Outbreak of methanol poisoning at a rural community in Southwest Nigeria: Results of laboratory analysis

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Abstract

Background: In the month of April 2015, an epidemic initially attributed to mysterious causes broke out in a rural community (Ode-Irele, Ondo State) of Nigeria. Twenty-five adults were affected, with a mortality of 18/25 (a rate of 72%). This is a report of laboratory analysis conducted at the College of Medicine, University of Ibadan, and the University College Hospital, Ibadan, Nigeria.

Methods: Samples of blood and urine (both coded A, B, and C) of the three victims of the disease, and three plastic containers of locally brewed alcoholic beverages (coded (D, E, F) reported to have been consumed by the victims were made available. All the samples were tested for copper, lead, zinc, and cadmium toxicity. The blood samples were tested for cholinesterase inhibition as an indirect test of probable pesticide poisoning. Methanol content of the samples was determined by gas chromatography.

Results: The urine and blood samples showed concentrations of lead, copper, zinc, and cadmium that were not uncommonly elevated. Zinc levels in the blood of two of the victims were low. There was a significant inhibition of cholinesterase activity in the three blood samples as follows; sample A 96.2%, sample B 31.8%, and sample C 2.5%. Methanol content of the alcoholic beverages ranged from 48 g/L (4.8%) to 625 g/L (62.5%), far above allowable limits of 0.005 g/L (0.0005%) in Nigeria.

Conclusions: Methanol poisoning was concluded to be the cause of the unknown disease. This finding guided measures that rapidly controlled and eventually ended the epidemic.

Keywords: *Methanol poisoning, Heavy metals, Nigeria*

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Résumé

Contexte : Dans le mois d'avril 2015, une épidémie initialement attribuée à des causes mystérieuses s'est déclarée dans une communauté rurale (Ode-Irele, État d'Ondo) du Nigéria. Vingt-cinq adultes ont été touchés, avec une mortalité de 18/25 (un taux de 72%). Ceci s'agit d'un rapport d'analyse laboratoire réalisée au Collège de Médecine de l'Université d'Ibadan et au Collège Hospitalier Universitaire d'Ibadan au Nigéria.

Méthodes : Des échantillons de sang et d'urine (tous deux codés A, B et C) des trois victimes de la maladie et trois récipients en plastique de boissons alcoolisées brassées localement (codés (D, E, F) qui auraient été consommés par les victimes ont été soumis à des tests de toxicité sur le cuivre, le plomb, le zinc et le cadmium, ainsi que sur l'inhibition de la cholinestérase dans les échantillons de sang, en tant que test indirect d'intoxication probable par un pesticide. La teneur en méthanol des échantillons a été déterminée par chromatographie en phase gazeuse.

Résultats : Les échantillons d'urine et de sang ont montré des concentrations de plomb, de cuivre, de zinc et de cadmium qui n'étaient pas inhabituellement élevées. Les concentrations de zinc dans le sang de deux des victimes étaient faibles. Il y avait une inhibition significative de l'activité de la cholinestérase dans les trois échantillons de sang comme suit; échantillon A 96,2%, échantillon B 31,8% et échantillon C 2,5%. La teneur en méthanol des boissons alcoolisées variait de 48 g / L (4,8%) à 625 g / L (62,5%), bien au-dessus des limites autorisées de 0,005 g / L (0,0005%) au Nigéria.

Conclusions: il a été conclu que l'intoxication au méthanol était la cause de la maladie inconnue. Cette découverte a guidé les mesures qui ont rapidement maîtrisé et éventuellement mis fin à l'épidémie.

Mots clés: *intoxication au méthanol, métaux lourds, Nigéria*

Introduction

Methanol (methyl alcohol) is structurally similar to ethanol and both alcohols share many chemical characteristics and metabolic pathways. When

ingested, methanol is rapidly absorbed and distributed in body tissues in proportion to water content. The lethal dose of methanol has been reported to be approximately 1g/kg, with a wide range of variations [1]. This dose is dependent on individual metabolism, and other factors that include age, liver enzyme activity, and renal function [1, 2]. In Nigeria, the National Agency for Food Drug Administration and Control (NAFDAC) in 2005 put the allowable limits of methanol in alcoholic beverages at 0.0005% (5mg/L) [3]. Epidemic methanol poisoning is rare and when it occurs is often unrecognized and generates public concern.

