PACIFIC JOURNAL OF MEDICAL SCIENCES

VOLUME 11, No. 1, APRIL 2013

PACIFIC JOURNAL OF MEDICAL SCIENCES

(Formerly Medical Sciences Bulletin)

ISSN: 2072 – 1625 Volume 11, No. 1, April 2013

A multidisciplinary journal for publication of medical and biomedical research findings on issues pertinent to improving family health and related issues of public health

INFORMATION

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APRIL 2013: ISSN: 2072 – 1625 VOLUME 11, No. 1

TABLE OF CONTENTS

Page #

CASE REPORTS

LETTER TO THE EDITOR

ASSESSMENT OF AGE AT MENARCHE OF NIGERIAN URBAN SCHOOL GIRLS

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ABSTRACT:

Age at menarche varies with time of onset and the influence of social factors depends on the population under consideration. The objective of the present study was to determine the age at menarche among secondary school girls in Benin City, Nigeria and identify some of the social factors that might influence it. In this cross sectional study information on age at menarche was obtained from 1,640 menstruating secondary school girls (aged between 10 and 20 years) using the status quo method. Information sought in the structured questionnaire used included date of birth, date of menarche, educational attainment and occupation of parents, birth position, family size and State of origin. Data was analyzed using SPSS version 12.0. The mean age at menarche was 13.44±1.32 years (95% Confidence Interval, CI= 13.36-13.50). One out of every 15 (6.7%) girls below 12 years of age had attained menarche, indicating early menarche. There was statistically significant relationship between the mean menarcheal age and socioeconomic status ($p< 0.001$), birth position ($p< 0.01$) and family size ($p< 0.01$). The current mean age at menarche among secondary school girls in Benin City is 13.44±1.32 years and it is influenced by socioeconomic status, birth position and family size.

Key words: Age, menarche, urban, schoolgirls, Nigeria.

(Submitted October 2012; Accepted November 2012)

INTRODUCTION:

Menarche (the first menstrual bleeding of a female [1]) represents the endpoint of a complex sequence of events that characterize sexual maturation and puberty in girls [2]. It is unique, and probably, the most accurately recallable indicator of puberty among girls and a widely used indicator of adolescence sexual maturation [3,4]. Variations in age at menarche between individuals and populations have been documented [5-7]. It is influenced by social, environmental and genetic factors [5-9].

The mean age at menarche varies from one population to another; in Iran it was 12.91±1.23 years [10], in South Africa 12.75±1.32 years [8], in India 13.18±1.08 years [9], and in Kenyan 12.5±2.8 years [11]. Within Nigeria, similar variations in age at menarche have been observed; 13.98±1.30 years in the West [12], 13.03±1.02 years in the East [13], and 13.50±1.33 years in the North [14]. A study involving one secondary school in Benin City, Nigeria reported a mean age at menarche of 13.16±1.22 years [15]. In both developing and developed countries, some studies have reported a decline in the average age at menarche [16-19]. This trend towards a reduction in the average age of menarche has been attributed to improvement in living standard and nutrition [1]. On the other hand, in some countries, this downward trend seems to have come to a halt [20]. In view of the reported secular trend, there is a need to monitor the age at menarche. Data on age at menarche are useful in health planning, establishment of adolescent health centres and improvement in health promotion services for girls [21]. In addition, contemporary issues such as introduction of sex education in Nigerian schools require knowledge of the age at menarche as well as the sequence of events of puberty; menarche being the last in this sequence.

An early age at menarche is associated with an increased risk of some clinical conditions, such as breast cancer [22], obesity [23], endometrial cancer [24], and uterine leiomyomata [25]. Some studies have indicated that women who attained menarche at the age of 11 years and below have a higher risk of development of breast cancer than those who attained menarche at the age of 12 years and above [26,27]. Late menarcheal age is thought to protect, at least to some extent, women in Sub-Saharan Africa from breast cancer [28]. In this regard, the observed trend towards a reduction in the average age at menarche in West African countries (Nigeria inclusive) portends some danger as it relates to occurrence of breast cancer [13,14]. In addition, there are indications in the literature that the age at menarche might be related to subsequent reproductive performance, such as the age at first intercourse, the age at first pregnancy and risk of subsequent miscarriage [29]. From the foregoing, it is obvious that there is a need to

monitor closely the average age at menarche in Nigeria. Majority of the Nigerian studies that focused on age at menarche were conducted several years ago [12-14].

The objective of the present study was to determine the age at menarche among secondary school girls in Benin City, Nigeria and identify some of the social factors that might influence it.

PARTICIPANTS AND METHODS:

This cross-sectional study was conducted among adolescent girls in two public secondary schools in Oredo Local Government Area (OLGA), Edo State, Nigeria. According to the Edo State Ministry of Education Statistics, there are nine public secondary schools in the local government area (LGA), comprising three co-educational, two boys and four girls' schools [30]. Consent for the study was obtained from the school authorities and from the parents. Two of the four girls' secondary schools were randomly selected. The student population of each of the two schools selected was 1,394 and 772, giving a grand total of 2,166, which represents the study population.

During data collection the girls were informed about the relevance of the study and the need to accurately fill the questionnaire without including their names, and that their participation was voluntary. Data was collected between October and November 2011, using a structured questionnaire designed by the authors. The questionnaire was pre-tested on 30 girls of the same age group in another all girls' school within the same LGA. Information sought in the questionnaire included: date of birth, date of onset of first menstrual bleeding, birth position among their siblings, family size (number of siblings), State of origin, level of education and occupation of both parents/guardian.

The family size was categorized into small size (no sibling or one or 2 siblings); medium size (3 or 4 siblings); large size (5 or more siblings). The socio-economic status of the parents was determined using the classification suggested by Ogunlesi et al [31]. This was analyzed via combining the highest educational attainment, occupation and income of the parents (based on the current mean income of each educational qualification and occupation in Edo State, Nigeria). In this Social Classification System, Classes I and II represent the high social class, Class III represents the middle social class, and Classes IV and V represent the low social class. In this way, the girls were categorized into high, middle and low socio-economic classes.

The data was analyzed using the SPSS (Statistical Package for Social Sciences), version 12.0.

RESULTS:

Seven girls declined to participate, thus the response rate was 99.7%. The questionnaires of 9 girls were excluded from the analysis because they were incompletely filled, thereby leaving a total of 2,150 (99.6%) questionnaires for data analysis. Girls in both schools had similar socio-demographic characteristics, thus analysis of data was carried out for the combined group of girls.

