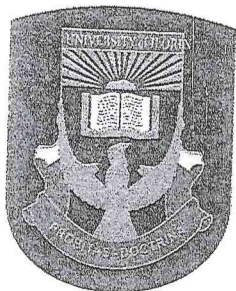


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Honey Prevents Lead Induced Renal Dysfunction By Improving Antioxidant Activities in Male Wistar Rats

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Abstract

This study investigates the protective potential of Honey against lead-induced renal dysfunction. Thirty-two male Wistar rats were used, and they were divided into 4 groups A-D (n=8). The control group (A) were given 1ml/kg distilled water orally; Lead group (B) received 0.2% lead acetate in drinking water; Group (C) and Group (D) rats were given 0.2% lead acetate in drinking water and 1.0 ml/kg and 1.5 ml/kg natural honey respectively for 4 weeks. At the end of the exposure period, animals were sacrificed, blood samples were collected and centrifuged. The plasma was thereafter separated for measurements of blood urea and creatinine. Also right kidneys were excised and homogenized in 0.25M sucrose solution. The homogenate was centrifuged and supernatants collected for estimation of total protein, malondialdehyde (MDA), antioxidant levels: reduced glutathione (GSH), glutathione s-transferase (GST), catalase and superoxide dismutase (SOD) in the kidney.

Results showed significant ($P < 0.05$) increase in the mean MDA of kidney of lead acetate group (Group B) with decreased total protein and antioxidant enzyme (GSH, GST, SOD,) activities. Also, significant increase levels of urea and creatinine were observed. However, honey alleviated the increased MDA levels, increased antioxidant enzyme activities and ameliorate the elevated urea (low dose honey) but not creatinine in the honey treated groups. Thus, the present results indicate that natural honey may have modulating effect on the damage in kidney cells from oxidative stress induced by lead toxicity in rats.

Keywords: Antioxidant, Honey, Lead, Renal.

Introduction

Lead is a well-known toxicant that affects virtually all body systems. It has been described as a non-essential toxic heavy metal that is widely

distributed in the environment and chronic exposure to low levels of lead has been a matter of public health concern in different parts of the World including Nigeria¹. It is a naturally occurring toxic metal like mercury, arsenic, vanadium etc.

Lead paint is the primary source of lead exposure and the major source of lead toxicity especially in children. Other sources include; drinking water, lead-glazed ceramics, cigarette smoke, lead solder used in canned foods etc². Lead exposure causes many undesired effects on different organs/tissues including brain and kidney³. There is no known safe blood lead concentration, but it is believed that, as lead exposure increases, the range and severity of symptoms and effects also increases.

One of the known mechanisms through which lead causes its toxicity is through induction of oxidative stress⁴ which is usually associated with imbalance between the production of free radicals and the ability of the body to counteract or detoxify their harmful effects through neutralization by antioxidants⁵. And this can lead to undesired effect in various organs of the body.

Preventive measures are preferred over the treatment remedies. Considering the toxic effects of lead, once it gets to the circulation, it is almost impossible to remove it completely or to reverse its damaging effects on the body⁶. Among the natural substances that have been investigated to have nephroprotective effects against lead induced toxicity are: curcumin⁷, green tea⁸, and honey⁹ etc. This protective role has been linked to the potent antioxidant property of those agents.

Honey has been known since ancient times to have protective and curative properties. It is a remarkable liquid, prepared by honey bees from the natural solutions called nectar obtained from various flowers. It is believed that some of the constituents of honey may act synergistically to produce its antioxidant effect¹⁰. Judging from the fact that amongst the natural medicinal products commonly used in Nigeria for management/treatment of various types of ailments, honey is one of such, it is therefore important to study its effects on renal dysfunction associated with lead exposure in Wistar rats with a specific focus on its antioxidant property.

