

Toxicological Investigation of Acute Cyanide Poisoning of a 29-year-old Man: A Case Report

Martin Anazodo Nnoli^{1*}, Nwidu Lucky Legbosi², Paul Alozie Nwafor³, Ijezie Innocent Chukwuonye⁴

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ABSTRACT

Background: Cyanide poisoning is frequently lethal, Because of the early onset of severe symptoms and difficulty in the diagnosis.

Case: The case was a 29-year-old boy who collapsed suddenly after taking a bottle of beer in a bar. Samples, such as peripheral blood, stomach contents, bile fluid, urine and mouth swabs, were prepared using standard autopsy procedure and were subjected to analysis for cyanide using visible spectrophotometric method. The cyanide contents in samples, included stomach content (260 ppm), bile fluid (272 ppm), blood (256 ppm), and mouth swab (265 ppm).

Conclusion: The cause of death was acute myocardial infarction following acute poisoning from ingestion of cyanide salts. Its lethality was related to the rapid onset of toxicity, non-specific nature of the symptoms, and failure to consider the diagnosis. Regarding the absence of pathognomonic symptoms for its toxicity, delay in acquiring a full history and unexplained sudden collapse or acidosis which worsen the prognosis, cyanide intoxication needs to be expeditiously diagnosed and managed.

Keywords: Cyanides, Myocardial Infarction, Poisoning, Shock, Sudden Death.

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INTRODUCTION

Acute cyanide poisoning in human is rare and is predominantly caused by smoke inhalation from fires and much more rarely by intentional ingestion of cyanide salts as in suicide or homicide attempts (1,2). Other sources of exposure could occur by smoke inhalation from residential/industrial fires, fumigation, water-soluble potassium and sodium cyanide salts, insecticides, and accidental contact in laboratory workers. Apart from potassium and sodium cyanide salts, numerous cyanide compounds exist, including gaseous hydrogen cyanide (HCN), and

water-insoluble mercury, copper, gold, and silver cyanide salts (3).

Because of the early onset of severe symptoms and difficulty in diagnosis, cyanide poisoning is frequently lethal. It is a rare experience for most clinicians to encounter but continues to be used in suicides and homicides (4,5). Its lethality is related to the rapid onset of toxicity, non-specific nature of the symptoms, and failure to consider the diagnosis. Acute cyanide toxicity can take place through ingestion, mucous membrane absorption, and inhalation. Since there are no pathognomonic symptoms for its toxicity, it is pertinent to acquire a full

1- Department of Anatomic and Forensic Medicine, University of Calabar, Calabar, Nigeria.

2- Department of Pharmacology and Toxicology, Niger Delta University, Bayelsa, Nigeria.

3- Department of Pharmacology and Toxicology, University of Uyo, UyoAkwa-ibom State, Nigeria.

4- Department of Internal Medicine, Federal Medical Center, Umuahia, Nigeria.

*Correspondence Author: E-mail: Mnnoli@yahoo.com

history and consider the diagnosis in cases of unexplained sudden collapse or acidosis.

This study presents a unique case of homicidal poisoning of a postgraduate chemist by cyanide which is the first report from the southeast part of Nigeria. The report is expected to alert the public and the emergency physicians about proper evaluation of other possible causes of sudden collapse and effective holistic approaches in management of patients in this class. The aim of this case is to inform physicians that cyanide poisoning does occur in the environment, although rare, it needs to be considered as differential diagnosis in cases of sudden death.

CASE REPORT

History was narrated by the father of a 29-year-old single Igbo man, a fresh university graduate. He was apparently well until about three months ago when his university classmate visited him at home and requested they visit a bar to celebrate their graduation. He was offered a bottle of beer by one of his former classmates. He initially opted to drink directly from the bottle; however, the friend offered him a glass to drink from. While drinking in the bar, his mood suddenly changed. He was said to have started vomiting and complained of dizziness. He also had dyspnea. Fearing that his situation was dire, he was rushed to a nearby private hospital where he eventually died despite efforts made by doctors on duty to save his life. He had no history of chronic or acute illnesses prior to the incident. Relatives suspected foul play and immediately reported the death to the police who

requested urgent autopsy to learn the cause of death. Both gross and histopathological examinations were carried out on the body and major organs using standard autopsy procedure. Toxicological analysis of cyanide contents in histopathological specimens (peripheral blood, stomach contents, bile fluids, and mouth swabs) was done using UV spectrophotometric method.