Of the 2,150 girls, 1,640 (76.3%) had attained menarche at the time of the study. Analysis of the questionnaire of the 1,640 girls showed that the mean age at menarche was 13.44±1.32 years (95% Confidence Interval, CI= 13.38-13.50). One out of the 15 (6.7%) girls who were below 12 years of age had attained menarche, indicating early menarche (Table 1).

The mean age at menarche was significantly higher in the low socioeconomic class compared to the high socioeconomic class (Table 2). Girls from the high socioeconomic class attained menarche 8.0 and 9.0 months earlier than the girls from the middle and the low socioeconomic classes respectively.

Table 1: Distribution of the girls according to their age at menarche

Age in Years	Total number of girls	Number (%) that attained menarche
10	2	0 (0)
11	13	1 (7.7)
12	109	16 (14.7)
13	296	95 (32.1)
14	356	218 (61.2)
15	380	337 (88.7)
16	490	471 (96.1)
17	315	313 (99.4)
18	112	112 (100.0)
19	62	62 (100.0)
20	15	15 (100.0)
Total	2150	1640 (76.3)

Table 2: Socioeconomic status and mean age (years) of the girls at menarche

Age at menarche (years)	Total number that attained menarche	Socioeconomic status (SES)		
		High N (%)	Middle N (%)	Low N (%)
10	0	0 (0.0)	0 (0.0)	0 (0.0)
11	1	1 (9.1)	0 (0.0)	0 (0.0)
12	16	10 (62.5)	6 (37.5)	0 (0.0)
13	95	47 (49.5)	38 (40.0)	10 (10.5)
14	218	106 (48.6)	95 (43.6)	17 (7.8)
15	337	31 (9.2)	176 (52.2)	130 (61.4)
16	471	8 (1.7)	143 (30.4)	320 (67.9)
17	313	2 (6.5)	16 (5.1)	295 (88.4)
18	112	0 (0.0)	13 (11.6)	99 (88.4)
19	62	0 (0.0)	57 (91.9)	5 (8.1)
20	15	0 (0.0)	12 (80.0)	3 (20.0)
Total	1640	205 (12.5)	548 (33.4)	887 (54.1)
Mean age (95%CI)	13.44±1.32 (13.38-13.50)	12.78±1.21[a] (12.61-12.95)	13.42±1.18[b] (13.32-13.52)	13.56±1.29[c] (13.42-13.59)
t-statistic (p-value)		a vs b =4.41 (<0.01)	b vs c=2.08 (>0.05)	a vs c=7.95 (<0.001)

As shown in Table 3, first-born girls attained menarche earlier than latter-born girls with first-born girls attaining menarche 8.0 months earlier than eight-born girls. Of the 1,640 girls, 8 (0.5%) were the only child in their family and the mean age at menarche was 12.80±1.11 years (95% CI= 12.03-13.57). Table 4 shows the mean age at menarche according to family size. Girls from small-size families attained menarche 4.0 and 7.0 months earlier than their counterparts from medium-size and large-size families respectively. Based on state of origin, the mean age at menarche did not differ.

Table 3: Birth position and mean age (years) of the girls at menarche

Birth position	Total number that attained menarche	Mean age (yrs) at menarche	Age 95% CI
1st	437	13.02±1.22	12.91-13.13
2nd	333	13.04±1.09	12.92-13.16
3rd	323	13.14±1.12	13.02-13.26
4th	164	13.30±1.23	13.11-13.49
5th	77	13.52±1.19	13.25-13.79
6th	33	13.53±1.38	13.06-14.00
7th	38	13.65±1.40	13.21-14.10
8th	27	13.68±1.43	13.14-14.23
Total	1640	13.44±1.32	13.38-13.50

Table 4: Family size and mean age (years) of girls at menarche

Family size	Number (%) that attained menarche	Mean age (yrs) at menarche	Age 95% CI	t-statistic (p-value)
Small size	221(13.5)	13.09±1.31[a]	12.91-13.26	a vs c: t=4.82 (<0.01)
Medium size	1063 (64.8)	13.41±1.28[b]	13.33-13.49	a vs b: t=3.32 (<0.05)
Large size	356 (21.7)	13.64±1.37[c]	13.50-13.78	b vs c: t=2.79 (<0.05)
Total	1640 (100.0)	13.44±1.32	13.38-13.50	

DISCUSSION:

The mean age (13.44 years) at menarche obtained in the present study in Edo State was comparable to 13.43 years reported from Port Harcourt among urban school girls [32] but lower than 14.22 years reported among rural school girls in Etche (both in Rivers State, Nigeria) [33]. The lower mean menarcheal age in the present study might be explained by differences in socio-demographic factors. In the present study the girls were urban school girls while in the study in Etche they were rural school girls. The report of a study in Plateau State, Nigeria indicated that rural school girls

tend to achieve menarche at an older age than urban school girls [14]. In contrast, Goon et al [34], reported that the age at menarche was comparable between urban and rural girls. They attributed this parity to improved living conditions among their rural population. It is important to note that the methods of collecting and analyzing data vary from one study to another, indicating the need to exercise caution when comparing age at menarche in different studies. Our current result indicated that one out of every 15 (6.7%) girls below 12 years of age attained menarche. This is similar to the findings reported from Wannune, Benue State, Nigeria but with higher prevalence rates of 16.4% and 37.0% at 10 and 11 years of age respectively [34]. A previous study in Benin City alluded to occurrence of early menarche in their study but the prevalence was not indicated [15]. The clinical implication is that this small group of girls with early menarche might be at increased risk of breast cancer, obesity, endometrial cancer and uterine leiomyomata [22-25], thus the need to advocate for them to be closely monitored. In addition, Schor reported that the age at menarche might be related to the age at first sexual intercourse [29], which may result in unwanted teenage pregnancy with its attendant risks. The suggested practical solution is that sex education in Nigerian schools should be started early, well before the age of 12 years.

As in previous studies in Nigeria [12-15], socioeconomic status (SES) of the parents influenced their daughters' age at menarche. According to our results, girls belonging to the high socioeconomic class attained menarche 9 months earlier than their counterparts in the low socioeconomic class. This finding is comparable to the 8.5 months difference reported in a previous study in Nigeria [13]. On the other hand, the observed difference in the present study is lower than the 11 and 12 months reported in two previous studies in Nigeria [15,35].