Though, other studies have also reported that honey has nephroprotective property against lead induced toxicity^{9,11}. But there is assumption that varying total phenol content and antioxidant capacity is

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expected for honey produced from diverse floral sources having different geographic regions. Hence, this study made use of UNILORIN honey which we have previously reported to possess anti-inflammatory, anti-nociceptive and neuroprotective properties¹¹.

Materials and Methods

Animals

Thirty-two (32) male Wistar rats weighing 170-190g were procured from the department of Biochemistry University of Ilorin, Ilorin. The animals were housed at the animal holding of the College of Health Sciences, University of Ilorin, Ilorin, under normal atmospheric conditions. They were fed with grower's mash purchased from Belkalid consult Ilorin and also given free access to water *ad libitum*. The animals were given a two weeks period of acclimatization before the commencement of the experiment.

Animal Grouping and Drug Administration

The animals were randomly divided into four groups (A-D) as follows:

Group A (control) received 1ml/kg distilled water orally for 28 days.

Group B received (0.2%) lead acetate through drinking water for 28 days and 1ml of distilled water orally for 28 days.

Group C and D rats were given (0.2%) lead acetate through drinking water and at the same time received daily oral administration of honey with group C taking 1ml/kg and group D 1.5ml/kg for 28 days.

Preparation of Drugs

0.2% Lead

Lead acetate was obtained from Guanghua Chemical Factory Ltd., China. 0.2% solution was prepared by dissolving 10g of lead acetate in 5L of distilled water.

Honey

Unilorin honey was obtained from honey research institute of University of Ilorin. It was mixed with distilled water in a ratio 1:1.

Animal Sacrifice

At the end of the 28 day administration, the animals were anaesthetized using 50 mg/kg ketamine. Blood samples were collected through cardiac puncture into heparinized bottle. Thereafter, the blood was centrifuged at 3000rpm for 15 minutes. Plasma was then separated and stored at -20°C for biochemical assays. Right Kidney was also collected from each animal, washed using cold normal saline solution, weighed and then homogenized with 0.25M sucrose solution. Homogenates were also centrifuged at 3000 rpm for 15 minutes and the resulting supernatant was

stored for biochemical analysis.

Biochemical Analysis

Kidney function tests

The plasma samples were used in the determination of kidney function tests: creatinine and urea nitrogen. The two tests were determined spectrophotometrically using standardized commercially available kits (Randox Laboratory Limited).

Determination of protein

The protein content was measured according to the method of Lowry et al. (1951). A standard curve was prepared as follows. Bovine serum albumin (BSA) powder was dissolved in distilled water and diluted to a concentration of 1 µg/µl. A series of dilutions (0, 1, 2.5, 5, 10, and 20 µg/well) were made in replicates of 4 with a final volume of 100 µl. Samples were diluted such that they would fall within the BSA standard range (0-25 µg / 100 µl) and 100 µl placed in each well. After standards and samples were diluted and transferred to the microplate, 200 µl of biuret reagent was added to each well and mixed thoroughly with repeated pipeting. Biuret reagent was prepared by mixing 0.5 ml of 1% cupric sulfate with 0.5 ml of 2% sodium potassium tartrate, followed by the addition of 50 ml of 2% sodium carbonate in 0.1 N NaOH. The mixture was then allowed to incubate at room temperature for 10-15 minutes prior to the addition of 20 µl per well of 1.0 N Folin & Ciocalteu's reagent. Samples were mixed immediately with repeated pipeting with each addition. Color was allowed to develop for 30 minutes at room temperature and the absorbance measured at 650 nm and blanked on the water only control. Protein determined was used for the calculation of GST, Catalase and MDA activities.

