JOURNAL OF CLINICAL TOXICOLOGY
ISSN: 2161-0495
OPEN ACCESS Freely available online
Case Report

# Clinical and CT Manifestations of Delayed Toxic Encephalopathy caused by Low-Dose Chlorfenapyr Poisoning: A Case Report

**Changjian Qiu[1*], Huan Sun[1], Hongqi Xiao[1], Yuan Cao[2,3]**

*[1]Mental Health Center, West China Hospital of Sichuan University, Chengdu, China;[2]Department of Nuclear Medicine, West China Hospital of Sichuan University, Chengdu, China;[3]Huaxi MR Research Center (HMRRC), Department of Radiology, West China Hospital of Sichuan University, Chengdu, China*

## ABSTRACT

**Case:** A 13-year-old girl attemped to suicide by ingesting only 10 ml of 10% chlorfenapyr. She presented with toxic encephalopathy after a latent period of more than a week, and rapidly progressed to death within days of symptoms. Chlorfenapyr intoxication could result in severe diaphoresis, high fever, general fatigue, rhabdomyolysis, and neurologic symptoms. Brain Computed Tomography (CT) showed a diffused impairment of white matter, brain stem, and cerebellum as well as wide edema in grey matter regions.

**Discussion:** The chlorfenapyr mainly impairs the function of energy-consuming vital organs and lead to corresponding clinical symptoms, and we should be treated it with caution at the first visit. Chlorfenapyr intoxication exhibited a latent period from 7 to 14 days, even asymptomatic patients should be actively observed. Commercial chlorfenapyr is lipophilic, and early multiple hemoperfusion is necessary.

**Keywords:** Toxic; Chlorfenapyr Poisoning; Low-dose; Intoxication

## INTRODUCTION

A girl attempted suicide by ingesting chlorfenapyr and attended to hospital after a week with severe diaphoresis, high fever, general fatigue, rhabdomyolysis, and neurologic symptoms which are the symptoms of chlorfenapyr intoxication rapidly progressed to death within days. Brain Computed Tomography (CT) had shown several changes in the brain with was cause by ingesting of 10 ml of 10% chlorfenapyr.

## CASE REPORT

A 13-year-old girl with 4 years of depression complaining of headache and vomiting for 10 days was admitted to the psychiatric ward. She experienced a negative life event 17 days before admission and committed suicide by ingested nearly 10 ml of 10% chlorfenapyr. After taking the pesticide, she vomited actively and was sent to the local hospital for gastric lavage immediately. No symptoms such as dizziness, headache, vomiting, fever, palpitations, and dyspnea occurred during the 7 days observation. Sudden onset of intermittent headache with 3 times a day and lasting about 10-20 min each time occurred on day 8. The above symptoms lasted for nearly a week without any diagnosis and treatment. On day 14, she began to present a persistent headache and frequent vomiting accompanied by fatigue and large sweat. She was treated with reducing cranial pressure and alkalizing urine in the emergency department for 3 days. However, no relief of symptoms was observed, then she was transferred to our hospital for further treatment.

Her vital signs on admission were as follows: body temperature, 36.7°C; pulse rate, 98/min; respiratory rate, 20/min; and blood pressure, 113/71 mmHg. She was conscious but lack cooperation in physical examination. No pathologic signs were elicited. The brain Computed Tomography (CT) of another hospital showed no abnormalities. The initial electrocardiograph (ECG) indicated sinus bradycardia without QT intervals prolongation. The renal function and serum electrolyte tests were normal, but other laboratory indicators showed a high level of serum aspartate transaminase (AST 130 IU/L), serum alanine transaminase (ALT 72 IU/L), and serum creatinine kinase (2727 IU/L). Serial laboratory results were shown in Table 1.

**Table 1:** Patient laboratory examination

| Parameters | Poisoning day 17 | Poisoning day 18 | Poisoning day 19 | |
|---|---|---|---|---|
| **Biochemistry** | | | | |
| ALT (IU/L) | 73 | - | 101 | 1009 |

**Correspondence to:** Qiu C, Mental Health Center, West China Hospital of Sichuan University, Chengdu, China, 610041, Tel:+028-85422633; Email: qiuchangjian18@163.com

**Received:** November 23, 2020; **Accepted:** December 07, 2020; **Published:** December 14, 2020

**Citation:** Qiu C, Sun H, Xiao H, Cao Y. (2020) Clinical and CT Manifestations of Delayed Toxic Encephalopathy caused by Low-Dose Chlorfenapyr Poisoning: A Case Report. J Clin Toxicol. 11:464.