Our result indicates that the first and early born girls tend to attain menarche at an earlier age than their counterparts who were born latter or were last born. A similar observation has been reported in a previous study [15]. There is no readily available explanation for this observation. However, it has been speculated that there is usually more pressure and expectation on the first-born girls to achieve, forcing them to mature faster that latter-born girls. It is, therefore, thought that this early attainment of maturity in first-born girls, make them attain menarche at an earlier age compared to their latter- born siblings.

Our result supports the reports by others [36], that girls from small-size families tend to attain menarche at a younger age than girls from large-size families. Family size may exert its effect on age at menarche through concealed poverty because the larger the family size the lower the income per capita. This effect is likely

to be more pronounced in societies with low socioeconomic status.

In conclusion, the current mean age at menarche in Benin City was 13.44 years; it is influenced by social factors such as socioeconomic status, birth position and family size.

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PREVALENCE OF HUMAN EDIBLE CRABS INFECTED WITH *PARAGONIMUS UTEROBILATERALIS* METACERCARIAE IN SOUTHEASTERN NIGERIA

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Running title: Human edible crab metacercariae infection

ABSTRACT:

This study was aimed at determining the seasonalityof relative abundance of human edible crab, *Sudanautes*caught from their natural habitats, and those sold for human consumption in local markets in endemic areas of South-eastern Nigeria, to assess their infection rate with *P. uterobilateralis* metacercariae. Crabs were caught from their natural habitat as well as bought from the market over a period of 12 months; they were then and examined in the laboratory for infection with metacercariae. The prevalence of metacercarial infection of the crabs in both groups showed no seasonal variation but oscillated throughout the year. In all, 151 (6.9%) of the crabs caught were infected with *Paragonimus* metacercariae. The monthly percentage of total infected crabs exhibited seasonality as the relative abundance, being relatively higher in the dry season months; peaking in the month of September but lowest in January. The monthly percentage of total infected crabs was higher than the percentage of crabs caught in five months: June, September, November, February, and May. There is need for innovative measures to discourage the local population from eating improperly cooked crabs so as to curb the epidemiological dangers of eating infected crabs.

Key words: Paragonimiasis, edible crab, *Sudanautes*, metacercariae infection, Nigeria

(Submitted January 2013; Accepted March 2013)

INTRODUCTION:

Crabs belong to a group of animals known as decapod crustaceans. Many species of crabs constitute an important part of the local food chain in sub-Saharan Africa [1]. Some of these crab species are of immense epidemiological importance.

The species *Sudanautes* is the intermediate host of *Paragonimusuterobilateralis* in eastern Nigeria [2]. Eating of large amount of crabs has continued in parts of Eastern Nigeria decades after second outbreak of paragonimiasis in the area [3]. Paragonimiasis is acquired as a result of consumption of raw or improperly cooked or prickled freshwater crabs or crayfish harbouring infective metacercariae [4].

Paragonimiasis is a zoonotic infection of tropical and sub-tropical significance clustered around the intertropical zone. About 200 million persons are at risk of being infected, while 20 million are already infected, aggravating the public health and socio-economic indices in the endemic areas [5].

Past research activities have been more on the prevalence of infection and identification of endemic foci [2, 3, 6, 7], as well as on the identification of the intermediate host [2]. The crab-eating behaviour of an endemic population has been studied and observations indicated that the risk of paragonimiasis increased with the frequency of eating of *Sudanautes* [3], and the diagnostic overlap of paragonimiasis with tuberculosis [8]. However, information on the seasonal abundance of *Sudanautes* and its seasonal rate of infection with *P. uterobilateralis* metacercariae is scanty. This is an important missing link in paragonimiasis research since seasonal variation in crab populations and the level of crab infection are important epidemiological factors in paragonimiasis transmission [9]. Thus longitudinal studies to obtain data on these parameters are steps in the right direction. This study aimed to determine the seasonal relative abundance of *Sudanautes* in their natural habitats, and to assess the infection rates of *Sudanautes* with *P. uterobilateralis* metacercariae in relation to seasonality among those sold in local markets in an endemic area of southeastern Nigeria.

MATERIALS AND METHODS:

The study area

The study was carried out in Oyigbo, a sprawling community in the suburb of Port Harcourt, Rivers State, Nigeria. Oyigbo is urban in nature;it constituted predominantly of non-indigenes, and could be described as assimilated into the larger Port Harcourt city. The community lies on the bank of Imo River, and therefore a considerable proportion of the population live off the freshwater resources from the river [3]. Generally, the population consists of subsistent farmers, fishermen, artisans, traders, river-food processors, and

significantly few white collar workers [3]. Imo River traverses three states mainly in rainforest Nigeria. The states are Imo, Abia, and Rivers states. The Oyigbo area is in the Lower Imo Basin. Here, crabs are caught both for subsistence and for commercial purposes and sold locally and in nearby towns where they are regarded as delicacy either to complement for meat and fish as sources of protein or to serve as alternative altogether. Crabs are now being used as partial substitute for fishmeal fed to fish being raised in fishponds [10].

Collection of crabs

In order to compare results, the basket traps and nets methods of Udonsi [2] were adopted with little modification. Nets were spread to cover Crab holes as much as possible and then the crabs were chased from their hiding places upstream by disturbing the surroundings. Baskets were placed where the crabs were most appropriate for the same purpose. Emerging crabs were trapped in the baskets or nets. Sampling was carried out comprehensively at various sampling points for two consecutive days every four weeks. The crabs were kept in sealed containers in iced packs and transported to the laboratory.

Sampling of crabs sold in the market

Sudanautes being sold in the market in the study areas were bought fresh during sampling days and preserved in iced packs and transported to the laboratory for examination.

Laboratory examination of crabs

The haemocoel contents of the individual crabs were examined in the laboratory using dissection microscope. The haemocoel was examined completely for encysted microcercouscercariae of *P. uterobilateralis* [2]. The number of cercariae per individual crab was noted. High magnification (x100) in Compound microscope was used for confirmation.

Data analysis:

Monthly percentage of total infected crabs (MPTI) was calculated as follows:

MPTI = (Number of infected crabs in a month x 100), divided by, total number of infected crabs collected in the entire 12 months.

Prevalence was calculated as follows:

Prevalence = (total number of infected crabs x 100), divided by, total number of crabs collected in the entire 12 months.