Determination of Malondialdehyde (Mda)

The concentration of malondialdehyde in the kidney was determined according to the procedure described by Reilly et al. (1999)¹³ for assessment of lipid peroxidation. Briefly, 0.4ml of test sample was mixed with 1.6 ml of Tris-KCl buffer to which 0.5ml of 30% Trichloroacetic Acid (TCA) was added. Then 0.5ml of 0.75% Thiobarbituric Acid (TBA) was added and placed in a water bath to incubate for 45 minutes at 80°C. The incubation produced a pink coloured reaction mixture which was then cooled in ice and centrifuged at 14000g for 15 minutes. The absorbance of the clear pink supernatant was then read at 532nm. Calculation:

$$\text{MDA (units/mg protein)} = \frac{\text{absorbance} \times \text{volume of mixture}}{E_{532} \times \text{volume of sample} \times \text{mg protein}}$$

Where E_{532} is molar absorptivity at 532nm = 1.56×10^5

Antioxidant Activities

Determination of Catalase Activity

Catalase activity was determined by the method described by Beers and Sizer (1952)¹⁴. 1.9ml of Distilled water was added to 1ml of Hydrogen peroxide (0.059M) and Incubated in spectrophotometer for 5 min to achieve temperature equilibration and to establish blank rate. Then 0.1ml of Enzyme source (appropriately diluted) was added. Decrease in absorbance was monitor at 240nm for 2 min immediately after the addition of the appropriately diluted enzyme source.

Calculation:

Change in absorbance ($\Delta A_{240}/\text{min}$) was calculated from the initial 45 seconds.

CAT activity was obtained from the following expression:

$$\text{Catalase activity (Units/mg)} = \frac{\Delta A_{240}/\text{min} \times 1000}{43.6 \times \text{mg protein/ml reaction mixture}}$$

Determination of Glutathione S-Transferase Activity

Glutathione S-transferase (GST) activity was determined according to the method of Habig et al., (1974)¹⁵. 30 μ l of reduced glutathione, 150 μ l of CDNB, 2.79ml of 0.1M phosphate buffer were added to 30 μ l of sample. Blank was prepared by mixing 30 μ l of reduced glutathione, 150 μ l of CDNB, 2.79ml of 0.1M phosphate buffer together. The reaction was allowed to run for 60 seconds each time before the absorbance was read against a blank at 340nm. The temperature was maintained at approximately 31°C.

Calculations:

The extinction coefficient of CDNB = 9.6mmol⁻¹cm⁻¹

$$\text{GST activity} = \Delta \text{absorbance} \times \frac{1}{\begin{matrix} \bullet & \text{mg/0.03ml protein} \\ & = \mu\text{mole/min/mg protein} \end{matrix}}$$

Determination of Superoxide Dismutase (SOD) Activity

SOD activity was determined by the method of Misra and Fridovich, (1972)¹⁶. 1ml of sample was diluted in 9ml of distilled water to give 1 in 10 dilutions. An aliquot of the diluted sample was added to 2.5ml of 0.05M carbonate buffer pH 10.2 to equilibrate in the spectrophotometer and the reaction started by the addition of 0.3ml of freshly prepared 0.3m M adrenaline to the mixture which was quickly mixed by inversion. The reference cuvette contained 2.5ml buffer, 0.3ml of substrate (adrenaline) and 0.2ml of distilled water. The increase in absorbance at 480nm was monitored every 30 seconds for two and half minutes.

Calculations:

Increase in absorbance per minute = $A_3 - A_0 \times 2.5$

where A_0 = absorbance after 30 seconds

A_3 = absorbance after 150 seconds

$$\% \text{ Inhibition} = \frac{\text{Increase in absorbance for substrate} \times 100}{\text{Increase in absorbance of blank}}$$

1 unit of SOD activity was given as the amount of SOD necessary to cause 50% inhibition of the oxidation of

adrenaline to adrenochrome during 1 minute.

