarized below.

A 61-year-old-female [3] swallowed 10 mL of chlorfenapyr and survived with chronic pancreatitis. A 44-year-old-female [4] progressed to irreversible paralysis because of ingestion of 10 mL of chlorfenapyr, but spat it out without swallowing. The MRI image displayed toxic leukoencephalopathy in white matter tracts throughout the brain, brain stem, and spinal cord. The woman survived probably because she did not swallow the pesticide. A 52-year-old-male [12] swallowed 100 mL of chlorfenapyr, but survived probably because HP was conducted on the fifth and eighth days. The sequela was leukoencephalopathy. A 44-year-old-female [14] swallowed 20 mL of chlorfenapyr and survived with toxic optic neuropathy.

The patients who swallowed 300 mL [5] or 250 mL [6] of chlorfenapyr died despite hemodialysis (HD) probably owing to the excessive intake. A 71-year-old-male [13] swallowed 100 mL of chlorfenapyr and died because of the excessive intake. A 66-year-old-male [7] ingested 20 mL of chlorfenapyr and died despite continuous renal replacement treatment (CRRT) and HP. A 28-year-old-female [8] died, but the exact intake was unknown. A 41-year-old-female [9] ingested 20 mL of chlorfenapyr and died probably due to brain damage. A 74-year-old-male [10] died after an intra-abdominal injection of 20 mL of chlorfenapyr. A 49-year-old-male [11] died after dermal exposure to chlorfenapyr.

From Table 2, we observe that patients with low intake of chlorfenapyr, no fever, and absence of altered consciousness may survive, although they may have sequelae, such as leukoencephalopathy. HP probably clears the poison from the blood, thereby saving the patients' lives. The lethal dose of chlorfenapyr is low and even skin contact may be lethal [11]. Most of the patients died because of the large intake of the poison. Fever and altered consciousness always indicate brain damage; doctors should be alert because those patients are more likely to die within few days. The death latency of chlorfenapyr poisoning is 7–20 days [3].

In this case, the patient might have had toxic leukoencephalopathy when admitted to the emergency room on the fourth day after poison intake, and we did not have the opportunity to perform a brain MRI. Chlorfenapyr may damage brain areas rich in nerve fibers, such as white matter, leading to toxic leukoencephalopathy [15].

Unlike the patients described in the above reports, the patient described in this report was young. The initial poisoning symptoms are not serious and are usually ignored; fever and unconsciousness are signs that one should be careful about; the patient needs to be observed closely. The limitation of the study is the lack of determination of drug concentration and effective treatment method for Chlorfenapyr poisoning. Chlorfenapyr is lipophilic; therefore, HP may be performed with resin to eliminate it from the body [16], but CRRT has little effect [17]. If the patient survives, hyperbaric oxygen therapy may be implemented to avoid leukoencephalopathy. We should raise awareness among doctors and the young population regarding chlorfenapyr poisoning and inform the population through the internet about its potential harm; children need to be educated not to drink unknown liquids. If chlorfenapyr poisoning is suspected, immediate observation and treatment should be implemented.

AUTHOR CONTRIBUTIONS

FY read and sorted out the relevant literature, and collected the medical records. DF designed the research and wrote the whole article, and revised it. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was obtained from the Ethics Committee of the First Affiliated Hospital of Medical College of Xi'an Jiaotong University (XJTU1AF2021LSK-057).

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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