| | | | | |
|---|---|---|---|---|
| AST (IU/L) | 129 | - | 128 | 814 |
| BUN (mmol/L) | 8.1 | - | 5.7 | 5.1 |
| Cr (umol/L) | 58 | - | 53 | 76 |
| CK (IU/L) | 2727 | - | 1844 | 1165 |
| LDH (IU/L) | 252 | - | 282 | 1163 |
| K+ (mmol/L) | 4.17 | - | 4.04 | 5.03 |
| Na+ (mmol/L) | 139.3 | - | 139.6 | 143.5 |
| Cl- (mmol/L) | 103.6 | - | 104.8 | 97.5 |
| GLU (mmol/L) | 5.82 | - | 6.96 | 22.14 |
| **BNP and cardiac markers** | | | | |
| BNP (ng/L) | 93 | - | - | 1480 |
| CK (ng/ml) | 72.18 | - | 98.20 | 180.10 |
| CK-MB (ng/ml) | 11.30 | - | 4.50 | 3.61 |
| cTnT (ng/L) | 6.0 | - | 5.1 | 232.4 |
| **ABGA** | | | | |
| pH | - | 7.399 | 7.416 | 6.875 |
| pO2 (mmHg) | - | 89.1 | 147 | 178.6 |
| pCO2(mmHg) | - | 31.6 | 31.3 | 75.8 |
| HCO-3 (mmol/L) | - | 19.1 | 19.7 | 13.7 |

Abbreviations: ALT: Alanine Transaminase; AST: Aspartate Transaminase; BUN: Blood Urea Nitrogen; Cr: Creatinine; CK: Creatine Kinase; LDH: Lactate Dehydrogenase; BNP: Brain Natriuretic Peptide; CK-MB: Creatine kinase; cTnT: Cardiac Troponin T; ABGA: Arterial Blood Gas Analysis.

After admission, the patient was provided with symptomatic treatment such as aggressive fluid therapy, reduction of intracranial pressure, alkalinization of the urine, and liver protection therapy. However, the symptoms were not alleviated but her condition worsened progressively. Twenty-four hours after admission, she had significant disturbance of consciousness with a Glasgow Coma Score (GCS) of 9. The brain CT examination indicated a diffuse white matter density reducing and grey matter swelling in Figures 1A and B. The brain stem was deformed; the basal cistern was squeezed, narrowed, and blurred, indicating a high risk of hernia (Figure 1C). Besides, the symmetric density reduction and edema were also found in the infratentorial cerebellum (Figure 1D). Her vital signs began to be unstable: heart rate fluctuated between 46-60/min; systolic blood pressure fluctuated between 100-120 mmHg; diastolic blood pressure fluctuated between 40-65 mmHg. Her cervical resistance and left Babinski sign were positive, but the bilateral tendon reflex was not elicited. Thirty-two hours after admission, her body temperature fluctuated between 37.5°C -38.2°C with persistent liver and heart damage. 48 h after admission, she suffered a sudden cardiac arrest. We performed advanced cardiac life support immediately, but she eventually died at 19 days after taking chlorfenapyr.

## DISCUSSION

We reported a fatal case of chlorfenapyr poisoning in a 13-year-old Chinese juvenile, which is the youngest poison case until now. The chlorfenapyr, a moderately hazardous pesticide, has been widely used for insect eradication over the past 15 years [1]. Although an insect pesticide, chlorfenapyr was considered to present a fatal effect in humans. The mechanism of chlorfenapyr is to uncouple oxidative phosphorylation in the mitochondria resulting in disrupted ATP production, cell death, and ultimately, death of the organism [2]. It has been reported that the chlorfenapyr mainly impairs the function of energy-consuming vital organs (such as the brain and skeletal muscle) and lead to corresponding clinical symptoms.