Determination of Reduced Glutathione (GSH)

The level of reduced glutathione (GSH) in the samples will be determined by the method described by Jollow et al (1974). 0.2 ml of the sample was added to 1.8ml of distilled water and 3 ml of the precipitating reagent was mixed with the sample. The mixture was then allowed to stand for 5 minutes and then filtered. At the end of the fifth minutes, 1ml of the filtrate was added to 4 ml of 0.1M phosphate buffer and finally, 0.5 ml of the Ellmans' reagent was added. A blank was prepared with 4 ml of the 0.1M phosphate buffer, 1 ml of diluted precipitating solution and 0.5 ml of the Ellman's reagent. The optical density was measured at 412 nm. GSH concentration was proportional at that wavelength and the estimate was obtained from the GSH standard curve.

Statistical Analysis

All results were reported as mean SEM (Standard Error of mean). Statistical comparisons between different groups were done using one-way Analysis of Variance (ANOVA) followed by Duncan post-hoc test and $p < 0.05$ was taken to be significant.

Results

Effect of Pb²⁺ Exposure on Kidney Function Parameters

Exposure to Pb²⁺ causes a significant reduction in total protein and increase levels of urea and creatinine compared to the control group. This is indicated in table 1 below. Meanwhile, when Pb²⁺ was co-administered with honey, level of urea significantly reduced and total protein increased compared to Pb²⁺-group but high dose of honey has no significant effect on urea (table 1). Also, honey administration has no significant effect on the level of creatinine both at low and high doses.

Effect of Pb²⁺ On Antioxidant Enzymes

Antioxidant levels; reduced glutathione (GSH), glutathione s-transferase (GST) and superoxide dismutase (SOD) activities were significantly decreased (table 2) when rats were exposed to lead compared to control group. But in Pb²⁺ + honey groups, their levels showed significant increase compared to Pb²⁺ group. Meanwhile, effect of Pb²⁺ on CAT activity was considered not significant.

Effect of Pb²⁺ On Lipid Peroxidation

There is significant increase in the

Table 1: Plasma levels of total proteins, urea and creatinine in different studied groups.

Variables	Control	Pb ²⁺ group	Pb ²⁺ +LDH	Pb ²⁺ +HDH
Total protein (μg/dl)	0.43±0.01	0.311±0.01*	0.34±0.01 [#]	0.355±0.01 [#]
Urea (μg/dl)	88.68 ±0.52	115.27±3.12*	83.21±2.39 [#]	136.17±10.77
Creatinine (μg/dl)	100±6.67	139±9.51*	140.16±3.18	146±3.35

Data are presented as Mean± SEM (n=8). * indicates significant difference compared to the control group. [#] indicates significant difference compared to Pb²⁺ treated groups (P< 0.05). LDH=low dose honey, HDH=high dose honey.

Table 2: Tissue levels of reduced glutathione (GSH) and enzymes activities of glutathione S -transferase (GST), catalase (CAT) and superoxide dismutase (SOD) in different studied groups.

Variables	Control	Pb ²⁺ group	Pb ²⁺ + LDH	Pb ²⁺ + HDH
GSH(mmol/g)	158 ± 29.35	142 ± 4.09*	144.4±17.54 [#]	151.17 ±22.24 [#]
GST(μmole/min/mg)	154.17±1.40	111.77±1.04*	122.44±2.0 [#]	119.52±0.36 [#]
SOD(unit/mg protein)	5.2 ±0.49	2.7 ±0.67*	4.7 ±0.42 [#]	4.9 ±0.40 [#]
CAT(μmole.min ⁻¹ .mg protein ⁻¹)	37.05 ±05	39.46 ±2.47	39.3 ±8.42	40.66 ±4.04

Data are presented as Mean± SEM (n=8). * indicates significant difference compared control groups. [#] indicates significant difference compared to Pb²⁺ treated groups (P< 0.05). LDH=low dose honey, HDH=high dose honey.