RESULTS:

Monthly Abundance of crabs and seasonal variation

A total of 2190 crabs were caught during the study. The abundance of crabs was highest in the months of June through September. Overall, the relative abundance of crabs peaked in August and was least in the month of January. It was significantly higher in the rainy season months (May to October) than in the dry season months (November to April; χ^2-test:

$p < 0.05$). The monthly abundance of crabs was more in the months of sustained heavier rainfall (see Table 1). The monthly variation in temperature was minimal throughout the year; hence the monthly temperature values recorded were comparable in both the rainy and dry seasons.

Table 1: Monthly abundance of crabs in relation to monthly rainfall and monthly temperature in southeastern Nigeria

Month	Abundance	Rainfall (mm)	Temperature (°C)
June	238	440	26.5
July	289	380	25.6
August	343	530	26.0
September	277	540	25.8
October	168	400	24.4
November	146	47	24.1
December	124	9	24.4
January	59	8	25.9
February	81	30	28.5
March	124	100	27.0
April	168	204	28.8
May	173	310	26.5

Infection rates between crabs caught from their natural habitats and those being sold in the market.

The prevalence of cercarial infection of crabs showed no seasonal variation but oscillated throughout the year in both crabs caught from their natural habitats and those being sold in the market. For the former category of crabs, the highest monthly prevalence was 7.5% in February; while the lowest was 3.8% in the month of July (see Table 2). For those sold in the market, the highest monthly prevalence of cercarial infection was 7.2% in the month of May and least (4.1%) in December and July.

Table 2: Comparison of monthly prevalence of crab infected with *Paragonimus* metacercariae between crabs caught in their natural habitats and those sold in the market in Southeastern Nigeria

Month	Percentage of total caught from the natural habitat (%)	Percentage of total sold in the market
June	5.7	5.2
July	3.8	4.1
August	4.9	5.2
September	6.6	5.8
October	5.9	5.8
November	5.2	5.8
December	3.9	4.1
January	5.9	4.2
February	7.5	5.8
March	5.0	5.5
April	5.3	6.2
May	6.3	7.2

Seasonality of percentage of crabs caught and percentage of infected crabs

In all, 151 (6.9%) of the crabs caught were infected with *Paragonimus* metacercariae. The monthly percentage of total infected crabs exhibited seasonality as did the relative abundance. As shown in Table 3, the monthly percentage of total infected crabs was relatively higher in the wet season months (June to September); peaked in September, but lowest in dry season month of January.

The proportion of infected crabs obtained in some months (in relation to total infected crabs obtained in 12 months) was higher than the proportion of crabs caught in those months (in relation to total infected crabs caught in 12 months). The months were June, September, October, January, February, and May. This difference was more marked in September.

Table 3: Seasonality of monthly percentages of total crabs and monthly percentages of total infected crabs in Southeastern Nigeria

Month	Percentage of crabs caught	Percentage of infected crabs
June	11.2	11.6
July	13.4	12.4
August	15.8	14.0
September	13.2	15.8
October	7.8	8.4
November	6.2	5.8
December	5.8	4.1
January	2.3	2.5
February	3.1	4.1
March	5.5	5.0
April	7.8	7.4
May	8.0	9.1

DISCUSSION:

The relative abundance of *S. africanus* in this study indicates that it is common in south-eastern Nigeria. Crabs generally are common and abundant in marine and freshwater bodies in West Africa [1]. The crabs are commonly harvested with crab pots, gill/set nets, and traps by local fishermen from streams, ponds, reservoirs and rivers for subsistence and commercial purposes [11]. In parts of southeastern Nigeria, there is frequent eating of *Sudanonautesafricanus*, the West African freshwater crab belonging to family Potamonautidae, with over 88 species, and is present in all the streams and river systems across Africa [12].

Data obtained in this present study confirms the seasonal variation in crab relative abundance in southeastern Nigeria as already reported by others [9, 13-16]. The seasonal effects on crab abundance are caused by variations in climatic factors such as rainfall and temperature [9]. Crab reproduction period is seasonal and is reported to coincide with the onset of rainfall [11]. However, there were no significant variations in the average monthly temperature observed throughout the yearin this study. This is expected as the study area is

close to the equator known to have minimal temperature variations. The minimal temperature variations may not have impacted significantly on the observed monthly relative abundance in this study. Elsewhere, crabs were more abundant and caught during the summer season, while lower abundance was recorded during the winter season for the same species [9, 17]. Epidemiologically six months (June, September, October, January, February, and May) were particularly striking in that the monthly percentage of total infected crabs in these months was higher than the percentage of crabs caught. This could suggest possible higher transmission indices in these months. This means that the transmission of paragonimiasis in South-eastern Nigeria is not seasonal.

The epidemiological parameters recorded from crabs caught in their natural habitats showed high infection rate. Massive eating of crabs in the area is driven mostly by the level of education and therefore could be attributed to level of awareness of the epidemiological implications of such eating behavior. Better awareness leads to better and proper preparation of the crab before eating [3]. On the other hand, better level of education most times could translate to better economic wellbeing, which means being in a better position to afford meat rather than relying on cheaper alternatives such as crab-eating [3].

Behavioural change must be a prominent part of any successful control strategies in South-eastern Nigeria. However, provision of alternative sustainable and inexpensive solutions to cause behavioral change has also been a restraint for many control programs [18]. Human behaviour plays a fundamental role in the epidemiology of parasitic infections such as paragonimiasis, both its emergence and spread.Human behavior is further complicated by the impacts of cultural, religious, ethnic, age and gender related variables. To have a desirable outcome in paragonimiasis control in the area, behavioural change must be directed to either curb crab-eating out-rightly or to properly cook them before eating. Secondly, there must be an alternative plan to encourage massive breeding and provision of infection-free crabs on a commercial scale for committed eaters. The crab species *S. africanus*is reported to have high fecundity, producing many eggs per individual, which is an indication that they are viable for farm production, and can be bred in captivity [11]. Selective breeding for rapid growth can be engineered to obtain more attractive and delicious specimens that will compete favourably with their naturally occurring counterparts.

CONCLUSION:

There is a high metacercariae infection rate among crabs in South-eastern Nigeria. There is need for innovative measures to discourage the local population from eating improperly cooked crabs.

ACKNOWLEDGEMENT:

The author appreciates the Chiefs of Oyigbo community and all the individuals that participated in the study. The contributions of my field and laboratory assistants are hereby acknowledged.