In the month of April 2015, a series of deaths attributed to unknown and mysterious causes occurred at Ode-Irele, a remote community of Ondo State, Southwestern Nigeria (Figure 1). At the first wave of the epidemic, 25 adults developed sudden onset of vomiting, abdominal pain, and blurred vision that resulted in blindness; the victims rapidly became unconscious within an hour of onset of symptoms. While approximately 18 of the 25 victims of the mysterious disease died, a few remained unconscious for several weeks under close medical care. One factor that was common to all the victims was the consumption of a locally brewed alcohol drink. Initial analysis of blood samples sent to laboratories at Lagos, Nigeria's commercial center, and the Zonal office of the World Health Organization were done on the basis of the possibility of Insecticide and heavy metal poisoning [4]. The mysterious deaths caused community anxiety as the deaths occurred at about the same period of Ebola Virus Disease (EVD) epidemic in Nigeria.

On April 18, 2015, a pathologist with the Ondo State Ministry of Health contacted the Chief Medical Director and sought the assistance of the University College Hospital Ibadan, Nigeria, in the management of five patients who were suffering from clinical effects of the Ode-Irele mysterious disease but were blind and unconscious. The victims of the mysterious epidemic were transferred to the medical ward of the University College Hospital, Ibadan for care and further investigation where they were managed by a team of neurologists, nephrologists, and ophthalmologists. Blood and urine samples that were obtained from the victims were delivered to the laboratory for analysis.

Of the five victims, fresh urine and blood samples were obtained from three; Mr. S.O, a 25 year old man, Mr. G.A a 70 year old man, and Mr. O.J., a 35 year old man, with similar histories of acute onset of vomiting, severe abdominal pain, visual disturbances, and ultimately unconsciousness. They were also reported to have ingested locally brewed alcoholic beverages, samples of which were brought to the hospital. The hospital management assembled a team of experts comprising laboratory scientists, Clinical Pharmacologists, toxicologists, and a Chemical Pathologist to identify the cause of the disease. Blood and urine samples obtained from the three patients were systematically analyzed to identify the toxicants responsible for the clinical state of the patients.

In a 2016 publication, Adeyanju and co-authors described the case identification, epidemiological investigation, and public health response of the methanol intoxication outbreak that we investigated in the laboratory [5]. The authors briefly stated the results of the laboratory analysis carried out at UCH. In this manuscript we are providing the details of the methods used in the laboratory analysis and the results. This will be a ready resource for investigation of similar outbreaks should they occur in the future.

Methods

A systematic approach, as described below, was used in the analysis of samples of the alcoholic beverage reportedly ingested by the victims as well as blood, and urine samples obtained from the three admitted victims of the unknown disease. The samples were analyzed for the presence of heavy metals, pesticides, and methanol in that order. Universal precautions guiding the handling of potentially infectious samples were strictly adhered to throughout the analysis.

Coding of samples

About 7 mL of blood and 40 mL of urine samples were obtained from the patients. Along with these were about 400 mL of three alcoholic beverage said to have been ingested by the victims. Triplicate blood samples were transported in plain, sodium EDTA, and heparinized plastic bottles. Urine samples were transported in 50 mL plain plastic bottles. The blood and urine samples were transported in ice packs. The beverages were transported in large plastic kegs in which they were sold. The samples were obtained at the general hospital at Ondo State, and sent immediately to the University College Hospital Ibadan, before the victims were transferred to Ibadan. On arrival at the laboratory the samples were coded in the following order, S.O. (sample A), G.A. (sample B), and O.J. (sample C). The alcoholic beverages reportedly ingested by the patients were also coded as beverage D, beverage E, and beverage F; it was not certain which alcoholic beverage was taken by

which victim. Samples were kept in a 4°C refrigerator; analyses were done within 48 hours of receiving the samples. It is however noteworthy that the samples were obtained from the victims three days after the acute onset of symptoms.