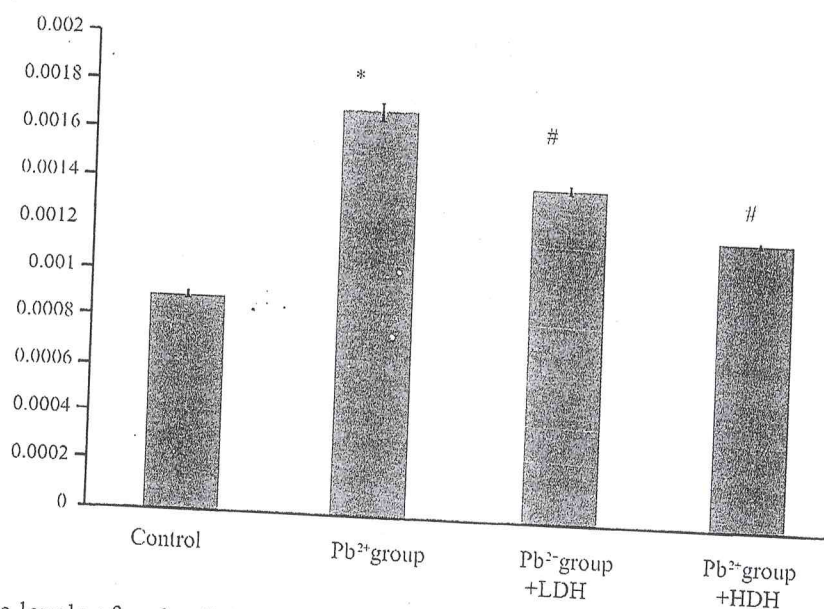


Figure 1: Tissue levels of malondialdehyde (MDA) in different studied groups.

*Indicates significant difference compared to the control group. [#] indicates significant difference compared to Pb²⁺ treated groups (P< 0.05). LDH=low dose honey, HDH=high dose honey.

malondialdehyde (MDA) of kidney of rats exposed Pb²⁺ compared to control indicating increase lipid peroxidation. Meanwhile it is reduced in Pb²⁺ + honey groups (as shown in figure 3).

Discussion

In the present study, exposure to lead acetate causes alteration in renal function parameters which

was reflected in the Pb²⁺ group with elevated plasma levels of creatinine and urea compared to the control group. In addition, decreased total protein, GSH, GST, SOD and enhanced lipid peroxidation were observed confirming increased oxidative stress and reduced antioxidant activities. However, renal dysfunction was prevented with the administration of honey to the lead exposed group.

Elevated urea and creatinine observed in Pb^{2+} group might be an indication of acute renal toxicity¹⁷ which is an example of lead associated tissue injury. This is in line with other studies¹⁸. Although Odigie *et al.*, (2004) reported that onset of renal function abnormalities appear to be related to the duration and dose of lead administration. The elevated urea and creatinine might be due to glomerular insufficiency and production of reactive oxygen species (ROS) as a result of effects of Pb^{2+} on membrane, DNA and antioxidant defense systems of cells¹⁹. Honey treatment ameliorate the kidney function (although not in the case of creatinine) which might be attributed to the several antioxidant components of honey that help to effectively inactivate the generated ROS and terminate the radical chain reaction²⁰ although only the low dose has significant effect in this study.

Decreased total protein in Pb^{2+} group might result from loss of protein which might arise from kidney dysfunction with protein excretion in urine. Honey treated groups showed an improvement in the level of protein. This might be due to the maintained level of membrane-bound enzymes and the activities of antioxidant enzymes²⁰.

Antioxidant enzymes (SOD, GSH, and GST) which were reduced in Pb^{2+} groups might be due to the effect of lead as it binds to the sulfhydryl group present in them and make them inactive²¹. The present study results indicated that honey co-administration with Pb leads to increase in the activities of antioxidant enzymes and thus can be said to eliminate the toxic effects of lead. The kidney level of malondialdehyde (MDA) was increased significantly in Pb^{2+} group compared to the control indicating increased lipid peroxidation. But it was reduced in honey + Pb^{2+} groups. This is correlated to the ability of Pb^{2+} to generate ROS that induce oxidative damage in kidney and then enhance lipid peroxidation.

