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Cardiac Consequences of Sodium Cyanide Exposure: An Animal Model Study

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A B S T R A C T

Sodium cyanide is a toxic substance widely used in various industries, posing a significant risk to human health and the environment. This study investigated the effects of sodium cyanide exposure on cardiac markers in rabbits over a 90-day period. Thirty-six New Zealand white rabbits were divided into control and experimental groups, with the latter receiving 10ml of 0.05mg sodium cyanide orally daily for 30, 60, and 90 days. Blood samples were collected and analyzed for lactate dehydrogenase (LDH), creatine kinase (CK-MB), and troponin I levels. The results showed significant increases in CK-MB and troponin I levels at all time points, indicating cardiac damage or stress. LDH levels showed a cumulative effect, with significant increases observed at 90 days. The findings suggest that sodium cyanide exposure can lead to cardiac damage and stress, with implications for human health, particularly in industries where exposure is possible. The study highlights the need for strict safety protocols, regular health monitoring, and environmental protection to mitigate the risks associated with sodium cyanide exposure.

Keywords: Sodium cyanide, cardiac markers, lactate dehydrogenase, creatine kinase, troponin I, cardiac toxicity.

INTRODUCTION

Cyanide is a rapidly acting, potentially lethal toxin that exists in various forms, including hydrogen cyanide (HCN), sodium cyanide (NaCN), and potassium cyanide (KCN) (Agency for Toxic Substances and Disease Registry, 2012). Exposure to cyanide can occur through natural sources, such as certain foods, industrial chemicals, and cigarette smoke (National Institute for Occupational Safety and Health, 2010).

The toxic effects of cyanide are attributed to its ability to inhibit cellular respiration, specifically by binding to cytochrome c oxidase (CcO) in the mitochondrial electron transport chain (Hall & Rumack, 1986). This inhibition leads to a shift from aerobic to anaerobic metabolism, resulting in cellular hypoxia and tissue damage. Organs with high metabolic demands, such as the brain and heart, are particularly susceptible to cyanide toxicity (Hall & Rumack, 1986).

The inhibition of CcO, leads to a decrease in tissue oxygen utilization and a subsequent increase in anaerobic metabolism (Way, 1984). This results in the production of lactate and a decrease in ATP production, ultimately leading to cellular death (Patel & Yim, 2017).

Clinical manifestations of acute cyanide poisoning include tachycardia, hypertension, and cardiac arrhythmias, which can progress to cardiac arrest and death (Kerns et al., 2017). Chronic exposure to cyanide can lead to tissue damage and the release of biomarkers into the bloodstream, which can be used to diagnose and monitor tissue damage (Bhattacharya et al., 2017).

In particular, the heart is susceptible to cyanide toxicity, and biomarkers such as lactate Dehydrogenase, troponin and creatine kinase can be used to diagnose cardiac damage (Tümer et al., 2017). Chronic cyanide exposure can also lead to histopathological changes in various tissues and organs, including the heart, liver, and kidneys (Kao et al., 2017).

MATERIALS AND METHODS

This study utilized 36 New Zealand white rabbits (Oryctolagus cuniculus) with an average weight of 1.1 kg. The rabbits were divided into two main groups: control and experimental groups.

Control Group

The control group consisted of 18 rabbits divided into three subgroups: Control Group 1 (CG1), Control Group 2 (CG2), and Control Group 3 (CG3), each containing 6 rabbits. These subgroups were reared for 30, 60, and 90 days, respectively. The control animals received water ad-libitum and daily feed for their respective rearing periods.

Experimental Group

The experimental group consisted of 18 rabbits and was also divided into three subgroups: Experimental Group 1(EG1), Experimental Group 2 (EG2), and Experimental Group 3(EG3) with 6 rabbits in each group. The animals in these subgroups were administered 10ml of 0.05mg sodium cyanide orally daily for 30, 60, and 90 days, respectively. Blood samples were collected from the rabbits after the treatment period for analysis.

Selection Criteria

The study utilized rabbits that met the following criteria:

- Age: 6-8 months
- Weight: 1.2-1.5 kg

Only rabbits within these age and weight ranges were selected to participate in the study.