Analysis for zinc, copper, lead, and cadmium
This was done by flame Atomic Absorption Spectrophotometry (AAS). The analyses were carried out at the Multidisciplinary Central Research Laboratory (MCRL) of the University of Ibadan, Nigeria. Double distilled deionized water (ddH_2O) was used as negative control and a calibration curve was prepared using certified laboratory reference standards of the heavy metals analyzed. All methods complied with those reported in the manual by Perkin Elmer [6], except for the analysis of blood lead levels in which nitric acid digestion was done. Only analytic grade reagents were used. All the samples (beverage, blood, and urine) were analyzed for Pb, Zn, Cu, and Cd. Two repeat measurements were done for every sample. The alcoholic beverages and urine samples were centrifuged at 4000g for 7 minutes and aliquots (4mL) taken from the supernatants. The aliquots of beverages and urine were diluted with ddH_2O to a dilution ratio of 1:5 to a final volume of 20 mL before injection into the flame AAS. Final contents of the metals were determined after due considerations of the dilution factors.

Analysis of blood for zinc, copper, lead, and cadmium
Clotted blood samples were centrifuged at 4000g for 7 minutes and serum was aspirated into plain plastic bottles. Aliquots of the serum were taken and diluted with ddH_2O to a dilution of 1:5 before injection into the flame AAS for determination of Zn, Cu, and Cadmium. Whole blood was used for analysis of lead levels. Lead extraction was done using the nitric oxide digestion method before analysis with AAS. Briefly, 3.8 mL of 0.1 N nitric acid was added to 0.2 mL of whole blood and allowed to stand for > 6 hours (overnight) at 4 °C (to liberate bound lead). The solution was then centrifuged at 4000g for 10 minutes. The clear supernatant was diluted with ddH_2O to a ratio of 1:5 before aspiration into the flame AAS.

Evaluation for pesticide intoxication
The possibility of pesticide intoxication was assessed indirectly by determination of cholinesterase activity. The test was performed using heparinized blood obtained from the three victims. Whole blood acetylcholinesterase enzyme activity was determined according to the modified Ellman's method as published by Worek *et al.* in 1999 [7]. Briefly, whole blood (control and samples from the victims) was diluted a hundred times in 0.1M sodium phosphate buffer (pH 7.4) containing 0.03% Triton-X-100. Acetylcholine (28.4 mM) and DTNB (10mM) were added to the mixture and incubated at 37° C for 10 minutes. Absorbance was read at 436 nm in a UV-VIS Spectrophotometer (BIO-RAD, SMART-SPEC PLUS, USA). An inhibition of 30% is considered clinically significant.

Analysis for methanol and ethanol levels
Determination of methanol and ethanol concentrations in the beverages, blood, and urine samples was done by gas chromatography using the method described by Helena Pontes *et al* [8]. Reagents, calibration procedures, sample preparation, and instrument setting were as described in the publication. The beverages were centrifuged at 4000g for 7 minutes, supernatants taken and diluted with ddH_2O to 1:5 dilution; 0.5 µL of the supernatants were injected sequentially into the chromatographic system. Urine samples were similarly treated and diluted before injection into the chromatographic system. Plasma was then prepared as described by Helena Pontes *et al* [8]. Prepared plasma was then diluted to 1:5 using ddH_2O before injection into the chromatographic system. All the chemicals used were of analytical grade.

Results

Heavy metal contents in samples of blood, urine, and beverage from the victims Zinc, copper, lead and cadmium
The urine and blood samples showed concentrations of Pb, Cu, Zn, and Cd that were not out of acceptable limits. Zinc levels in the blood samples of patients A and B were low. Analysis of the alcoholic beverages showed concentrations of analyzed metals within recommended allowable limits. Table 1 shows a summary of the results of metals analysis. It should be noted that alcoholic beverage coded D, E, and F do not correspond to patients A, B, and C. as it was not certain the alcoholic beverage each of the patients ingested.