Several works have been published on the mechanism of Pb^{2+} related pathologies many of which are direct result of oxidant effect of Pb^{2+} on tissue and cellular components which may be mitigated by improving the cellular availability of antioxidants. Green tea, selenium and alpha tocopherol have all been shown to produce antioxidant effects against lead-induced reactive oxygen species generation. They help to normalize SOD, GSH, GST (and others) which are antioxidants that could have helped the cells in scavenging the reactive oxygen species generated from lead exposure.

In conclusion, honey in this study helps to prevent lead-induced renal dysfunction by increasing antioxidant capacity of the cells in addition to its ability to prevent reactive oxygen generation as reported by other authors^{6,11}.

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Suspected Child Abuse of an Infant to the Point of Death by the Father - Need for Enforcing Protection Acts for Nigerian Children

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Abstract

Child abuse occurs when a parent or caregiver, whether through action or failing to act, causes injury, death, or risk of serious harm to a child. There are many forms of child maltreatment, including neglect, physical abuse, sexual abuse, exploitation, and emotional abuse. It is a common phenomenon that can be easily missed by the health workers, thus it has a potential of becoming perpetuated. Some are characterized by tell-tale signs that give the diagnosis away very easily while some are not. It is generally acceptable in several African cultural settings for a parent or caregiver to "discipline" his or her child or ward by spanking or by any other physical measures than 'spare the rod and spoil the child'

Case of an eleven-month old female child who was assaulted by her father while 'disciplining her' with resultant fatal outcome was reviewed. A review of some of the United Nations Articles and Acts on protection of the child and the merits or otherwise in the context of our cultural practice are highlighted.

An eleven-month old female child recuperating from a febrile illness was physically abused by her father with resultant massive subdural haemorrhage only discovered at post mortem.

The child was killed probably by the rage and over reaction of the father in attempt to discipline her. Protection Articles and Acts in the Nigerian Constitution and indeed those of other developing countries should be emphasized and enforced, given better publicity to reduce the magnitude of child abuse.

Keywords: Child Abuse; Protection Acts; Nigerian; Children

Case report

GT was an 11-month old female child that was in a stable state of health until a week prior to presentation when child was brought to the Emergency

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Paediatric Unit (EPU) by both parents with complaints of fever of one week and loss of consciousness of 16 hours duration. Fever was high and intermittent, worse in the evenings. There was no history to suggest any focus of infection. There was no history of diarrhea or vomiting. The child was taken to a military hospital shortly after the onset of the illness and was given some oral medications and injections. The fever subsided; however, child refused feeds and cried most of the time. About 18 hours prior to presentation, child was said to have been beaten by the father on account of defecating on the floor of their sitting room. Shortly after, the mother claimed to have heard what she described as "unnecessary excessive screaming". The cause of the excessive screaming was not known by the mother but she suspected that the father must have done something to the child out of anger. She (the mother) rushed towards the room to see the child on the floor in a pool of her own faeces, with the father in the same room, fiercely staring at the child. Shortly thereafter, the child developed generalized convulsions and was rushed to the military hospital from where the child was referred to the University of Ilorin Teaching Hospital (UITH). The child was the fourth and last child in a monogamous family of 6 members. The father was a military officer while the mother was a full time house wife.

Examination revealed a well fed, unconscious, pale, and febrile (38.4°C) child with no signs of meningeal irritation or abnormal posturing. There were no bruises. Anthropometry was essentially normal for age.

Central nervous system examination revealed a floppy child, deeply comatose (Blantyre Coma score of 2), with hypotonia and hyporeflexia.

Respiratory system revealed bradypnoea (Respiratory rate 12 cycles/minute), resonant percussion note, and coarse crepitations globally.

Cardiovascular system revealed barely palpable pulse, heart rate of 186 beats /minute, only first and second heart sounds were heard, and there was no murmur.

Abdomen was full, soft and moved with respiration. There was no area of tenderness, liver was 2cm palpable below the right costal margin, hepatic span was 8cm, soft and non-tender.