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NODDING SYNDROME IN UGANDA - A DISEASE CLUSTER: AN EPIDEMIOLOGICAL DILEMMA

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ABSTRACT:

Nodding Syndrome (NS) was recently described in children in Northern Uganda. The affected children were in the age group 5 – 15 years. They were stunted, malnourished, dehydrated, mentally retarded and get recurrent seizures. The objective of this study was to describe the cluster distribution of NS cases in Northern Uganda. We conducted a cross-sectional study using available data on the burden of NS in Northern Uganda and used GPS to map the locations of the most affected areas. The results obtained indicate that Nodding Syndrome in Northern Uganda occurs in clusters in the following locations Odek, Atiak, Angagura, Awere, Laguti, Labongo-Amida, Atanga, Pajimo, Palabek Kal and Palabek Gem sub counties and mainly along Aswa and Pager rivers and their tributaries. Nodding Syndrome in Northern Uganda occurs in clusters predominantly along two rivers; perhaps it is an indication for environmental, dietary and common epidemiological exposures for the syndrome.

Keywords: Nodding Syndrome, cluster, Northern Uganda
(Submitted February 2013; Accepted March 2013)

INTRODUCTION:

Nodding Syndrome (NS) is an unexplained neurologic illness that has been reported among persons in some sub-Saharan African countries in recent years [1, 2, 3]. In two of the three regions where it has been reported recently, the syndrome has occurred among internally displaced persons or those formerly displaced and later returned to their villages [1-5]. Nodding Syndrome is a clinical constellation of symptoms that most times begin with head nodding and later may result in progressive neurological deterioration [1-5]. Onset is common in children between the ages of 5 and 15 years. The most characteristic feature is a paroxysmal "spell" in which the head bobs forward repeatedly over a period of minutes; in most cases, the child appears unresponsive during the episode [1-5]. The disease has been investigated previously in Southern Sudan in 2001-2003 [3] and Tanzania in 2008 [1].

Ministry of Health in Uganda began investigating a cluster of cases of NS in early 2009 and made several trips to affected district of Kitgum in northern Uganda over the course of the year in an attempt to understand and control the disease [6]. It was estimated by the district health officer that the outbreak in Kitgum district may have affected up to 2000 children [6]. Subsequent investigations resulted in the generation of an extensive list of possible causes for the syndrome including viral, bacterial, parasitic infectious diseases, nutritional deficiencies, and genetic disorders, exposures to heavy metals or pesticides, slow virus or prion diseases, exposures to munitions from the war, post-traumatic stress, mass hysteria, pseudo-seizures and a number of others [6]. Because of the complex nature of the disease and the range of possible aetiologies, the Uganda Ministry of Health requested assistance from the U.S. Centers for Disease Control and Prevention (CDC) and World Health Organization (WHO) to conduct a more detailed investigation [6].

The investigative approach was multi-faceted and included a formal review of existing knowledge about the disease, the establishment of a multidisciplinary investigative team, and a phase of hypotheses refinement using qualitative focus groups and key informant interviews [6]. The investigation suggested that NS is a distinctive clinical entity affecting many children in Kitgum, Pader, and Lamwo districts [6]. In addition, NS in these districts share most of the epidemiological and clinical features of previously described "Head Nodding Syndrome" in Southern Sudan and in Tanzania. It was concluded that it was likely that the illnesses occurring in these three geographic areas (Northern Uganda, Southern Sudan and Tanzania) represented the same clinical entity [6].

Clustering of Nodding Syndrome in Northern Uganda:

There is a clear observation that NS occurs as clusters and mainly along the two major rivers of Pager and Aswa in Northern Uganda; perhaps an indication of environmental contaminations. This cluster occurrence presents both valuable opportunities and methodological problems for the study of epidemiology of this syndrome [7]. Researchers have argued that epidemiological studies could uncover leads to the cause of a particular disease especially those occurring in clusters; it could help evaluate the cause/risk factors of the disease and the possible effect of environmental contaminant which could be important for the assessment of human responses to multiple exposures such as chemicals [7]. This knowledge could in turn help to identify causes/risk factors of NS and could guide policy makers in decision making in the case of environmental contamination as the risk factor [7].

The aim of this epidemiological study was therefore to understand and describe the pattern of occurrence of NS and by inference develop approaches to aid prevention.

The objectives of this study were to use Global Positioning System (GPS) to mark, correlate and map high burden areas of NS in northern Uganda and to compare the data obtained with the CDC findings of 2009; in addition to describe the sites with the heaviest burden of Nodding Syndrome in relations to river network in the region.

MATERIALS AND METHODS:

The study was conducted in Pader, Kitgum, Lamwo, Amuru and Gulu districts in August-September 2012. It was estimated through surveillance records from the offices of the district health offices that over 3000 children in these 5 districts had NS and that over 200 children had died of diseases related to it. Part of these areas under study was part of the areas covered by the CDC study in 2009 [6]. This region is just recovering from over 20 years of civil war. Gulu is one of the regional centers for northern Uganda and draws a largely rural population; many of whom lived in the internally displaced people (IDP) camps for the past 10 to 12 years for safety from the insurgency.

This was a cross sectional study design; GPS was used to map out the sub counties with the highest density of NS. The study sites were selected based on the number of NS already confirmed and available at the District Health offices. These cases were screened and confirmed as probable cases using the WHO epidemiological surveillance case definition for Nodding Syndrome [19].

Data collection: GPS locations for sites with high density of probable cases of NS were

marked and recorded with the Trimble GeoXT GPS unit. Surveillance data on probable NS included the names of the patients, their villages; parishes and sub counties which were recorded and used to locate the homes of the probable cases. The location was identified and marked on the GPS machine and coordinates stored in a developed shape files and later aggregated and plotted on a map to show the number of cases per Sub County.

Data Analysis: GPS recordings were transferred to a database and plotted on map of the region for easy interpretation and evaluation. The maps including the river network were integrated with the aggregated data of the probable cases of NS per Sub County. The machine was also used to calculate the distances of the cases from the rivers in the region.

RESULTS:

The pattern of distribution of probable cases of NS have remained consistent over the last three years as observed by the studies conducted in CDC in 2009 and another repeated in 2012 by the same authors [6]. As can be observed the first two figures, river Pager appears prominently in this GPS map distribution for both studies of 2009 and 2012.

Figure 1 show the CDC findings of 2009 where samples were collected from probable cases of NS [6]. Probable cases were marked by green dots and suspected cases marked with yellow dots. The areas marked by large circle (red and blue lines) represented those areas in Kitgum and Lamwo districts with the highest burden of NS at the time and most of them were observed to occur along the Pager River.