Analysis for possible pesticide toxicity
Cholinesterase activity: There was a significant inhibition of the activity of the enzyme in the three blood samples as follows; average values of inhibition were sample A 96.2%, sample B 31.8%, and sample C 2.5%.

can be life-threatening [9]. Victims of methanol poisoning often seek medical care after significant delay, mainly because there is a lag period between ingestion and toxic effects and due to protean nature of signs and symptoms [10].

Methanol levels were very high in the alcoholic beverages consumed by the victims of the epidemic reported in this paper. However, the source of methanol in the alcoholic beverages remains to be determined. Possible sources of the contamination could have been from illegal addition of methanol by vendors or contamination during the fermentation process [3]. Epidemic methanol poisoning of this scale had not been previously reported from Nigeria to the best of our knowledge and this may have led to a delay in recognizing the cause of the symptoms and deaths that occurred. Levels of methanol detected in the blood and urine of the victims tested supported the diagnosis of methanol poisoning. It is also of note that the blood' and urine levels detected may have been reduced by the time the samples were collected. The level of inhibition of acetylcholinesterase detected in the analysis could be attributed to the effects of methanol, bearing in mind that this could also be due to other causes that may include exposure to organophosphates.

Low levels of zinc were also detected in the victims tested. Zinc is a micronutrient required for the activities of over 300 enzymes, including alcohol dehydrogenase (ADH), may have been reduced where there is high demand. There is evidence that severe infections and other forms of stress, particularly when encountered in hospitalized adults or accompanied by fever or other indicators of an acute phase response, produce a drop-in plasma zinc concentration [11]. Probably, zinc levels were low in the blood samples of the victims on account of increased demand for Zn due to increased metabolism of methanol during the illness. This may have contributed to the progression of the observed signs of methanol poisoning in the victims including the loss of vision. There is a need to conduct studies aimed at determining the relationship between methanol toxicity and plasma concentration of zinc.

Once ingested, methanol is metabolized by dehydrogenation to formaldehyde and then to formic acid leading to profound acidosis. These two metabolites are highly reactive, binding readily to tissue proteins with resultant inhibition of metabolic enzymes and cytochrome oxidase systems [2, 12]. The toxic effects and ocular effects of methanol are attributable to toxicity of formic acid [13, 14]. The same enzymes responsible for ethanol metabolism are responsible for the metabolism of methanol with higher Km for methanol. Presence of ethanol will inhibit the metabolism of methanol. Clinical features of toxicity have been shown to be dose-dependent, and can be ameliorated by concomitant administration of ethanol diverting the pathway to that of intermediates less toxic than formic acid.

Accidental ingestion of methanol-containing solvents in homes is an alternative source of toxicity. Methanol-containing products may be found in homes, as constituents of a large variety of solvents, chemicals, and pesticides. However, epidemics of methanol poisoning are more likely the result of ingestion of alcoholic products containing high concentrations of methanol. In 1951, an epidemic of methanol poisoning was reported to have occurred in Atlanta [15]. The epidemic was caused by city-wide distribution of methanol containing illicit whisky. Over a five-day period, a total of 323 cases, including 41 deaths, occurred during the epidemic [15]. An outbreak of acute methyl alcohol intoxication was also reported to have occurred in Port Moresby, Papua New Guinea, in March 1977. Twenty-eight young men attended a drinking party and drank methyl alcohol. All 28 became ill within 8 to 36 hours after drinking and were hospitalized. The most commonly observed clinical syndromes among the Port Moresby patients were: acute metabolic acidosis, severe visual impairment and acute pancreatitis. Four died within 72 hours after admission to the hospital [1]. Another report of acute methanol poisoning from Chaoyang County [17]. The Chaoyang episode was due to poisoned wine which caused acute vertigo, headache, weakness, vomiting, night sweat, dyspnea and blurring of vision within 6 to 120 hours of ingestion. Twenty-nine people were reported to have drunk the wine, fourteen of them died, two of them became blind. On further analysis, high content of methanol was detected in the spirit; the victims' blood and urine also had methyl alcohol. Laboratory analysis showed that the content of methyl alcohol was between 16.6 and 40.69 g/100 mL [17]. The Ode-Irele methanol poisoning epidemic is consistent with previously reported occurrences.