A diagnosis of child abuse with severe head injury was made. Packed cell volume was 18%, random blood glucose was 2.0mmol/L, lumbar puncture was

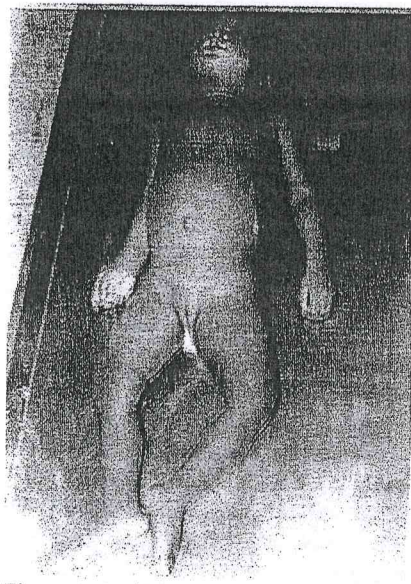


Figure 1: Well fed, severely pale child



Figure 2: Blood Clots in the sub-dural space



Figure 3: Massive subdural hematoma.

deferred as the child's cardiovascular status was considered not stable enough.

Child was given a bolus of 0.4mls/kg of 50% dextrose in triple dilution, and intravenous infusion was continued with 10% dextrose in 1/5th saline solution. The child's condition continued to deteriorate and she eventually died within 4 hours of presentation. Both parents were crying and the mother accused the father of inflicting fatal injury on the child. The father was remorseful and did not deny the mother's accusation.

Post mortem examination revealed a well fed child, severely pale, (Figure 1) with two subcutaneous haematomata measuring 1.5-2cm each in the frontal region of the head and 2.5cm in the left occipital region. The calvarium was opened to reveal a bulging dura with massive subdural hematoma. (Figure 2 and 3) About 700mls of fresh blood was evacuated. The brain weighed 1.1kg. The anatomical diagnosis was subdural haematoma. The management of the father's institution was officially communicated about the incident but there was no feedback from the institution and the case was not followed up.

Discussion

Child abuse is probably more common than what is often reported especially in Africa and other developing countries and it is often carried out by someone close to the child.¹ Given that only a small proportion of child abuse is reported, the prevalence has been difficult to quantify thus evaluation of prevalence and pattern of child abuse is a very vital issue and it is often under reported in paediatrics practice. In Nigeria and other developing countries, it may be difficult discussing the issue of child abuse without eliciting the innate perspective of such countries.² Certain religio-culturally accepted child rearing practices such as employing physical

reprimand in correcting the child may make it difficult to delineate child abuse from the culturally accepted child rearing practices.³

In Nigeria for instance, Mbakogu¹ examined forms of Child Abuse in Nigeria and opined that several United Nations Articles and Acts to protect children from such abuses are merely on paper. Some of such rights include^{2,4,5} the right to a protected and united family (Article 18), the right to parental care and protection (Article 19). Child abuse and neglect has been linked to various negative outcomes such as physical injuries, physical ill-health, emotional impairment, psychiatric disorders, suicidal behaviours and even death,⁶⁻⁹ just as seen in this case.

In this case, the child apparently had maternal care and protection with suspected paternal brutality culminating in her demise. The cultural approval of corporal punishment in child rearing among many African cultures¹⁰ and even religious circles could inadvertently encourage physical abuse through the teachings such as "whoever spares the rod spoils the child (Proverbs 13:24)

The child was probably ill due to treatable illness to which she was apparently responding to the treatment. There was no significant obvious physical injury on the child except for the scanty subcutaneous haematomata on the head suggesting some form of trauma.

Parents under the influence of emotional stress, poor impulse control, personality disorders, physical and or mental ill-health, use of psychotropic substances and poor quality of parent-child relationship could exhibit child abuse.¹¹⁻¹² If a child is a member of a home where there is domestic violence, such a child is prone to physical abuse.