Ethical Approval

Ethical approval was received from the Faculty of Medical Laboratory Science Research and Ethical Committee, Federal University Otuoke, Nigeria, and all procedures in accordance with the guidelines and regulations approved for the use and care of animals were strictly followed.

Sample Collection

At the end of 30, 60 and 90 days respectively, 5ml of blood were collected by cephalic vein (CV) puncture from the rabbits in each group, under light ether anesthesia. The collected blood samples were centrifuged at 10,000 rpm for 10 minutes to separate the serum.

Laboratory Analysis

The separated sera were used to measure the levels of LDH, Troponin I and CK-MB. AccuBind Enzyme Linked Immunosorbent Assay (ELISA) kits were used to measure the levels of Troponin I and CK-MB (Bhayana and Henderson, 1995) (Apple, 1992) while Elabscience ELISA kit was used to measure the levels of LDH (Stevens et al., 1983).

Statistical Analysis

Data were analyzed by T-test using Graph Pad Prism (version 9.5.1) software. The results were expressed as means and standard deviations. Statistical significance was considered at a 95% confidence interval ($P<0.05$).

RESULTS

Table 1 shows the measured levels of cardiac markers 30 days after administration of 10ml 0.05mg sodium cyanide. The data compares the levels of Lactate Dehydrogenase, Creatine Kinase, and Troponin I between the control and the test group.

Key Findings:

1. LDH: No significant difference was seen between the control and test groups ($P = 0.3511$).
2. CK-MB: A significant increase was observed in the test group as compared to the control group ($P < 0.0001$).
3. Troponin I: A significant increase was observed in the test group compared to the control group ($P <0.0001$).

Table 1: Cardiac Markers 30 Days after Administration of 10ml 0.05mg Sodium Cyanide

Study Group	Lactate Dehydrogenase (ng/ml)	Creatine Kinase (CK-MB) (ng/ml)	Troponin I (ng/ml)
CG1	18.24 ± 0.22	237.10 ± 1.67	0.41 ± 0.08
EG1	18.37 ± 0.24	259.20 ± 2.55	0.82 ± 0.13
P- Value	0.3511	<0.0001	<0.0001

Table 2 represents the three measured cardiac biomarkers 60 days after administration of 10ml of 0.05mg sodium cyanide. The data compares the levels of the three cardiac markers between the control group and the treated group.

Key Findings:

1. LDH: There was no significant difference between the control and the treated group (P = 0.0649).
2. CK-MB: A highly significant increase was observed in the treated group compared to the control group (P < 0.0001).
3. Troponin 1: A highly significant increase was also observed in the treated group as compared to the control group (P < 0.0001).

Table 2: Cardiac Markers 60 Days after Administration of 10ml 0.05mg Sodium Cyanide

Study Group	Lactate Dehydrogenase ng/ml)	Creatine Kinase (CK-MB) (ng/ml)	Troponin I (ng/ml)
CG2	18.53 ± 0.26	236.20 ± 1.45	0.41 ± 0.05
EG2	18.78 ± 0.14	280.90 ± 2.33	1.33 ± 0.26
P- Value	0.0649	<0.0001	<0.0001

The data in table 3 compares the levels of the three cardiac biomarkers between the control group and the treated group 90 days after administration of 10ml of 0.05mg Sodium Cyanide.

Key Findings:

1. LDH: There was a significant increase in the treated group as compared to the control group (P = 0.0042).
2. CK-MB: There was a highly significant increase in the treated group as compared to the control group (P < 0.0001).
3. Troponin 1: There was also a highly significant increase in the treated group as compared to the control group (P < 0.0001).