Methanol toxicity occurs uncommonly worldwide, it is usually not recognized early and causes a lot of confusion in the society. The diagnosis is often elusive and requires a high index of suspicion. The Nigeria epidemic reported in this case occurred in an area that is within the belt stretching from the Midwestern to the South-South regions of the country where locally brewed alcohol is widely produced and consumed.

Victims of the epidemic developed acute episodes of severe abdominal pain, vomiting seizure, visual disturbances and loss of consciousness within

Table1: Heavy metal contents (Mean mg/L ± SD) in samples of blood, urine, and beverage from the victims

Sample	Heavy metal	Level of metals in individual patient's samples		
		A	B	C
Blood	Lead	0.00 ± 0.03	0.00 ± 0.03	0.00 ± 0.07
	Cupper	0.06 ± 0.00	0.28 ± 0.05	0.28 ± 0.09
	Zinc	0.19 ± 0.01	0.41 ± 0.02	1.21 ± 0.01
	Cadmium	0.00 ± 0.01	0.00 ± 0.01	0.01 ± 0.01
Urine	Lead	0.06 ± 0.03	0.09 ± 0.05	0.00 ± 0.07
	Cupper	0.00 ± 0.02	0.00 ± 0.01	0.05 ± 0.01
	Zinc	0.19 ± 0.02	0.11 ± 0.01	0.05 ± 0.01
	Cadmium	0.03 ± 0.01	0.05 ± 0.01	0.05 ± 0.01
Beverage	*Heavy metal*	*D*	*E*	*F*
	Lead	0.04 ± 0.05	0.00 ± 0.04	0.00 ± 0.01
	Cupper	0.19 ± 0.02	0.00 ± 0.01	0.01 ± 0.01
	Zinc	0.00 ± 0.01	0.00 ± 0.02	0.00 ± 0.02
	Cadmium	0.00 ± 0.01	0.00 ± 0.01	0.01 ± 0.01

SD = Standard Deviation

Table 2: Average Methanol and Ethanol levels in the blood, urine, and beverages (g/L and percentage alcohol)

Sample		Methanol content - g/L	Ethanol content – g/L
Blood	A	1.40 (0.14%)	0.695 (0.07%)
	B	1.05 (0.11%)	1.095 (0.12%)
	C	0.25 (0,03%)	0.235 (0.02%
Urine	A	0 (0%)	0.04 (0.004%)
	B	0.18 (0.02%)	0.015 (0.001%)
	C	Inadequate sample	Inadequate sample
Beverage	D	625 (62.5%)	127.5 (12.75%)
	E	48 (4.8%)	428.5 (42.85%)
	F	325 (32.5%)	205 (20.5%)

Methanol and Ethanol levels in the blood, urine, and beverages

Methanol content: Methanol was detected in the blood in concentrations above tolerable limits. Methanol content of the alcoholic beverages was also above allowable limits. Table 2 shows a summary of findings of tests for methanol and ethanol contents.