The issue of torture is discussed by several instruments applicable in Nigeria.^{2,4} The Constitution

of the Federal Republic of Nigeria states in its Article 34 (1) that: "Every individual is entitled to respect for the dignity of his person, and accordingly (a) no person shall be subject to torture or to inhuman degrading treatment." Torture is also prohibited under the African Charter on Human and Peoples' Rights (Ratification and Enforcement) Act 59: Nigeria signed the Convention against Torture and Other Cruel, Inhuman, and Degrading Treatment or Punishment (CAT) on July 28, 1988 and ratified it on June 28, 2001. Despite this, several evidences of tortures are still part of daily occurrences in Nigeria. This case is also one corroborating such.

Very recently in Nigeria, in July 2016, two news broke of fathers who claimed to be pastors that chained their children for months in the name of casting out evil spirit, starving them while the event lasted. It is believed that if these cases are well prosecuted; they may serve as deterrents to some other parents or guardians with similar disposition to child abuse.¹³⁻¹⁴

In Nigeria, the constitution is not uniform as regards Child Acts, while the North operates Penal Code, the South operates Criminal Code, The Penal Code gives room to the use of torture on children. For instance, Article 55 (1) (a) of the Penal Code (North) stipulates that: "Nothing is an offence which does not amount to the infliction of grievous hurt upon any person and which is done: by a parent or guardian for the purpose of correcting his child or ward such child or ward being under eighteen years of age". Article 295 of the Criminal Code (South) also admits corporal punishment as a measure for the education of a child: "A blow or other force, not in any case extending to a wound or grievous harm, may be justified for the purpose of correction as follows: a father or mother may correct his or her legitimate or illegitimate child, being under sixteen years of age, or any guardian or person acting as a guardian, his ward, being under sixteen years of age, for misconduct or disobedience to any lawful command". The article goes on reading: "a master may correct his servant or apprentice, being under sixteen years of age, for misconduct or default in his duty as such servant or apprentice" and "a father or mother or guardian, or a person acting as a guardian, may delegate to any person whom he or she entrusts permanently or temporarily with the governance or custody of his or her child or ward all his or her own authority for correction, including the power to determine in what cases correction ought to be inflicted; and such a delegation shall be presumed, except in so far as it may be expressly withheld, in the case of a schoolmaster or a person acting as a schoolmaster, in respect of a child or ward;"

The legal definition of "grievous hurt/harm" for the purpose of the Criminal Code means "any harm which amounts to a main or dangerous harm, or which seriously or permanently injures health, or which is

likely to injure health, or which extend to permanent disfigurement or to any permanent or serious injury to an external or internal organ, member, or sense." As described in Article 295 of the Criminal Code.¹

The fact that the father was remorseful and did not make any response to the mother's accusation suggested that he might not have intended to kill the child but did, admittedly mistakenly. When this happens, the act above is silent on what the spouse should do in terms of legal pursuit of justice for such injured or killed child.

The volume of blood in the subdural space seen at post mortem examination was massive; nearly the whole of projected blood volume for the child (680-750mls) using the formula 80-85mls/kg.¹⁵ For 700mls of blood to have accumulated over a short period of time in the subdural space speaks volumes about the severity of the impact of the trauma to the head. Also, the concomitant imbalance in the cardiovascular status also contributed to the quick demise of the child. The fact that nothing apparent was done to the father and the issue was settled as a family mis-adventure is something that should continually challenge the role of doctors- especially the Paediatrician in the prevention of child abuse.

Conclusions

There is need to institute measures targeted at educating all those involved in the health and wellbeing of children about Child abuse. Efforts should be made to address factors associated with child abuse especially those that operate within the family.

All cases of suspected child abuse should involve the legal department of the hospital and the police from the onset. More awareness should be provided to the public on the types of child abuse and how to prevent its occurrence. The protective articles and acts in our Constitution must be made to work rather than being a paper documentation alone.

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