Figure 2 shows the locations of the current distribution of probable cases of NS in the sub region. The round white in pink circles shows the current distribution of probable cases. The current marking on the map was superimposed on the map used by CDC in 2009 to show and compare whether there were any significant differences in the distribution of NS in the region. These sites were found in Kitgum, Pader, Lamwo, Amuru and Gulu districts.

The areas marked by large circle (blue and red) covered those areas with the highest burden of NS in Kitgum, Pader and Lamwo districts and were found mainly along the Pager River and its tributaries (Lanyadyang and Awuc). The areas marked by small-yellow circles indicated sites that were described to have suspected cases in 2009 but were confirmed as probable cases in 2012 by the Ministry of Health.

Figure 1: Map of Kitgum, Lamwo and Pader districts where NS were previously sampled in 2009 by CDC [6]

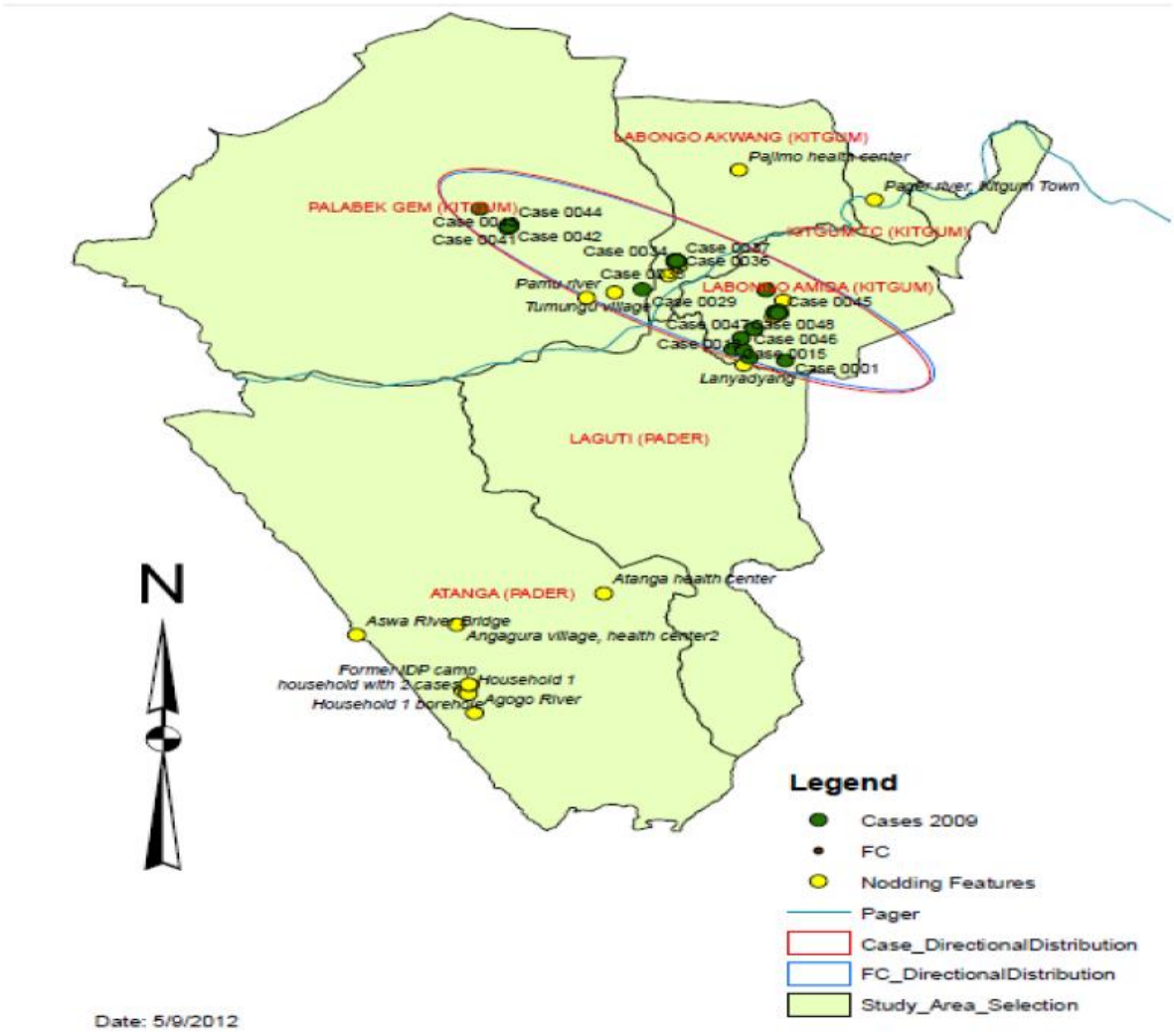

Figure 2: Map of Kitgum, Lamwo and Pader districts were NS were currently confirmed in 2012 [6]