RESULTS

The results of autopsy and toxicological analysis are presented as follows:

Autopsy Findings

There was generalized cyanosis of the body with patchy dark to brick red areas all over the face and the neck region. There were streaks of bloody vomitus oozing out of the left corner of the mouth. The esophagus was hyperemic with recent ingested food and was stained with blood. There was moderate to severe laryngo-trachea hyperemia with frothy discharge. The lungs were pink, wet, heavy and shining weighed 720 and 685 grams (left and right, respectively). The heart appeared grossly pale brick red and its cut sections showed streaks of frank blood within the myocardium, and 70% of the surface was pale which suggested recent myocardial infarction (Figures 1 and 2). The splenic capsule was wrinkled (Figure. 3). The stomach had multiple patchy discrete points of petechia and ulceration on the mucosal linings (Figure. 4). Other organs and small intestines showed no pathological changes.



Figure 1. The cyanosed heart.

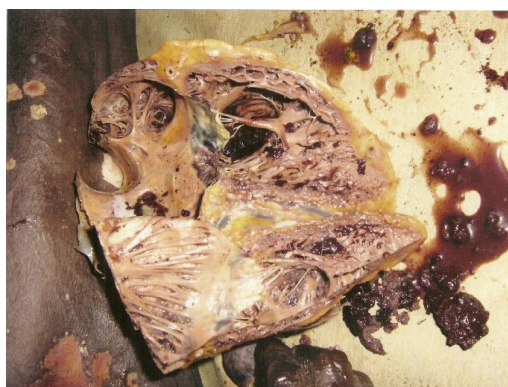


Figure 2. The hemorrhagic area with an area of infarction.



Figure 3. The shrunken splenic capsule.



Figure 4. Patchy ulceration of the mucosal lining of the stomach rimmed by hemorrhage.

Table 1. Toxicological analysis of histological specimens for cyanide content.

S/No.	HISTOLOGICAL SPECIMEN	QUANTITY OF CYANIDE (ppm)
1.	Peripheral blood	256
2.	Stomach contents	260
3.	Bile fluid	272
4.	Mouth swabs	265

ppm = part per million

Toxicology

The samples submitted to spectrophotometric analysis revealed cyanide contents in all histological specimen ranging from 266-272 part per million as shown in Table 1.

Autopsy and toxicological findings corroborated each other and the report reveals that cause of death was due to acute myocardial infarction following acute poisoning from ingestion of cyanide salts.

DISCUSSION

Cyanide poisoning is difficult to diagnose, except in cases of early confession by the person that administered the poison. Regrettably, recognition of cyanide poisoning may be delayed because the majority of clinical and laboratory findings are non-specific (6-8).

Cyanide exerts its toxic effects by binding to the ferric ion in the a-a³ complex of cytochrome oxidase resulting in the inhibition of aerobic metabolism (9-12). This may account for cellular damage seen in most of the tissues as metabolism shifts from aerobic to anaerobic, with consequent production of lactic acid.

The symptoms experienced by our patient prior to death were similar to those reported in Robert *et al.*'s study (2) which revealed a dose-related incidence of unconsciousness, dyspnea, and cyanosis with a non-cardiogenic pulmonary oedema. These were also seen in our case and the heavy, wet, and shining lungs features corroborated severe pulmonary oedema.

The sudden collapse and cellular hypoxia results from inhibition of cytochrome oxidase and accounts for sudden deaths (13). This is because cyanide has a distinct inhibitory mechanism that is exerted at two different levels of respiratory chain. Carbon monoxide binding to hemoglobin is potentiated by hydrogen cyanide that binds at cellular level (14). This inhibits the mitochondrial cytochrome oxidase; hence,

causing deprivation of oxygen consumption at cellular level. These, in turn, result in a shift towards an anaerobic process with energy depletion, intracellular acidosis, and cell death.

Death from cyanide poisoning is rare; however, it needs to be considered in differential diagnosis in evaluation of cases of sudden death. This is because it is an easy way to poison someone, as it is difficult to diagnose without the aid of autopsy. This case further highlights the need to carry out autopsy on all patients experiencing sudden death as this may reveal more cases of cyanide poisoning in our society, making it difficult for the culprits to go unpunished.

CONCLUSION

The cause of death was determined to be acute myocardial infarction following ingestion of cyanide salts. Its lethality was related to the rapid onset of toxicity, non-specific nature of the symptoms, and failure to consider the diagnosis. Regarding the absence of pathognomonic symptoms for its toxicity, delay in acquiring a full history and unexplained sudden collapse or acidosis which worsens the prognosis of cyanide intoxication needs to be expeditiously diagnosed and managed. This report was aimed at alerting the public and the emergency physicians about proper evaluation of other possible causes of sudden collapse and effective holistic approach in management of patients in this class. The limitation of our study was our inability to retrieve the sample of drink(s) and or the glass, which he used in the pub for analysis. Retrieving these objects is strongly recommended in suspicious cases.

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