Discussion

Methyl alcohol (Methanol) is a widely available solvent with industrial and domestic applications. Methanol has a relatively low intrinsic toxicity; however, it is metabolized to highly toxic compounds, such as formic acid which can cause blindness, coma, and metabolic disturbances and that

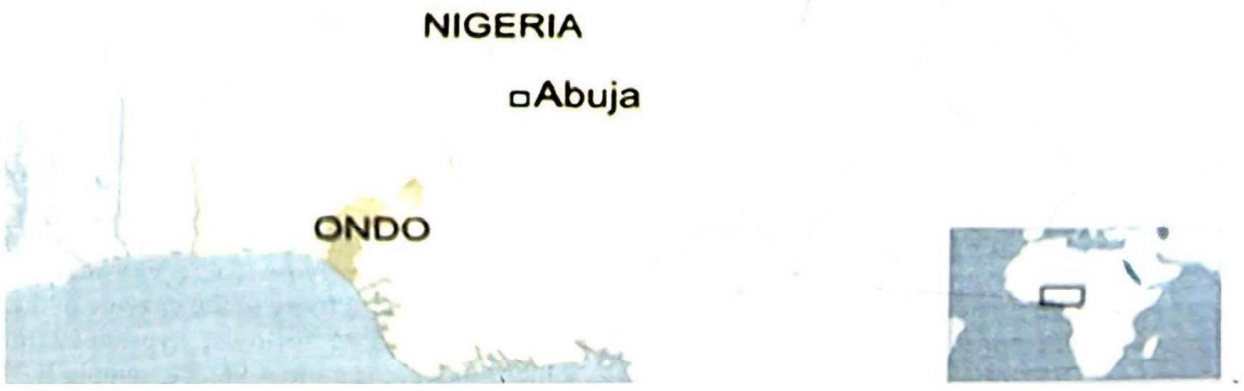

Fig. 1: Map of Nigeria showing Ondo State

humans. Annals of occupational and environmental medicine. 2017 Dec; 29 (1): 44-49.

2. Nicholls P. The effect of formate on cytochrome aa3 and on electron transport in the intact respiratory chain. Biochim Biophys Acta 1976; 430 (1): 13-29.
3. Ohimain EI. Methanol Contamination in Indigenous Fermented Aloholic Beverages. In: Food Safety and Regulatory Measures International Conference; 2015 17 - 19 August 2015; Birmingham, UK; 2015.
4. WHO ARO. Unknown disease in Ondo State, Nigeria. In: World Health Organization; 2015.http://www.afro.who.int/en/clusters-a-programmes/dpc/epidemic-a-pandemic-alert-and-response/outbreak-news/4609-unknown-in-ondo-state-nigeria.html. (accessed on 12th October 2016)
5. Adeyanju D, Fatiregun AA, Ekundare-Famiyesin O, *et al.* Investigation of an Outbreak of Acute Methanol Poisoning in a Southwest State of Nigeria. International Journal of Tropical Disease & Health. 2016; 14 (4): 1-8.
6. Elmer P. Analytical Methods for A.A.S. In: Analytical Methods for Absorptin Atomic Spectroscopy, the PerkinElmer Inc.; 1966 (Revised 1996).
7. Worek F, Mast U, Kiderlen D and Diepold C,P.E. Improved Determination of Acetylcholinesterase Activity in Human Blood. Clinica Chimica Acta 1999; 288: 73 - 90.
8. Pontes H, Guedes de Pinho P, Casal S, *et al.* GC determination of acetone, acetaldehyde, ethanol, and methanol in biological matrices and cell culture. J Chromatogr Sci 2009; 47 (4): 272-278.
9. Barceloux DG, Bond GR, Krenzelok EP, Cooper H and Vale JA. American Academy of Clinical Toxicology practice guidelines on the treatment of methanol poisoning. J Toxicol Clin Toxicol 2002; 40 (4): 415-446.
10. Hovda KE, Hunderi OH, Tafjord AB, *et al.* Methanol outbreak in Norway 2002-2004: epidemiology, clinical features and prognostic signs. J Intern Med 2005; 258 (2): 181-190.
11. Brown KH. Effect of infections on plasma zinc concentration and implications for zinc status assessment in low-income countries. The American journal of clinical nutrition. 1998 Aug 1; 68 (2): 425S-9S.
12. Shahangian S and Ash KO. Formic and lactic acidosis in a fatal case of methanol intoxication. Clin Chem 1986; 32 (2): 395-397.
13. Jacobsen D and McMartin KE. Methanol and ethylene glycol poisonings. Mechanism of toxicity, clinical course, diagnosis and treatment. Med Toxicol 1986; 1 (5): 309-334.
14. Martin-Amat G, McMartin KE, Hayreh SS, Hayreh MS and Tephly TR. Methanol poisoning: ocular toxicity produced by formate. Toxicol Appl Pharmacol 1978; 45 (1): 201-208.
15. Methyl alcohol poisoning; an account of the 1951 Atlanta epidemic. J Med Assoc Ga 1952; 41 (2): 48-51.
16. Naraqi S, Dethlefs RF, Slobodniuk RA and Sairere JS. An outbreak of acute methyl alcohol intoxication. Aust N Z J Med 1979; 9 (1): 65-68.
17. Xiao JH. [Accidental methyl alcohol poisoning]. Zhonghua Yu Fang Yi Xue Za Zhi 1990; 24 (3): 160-162.