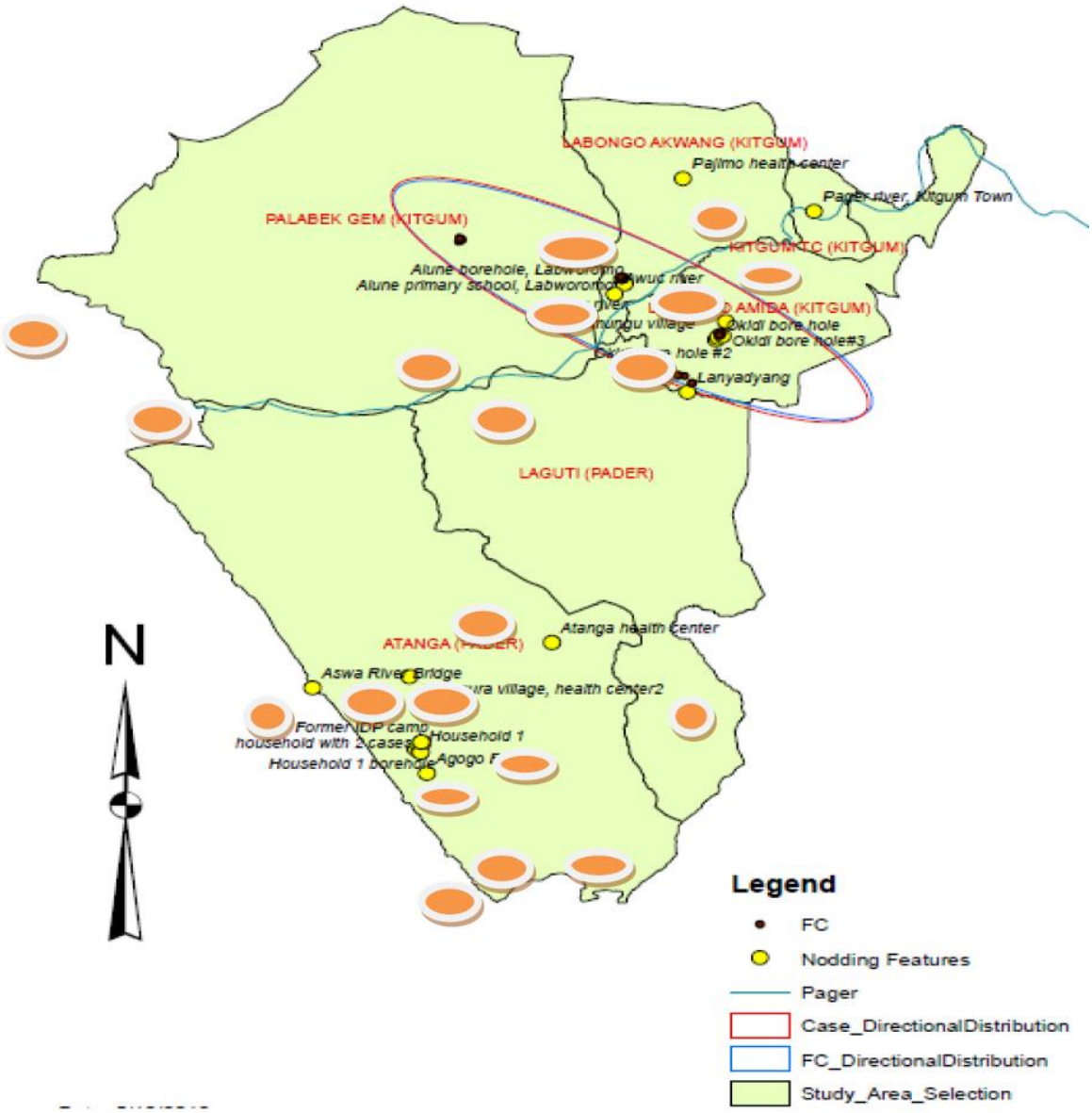

Table 1: Shows the occurrence of Nodding Syndrome in sub counties along specific rivers in Northern Uganda

Gulu District	Sub County	Number of NS cases	River
	Odek	100	Aswa
	Palaro	10	Aswa
	Cwero	15	Aswa
	Paicho	15	Aswa
Amuru District			
	Atiak	80	Unyama/Aswa
Pader District			
	Awere	80	Aswa
	Atanga	314	Ajan
	Laguti	215	Ajan
	Angagura	210	Aswa/Agago
	Acholi bur	30	Lanyadyang
	Puranga	15	Aswa
	Kilak	30	Agago
	Pajule Lapul	64	Agago
	Patongo/Pader	2	Agago
Kitgum District			
	Labongo-Amida	80	Pager/ Lanyadyang
	Labongo-Layamo	40	Pager
	Labongo-Akwang	50	Pager
Lamwo District			
	Palabek Kal	60	Pager/Aswa
	Palabek Gem	120	Pager/ Aswa

(Source of data is from the DHO's office of respective districts)

Table 1 shows the distribution of NS by Sub County in the 5 districts of the Acholi sub-region: Odek, Palaro, Cwero, Paicho sub counties in Gulu district; Atiak in Amuru district; Awere, Atanga, Laguti, Angagura, Acholibur, Pajule-Lapul, Puranga, Kilak, Pader/Patongo in Pader district; Labongo-Amida Labongo-Akwang and Labongo-Layamo in Kitgum district; Palabek Gem and Palabek Kal in Lamwo district. The four sub counties in Gulu districts were situated along Aswa River; Atiak in Amuru district is along Unyama River which is a tributary of Aswa River. Similarly, the 2 sub counties in Lamwo are situated and bordering

Pager and Aswa rivers and the sub counties in Kitgum district are traversed by Pager River. The sub counties in Pader have several tributaries of the 2 main rivers (Pager and Aswa) that traversed it. These tributaries included; Lanyadyang in Acholibur; Ajan in Laguti and Atanga; Agago River in Angagura, Pajule-Lapul and Kilak sub counties.

The Rivers shown in the map above includes Pager which traverses through Kitgum town and later joins Aswa; Agago River which joins Aswa in Pader district. These main rivers (Pager, Agago and Aswa) all originate from Karamoja region (the mountains in Moroto and Kotido districts) [20].

DISCUSSION:

The distribution of Nodding Syndrome in northern Uganda represents a disease clustering in specific geographical locations. The cases mainly occur along the tributaries of the 2 major rivers - Aswa and Pager whose main water supplies originate from the hills and mountains in Karamoja region in north eastern Uganda. These NS clusters are on either side of these 2 rivers and within specific locations of: Odek, Paicho, Palaro and Cwero in Gulu district; Atiak in Amuru district (Western bank of Aswa River); Awere, Angagura, Kilak, Laguti, Acholibur, Pajule lapul, Puranga and Bolo in Pader district (Eastern bank of Aswa river); Palabek kal, Palabek Gem in Lamwo district (Eastern bank of Aswa River).

Nodding Syndrome clusters in Laguti and Atanga are along Ajan River which is a tributary of Aswa River; Okidi in Labongo-Amida; Pajimo and Tumangu –in Labongo-Layamo Sub Counties are drained by rivers such Lanyedyang and Awuc which are tributaries of Pager River [20].

Knox described a cluster as a series of cases that are delimited both temporarily and geographically; this may be applied to an illness that may appear in clustered form or that is of such size and concentration that is highly unlikely to be a product of chance [7]. Indeed the occurrence of NS in these specific locations and number of cases found is certainly not by chance.

Another researcher in occupational lung diseases stated that in a disease cluster, a case should be related through biological and/or social mechanisms or may have a relationship with a specific events or circumstances [8].

Figure 3: Map of Uganda showing NS clusters in relations to the river network in the Acholi sub region [20]

Nodding syndrome clusters along the major rivers in Northern Uganda

The findings observed among the probable cases of NS in the Acholi sub-region is that they occur among specific age groups (5-15 years) and in specific locations and mainly along the 2 major rivers (Pager and Aswa) (Maps in Figures 1, 2 & 3). Previous studies have indicated and confirmed that more cases were found in families where there was already a case of NS. This therefore perhaps represented the biological and social relationships of Nodding Syndrome in the region [6, 10,12].