Table 3: Cardiac Markers 90 Days after Administration of 10ml 0.05mg Sodium Cyanide

Study Group	Lactate Dehydrogenase (IU/L)	Creatinnee Kinase (CK-(CK-MB) (ng/ml)	Troponin I (ng/ml)
CG3	18.45 ± 0.24	236.70 ± 1.57	0.40 ± 0.05
EG3	18.93 ± 0.21	296.30 ± 2.18	1.87 ± 0.12
P-value	0.0042	<0.0001	<0.0001

Discussion

The presented results show the effects of 10ml 0.05mg sodium cyanide administration on cardiac markers in rabbits over a 90-day period. The results show significant increases in lactate dehydrogenase, creatine kinase and troponin I levels, indicating cardiac damage or stress.

LDH levels showed no significant change at 30 days (P = 0.3511), a non-significant increase at 60 days (P = 0.0649), and a significant increase at 90 days (P = 0.0042). This suggests a potential cumulative effect of sodium cyanide on LDH levels.

CK-MB levels show a significant increase at all time points (30 days: P < 0.0001, 60 days: P < 0.0001, 90 days: P < 0.0001). This indicates significant cardiac damage or stress caused by sodium cyanide administration.

Troponin I levels show a significant increase at all time points (30 days: P < 0.0001, 60 days: P < 0.0001, 90 days: P < 0.0001). This confirms cardiac damage or stress as indicated by CK-MB levels.

The significant increases in CK-MB, troponin I and LDH levels indicate cardiac damage or stress, which can lead to serious health complications like cardiac arrhythmias (abnormal heart rhythms) due to cardiac damage (Kumar et al., 2017). Prolonged cardiac stress can lead to cardiac failure (Mann, 2015). Severe cardiac damage can also increase the risk of sudden cardiac death (Priori et al., 2015). Exposure to sodium cyanide can also cause neurological effects, including headache, dizziness, and confusion (Baskin et al., 2016). It can also lead to negative respiratory effects, including shortness of breath and chest pain (ATSDR, 2019).

The findings of this study are consistent with previous researches on the toxic effects of sodium cyanide on the cardiovascular system. Previous studies have shown that cyanide exposure can cause cardiac damage and stress, leading to increased levels of cardiac biomarkers such as creatine kinase and Troponin I (Baskin et al., 2016; Borowitz et al., 2017).

The mechanism of cyanide toxicity involves the inhibition of cellular respiration, leading to a decrease in ATP production and an increase in oxidative stress (Ballantyne, 1987). This causes cardiac damage and stress, leading to the release of cardiac biomarkers as observed.

Animal studies have also shown that cyanide exposure can cause cardiac damage and stress in various species, including rats (Kanthasamy et al., 2017) and mice (Liu et al., 2018).

The findings of this study have significant implications for human health, particularly in situations where exposure to sodium cyanide is possible. Workers in industries that use sodium cyanide, such as mining, electroplating, and chemical manufacturing, are at risk of exposure (ATSDR, 2019). Prolonged exposure can lead to cardiac damage and stress, as seen in this study.

Environmental exposure to cyanide can occur through contaminated water, soil, or air (EPA, 2020). Communities living near industrial sites or waste disposal areas may be at great risk of exposure effects.

Sodium cyanide can also contaminate food, particularly fruits and vegetables, through the use of pesticides and fertilizers (FAO, 2018). Consumption of contaminated food can lead to exposure and associated toxic effects.

Based on this study's findings, the following recommendations should be considered. Industries that use sodium cyanide should implement strict safety protocols to minimize worker exposure, provide personal protective equipment (PPE), and conduct regular health monitoring to detect signs of cardiac damage or stress.

To protect the environment, industries must ensure proper waste disposal, and regulatory agencies should enforce guidelines for sodium cyanide use and disposal. Regular monitoring of water and soil quality is also crucial to prevent and detect contamination.

In terms of public health, educating the public about the risks associated with sodium cyanide exposure is essential, as is monitoring food quality for potential contamination. Communities should also develop emergency response plans in case of sodium cyanide exposure incidents.

Future research should focus on further understanding the mechanisms of sodium cyanide toxicity and its effects on human health. This could involve exploring the development of antidotes or treatments for sodium cyanide exposure and investigating alternative chemicals that can replace it in various applications. By taking these steps, we can mitigate the risks associated with sodium cyanide exposure and protect both human health and the environment.

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