five days of ingestion of local alcohol. Initially, the community attributed the deaths to spiritual 'attacks' and the people blamed the 'gods' for the disease. The laboratory investigation conducted at the University College Hospital, Ibadan, provided the first evidence that implicated methanol as the cause of the mysterious disease. An important observation was that after collation of all laboratory data, a remarkable congruence or correlation was strikingly evident with clinical data; the patient with the lowest blood concentration of methanol recovered faster than the other victims whose samples were analyzed. These results guided the government's measures that rapidly ended the epidemic. Confirmations of the findings by other laboratories added to the evidence provided by the research team at Ibadan.

In developing nations, rare epidemics (when they occur) pose many challenges to community healthcare institutions due to unpreparedness. The delay in recognition of the etiology causes panic and unnecessary deaths. In addition, due to the epidemic being uncommon, index of suspicion is usually low. In the case reported here the affected community within the local alcohol belt described above should have raised index of suspicion. An additional challenge was the lack of readiness of the laboratory in conducting the required analyses. Most of the reagents used in the analysis were not available and had to be rapidly procured during the epidemic. The methods used were run for the first time and analysis for formic acid content could not be done. Such are some challenges faced in resource-scarce countries, however, with the close collaboration of several disciplines (clinical and laboratory) and full support of the hospital management, the team was able to identify the cause of the widely reported 'mysterious disease'. The success reported here showed that even with little resources, with the right leadership and human resources, a lot can be achieved locally. This calls for need to have well equipped toxicology laboratories to avoid unnecessary morbidity & mortality. In addition, it is recommended that index of suspicion should be kept high especially when episodes occur within the belt described above. Laboratory and human capacity should be strengthened for rapid response in similar circumstances. In the present era of infectious disease epidemics, healthcare workers and stakeholders should remain vigilant for epidemic or sporadic cases of methanol poisoning or other uncommon conditions that may occur at any time. The government should put in place mechanisms of routinely sampling locally made alcohol for methanol content to prevent future outbreaks of methanol poisoning while the dangers of illicitly brewed alcohol should be made known to communities.

Conclusions

The clinical and laboratory findings in affected victims during the widely reported epidemic of a mysterious disease in the month of April 2015 in Southwest Nigeria was due to consumption of locally brewed alcohol contaminated with high concentrations of methanol. The findings of associated inhibition of acetylcholinesterase, low levels of zinc in tested victims, and the high degree of correlation between laboratory and clinical presentation support the diagnosis. It is recommended that rapid analysis of beverages, blood, and urine be carried out should there be fresh cases of the disease, with prompt and appropriate management of victims at tertiary hospitals. The public should be informed of the dangers of locally brewed alcohol; in the communities affected by the disease. People should be admonished to stop drinking improperly brewed alcohol and to be aware of the possibility of contamination with methanol. Finally, one key lesson from this report is that with increasing urbanization and industrialization the risk of chemically- induced disease should be given high index of suspicion and priority in Nigeria and many other developing countries.

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References

1. Moon CS. Estimations of the lethal and exposure doses for representative methanol symptoms in