Previous studies have reported the clinical and radiologic findings of chlorfenapyr induced central nervous system damage [3-7]. Chlorfenapyr intoxication could result in severe diaphoresis, high fever, general fatigue, rhabdomyolysis, and neurologic symptoms which would get worse rapidly until death. In our case, the patient experienced a severe headache, diaphoresis, and fever. Without knowing the specific toxicants, these clinical symptoms could provide a basis for early identification of chlorfenapyr intoxication, which should be treated with caution. Except for a young age, the difference between our case and previous reports is the ingested volume. Previous case reports suggested intake of approximately 20 to 250 ml, but our patient ingested only 10 ml and vomited immediately. Two cases with ingesting volume over 200 ml appeared clinical symptoms immediately and death ultimately [7,8] whereas, the present case experienced poisoning for only 7 days without any clinical symptoms and developed delayed toxic encephalopathy. The results suggested that the clinical course of chlorfenapyr intoxication was mainly related to the ingested volume. Despite low doses of chlorfenapyr were ingested, it still caused patients' death. Notably, before the rapid deteriorating fatal neurologic manifestation, a 7 to 14 days latent period has been reported by previous cases [5,8]. Our patients also experienced a latent period with no symptoms for 7 days after chlorfenapyr ingestion. These cases alert physicians to pay further attention to the latent period, especially, for those patients without any symptoms and stable vital signs even after early or symptomatic treatment should also be closely monitored for 7-14 days in the intensive care unit.

As for imaging findings, through the CT images, we also found a diffused impairment of white matter, brain stem, and cerebellum as well as wide edema in grey matter regions. Regrettably, the Magnetic Resonance Imaging (MRI) examination was not performed because

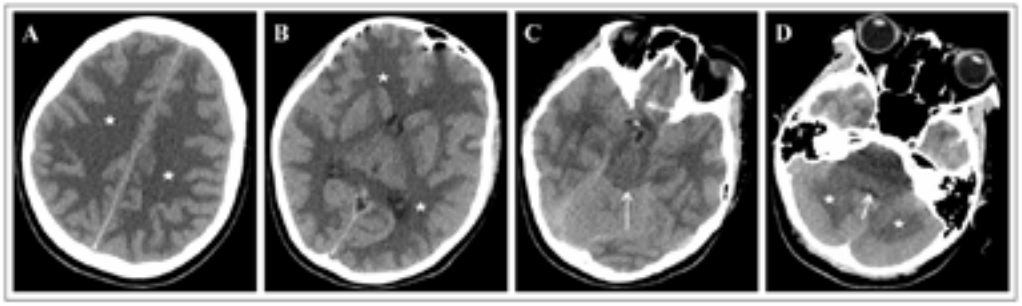


**Figure 1:** A 13-year-old girl with major depressive disorder by taking chlorfenapyr for a suicide attempt. The CT images showed a diffuse impairment involved the white matter with a white matter density reducing (white pentagram) as well as an extensive grey matter swelling (Figure 1 A and B,). The volume of the brain stem was decreased and the density of which was reduced. The brain stem was squeezed by the surrounding brain tissue, resulting in brain stem deformation with anteroposterior diameter increased (white arrow); basal cistern was squeezed and narrowed, and blurred (Figure 1C). The reduced symmetric density and edema on the infratentorial cerebellum (white pentagram). The fourth ventricle was squeezed and distortion (white arrow, Figure 1D). A: the slice of centrum semiovale; B: the slice of basal ganglia; C: The slice of skull base; D: the slice of the cerebellum.

of the rapid progression of this disease. Nevertheless, our CT-based evidence has also demonstrated extensive brain damage does exist in this disease.

For the treatment opinion, we have the following thoughts from our painful lesson. The commercial chlorfenapyr pesticide usually contains the active ingredient (6%–10%) and detergent (90%–94%), and the function of detergent surfactants makes chlorfenapyr lipophilic [2]. In theory, the chlorfenapyr could be removed by hemoperfusion [9]. To our knowledge, the positive symptomatic treatment was performed in previous cases, but most of the patients died due to extremely severe delayed poisoning. In addition to aggressive symptomatic treatment, multiple hemoperfusion as early as possible may save the patient's life.

## CONCLUSIONS

Based on this case and previous reports, there are three suggestions for the diagnosis and treatment of chlorfenapyr poisoning. First, severe diaphoresis, rhabdomyolysis, and neurologic symptoms are specific clinical symptoms of chlorfenapyr poisoning; head MRI examination and dynamic monitoring are necessary. Second, besides routine poisoning treatment, patients with chlorfenapyr poisoning need to be treated with repeated hemoperfusion in time. Last but most important, patients with stable conditions after treatment should be hospitalized for close observation until the latent period is exceeded.

## STATEMENT OF ETHICS

The research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. Relatives of patient have given their written informed consent to publish the case.

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