Knox called this tendency towards grouping of many diseases in time and space, “clustering”. Thus clustering is the regular tendency of a single/ or many diseases to present themselves

irregularly in time and space once the population density and chances are accounted for [7]. Nodding Syndrome pattern of distribution has regional trends because it occurs in relation to geographic coordinates (Maps in Figs 1, 2, 3). However, since most cases of NS lived in the IDP camps, there is perhaps an indication that there is a relationship between NS and time; particularly IDP camp exposures [1, 4, 6, 9, 10, 11, 12].

It is our view that because of the clustering of NS, the identification of the pattern of clustering can facilitate the establishment of the original aetiological hypotheses [7, 8].

The present epidemiological study relied heavily on statistics for establishing and quantifying the relationships between the locations and occurrence of NS, and for attempting to establish whether or not there was an excessive number of NS occurring in a specific geographic area [7, 8].

In clustered disease, both retrospective and prospective studies are useful in discovering links between environmental exposures and a particular disease [7, 8]. These epidemiological studies are especially significant when they uncover very high incidence of an unusual disease in a target population [7, 8].

The standard guidelines and protocol for epidemiological studies are well documented [7] and in addition, the epidemiological evidence can only show that a risk factor may be associated with higher incidence of the disease in the population exposed to that risk factor. Thus, the higher the correlation, the more certain the association between the risk factor and the disease [7, 8, 9]. Furthermore, the study should determine what behavioural, environmental and health factors being studied are the possible risk or protective factors [7, 8, 9, 10, 11].

In an event that an inappropriate risk factor is chosen for an epidemiological study, an association may be found between an inappropriate factor and the disease because this factor is associated with another factor which is actually related to the disease, but which was not studied. In such an instance, the inappropriate factor becomes a confounding variable, because it distorts the interpretation of the results of the study [7, 8]. The occurrence of the Onchocerciasis in the region among NS may perhaps be one of the confounders to the risk factors to NS. This is because, many regions in Africa and particularly Uganda have had very high prevalence of Onchocerciasis but there has not been such clinical presentation of NS [9, 10, 13]. More so, there has always been Onchocerciasis in the Acholi sub-region right from the colonial times (1900) and the community have a local name for Onchocerciasis “two ajonga miya” [10, 12]. It is therefore more likely, that NS may have resulted from a new factor which has plunged the sub-region as a result of perhaps environmental activities that has not yet been identified. Reports from Mulago Hospital, the National Referral and Makerere University

Teaching Hospital where 24 children with NS were transferred from Kitgum for specialized investigations and treatment showed that only 3 children had the antibodies for Ochocerca volvulus [14]. This finding would make Onchocerciasis a less likely cause/risk factor of NS. It is the researchers' view that the association between Onchocerciasis and NS therefore, seems a non-causal, but related phenomenon in the development of NS.

It is probable that the open mining system alleged to be taking place in the Karamoja region which is the water shed for the rivers in the Acholi sub-region may perhaps be the source of contamination of waters and environments of the population downstream. These contaminations got exposed to the children who already had a deranged metabolic condition and were unable to neutralise the effects of the contaminants [10, 12, 15, 16]. A case control study conducted by researchers at Gulu University showed that Children with NS were in a state of metabolic acidosis compared to their matched controls [10]. Several studies have already shown that metabolic acidosis undermines the ability of the human body from binding heavy metals. Acidotic and probably malnourished individuals may therefore develop complications even with the slightest change in the heavy metal concentration in the body [10, 17].

There are several reports from Uganda geological departments showing that on the slopes of Mount Moroto in the remote north-eastern corner of Uganda where most rivers in the Acholi sub-region originate, members of the Karamojong tribe, including children, mine for gold, phosphate and uranium in the parched red earth [15, 16]. Some researchers have argued that NS was a result of contamination of the environment by heavy metal which became exposed to the young population in this region [11, 12, 15]. They argued that heavy metal toxicity could result in damaged or reduced mental and central nervous function, lowered energy levels and damage to vital organs [18]. Furthermore, they observed that long-term exposure to heavy metal could result in slowly progressive muscular and neurological degenerative processes similar to Alzheimer's disease, Parkinson's disease, Muscular Dystrophy and Multiple Sclerosis [18]. Organophosphate poisoning, which can occur from pesticide or chemical weapon exposures or from open mines, could similarly manifest into lethargy, coma and seizures [18]. Previous studies have shown that if the enzyme Acetyl-cholinesterase was suppressed in the nervous system such as in organophosphate poisoning, it could cause illness in animals and humans [18]. These arguments were derived from some of the typical presentation (signs and symptoms) of Nodding Syndrome that was observed in children in this region.

We recognised the fact that this epidemiological study was cross sectional and therefore it has its own limitations. In spite of our several postulations, follow up studies are

underway to extensively examine the environmental factors contribution to NS including tests for heavy metals in the water, silk, fish and tree root in these rivers.

CONCLUSION:

Nodding Syndrome in the Acholi sub-region presents in clusters involving certain specific geographic locations. These geographic locations are on either sides of Aswa and Pager Rivers which have their source of water in the Karamoja region where there are suspected open mines with several minerals including Gold and Uranium. It is recommended that detailed analysis of water, silk, fish, barks of trees, roots and soil samples in the clustered areas be conducted. Samples for analysis should be collected along these rivers from the source in Karamoja to Amuru.

Conflict of interest: Authors declare no conflict of interest

ACKNOWLEDGEMENT:

We wish to acknowledge the support and contribution of the District Health offices in the Acholi sub-region and the support of the village health teams for guiding us throughout the region. Special appreciation goes to the children with NS and their families for their consent and willingness to participate in the study.

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CASE REPORTS

ERUPTION CYST: A CASE REPORT

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Running Title: Eruption Cyst

ABSTRACT:

Eruption cyst (EC) is a benign cyst associated with a primary or permanent tooth in its soft tissue phase after erupting through the bone. It is most prevalent in the Caucasian race. It is clinically significant in that knowledge among general dentists is very essential regarding this developmental disturbance to reach the correct diagnosis and to provide proper treatment. We are reporting a case of eruption cyst in an 11 year old boy.

Keywords: Benign cyst, eruption cyst, eruption hematoma, simple excision

(Submitted December 2012; Accepted February 2013)

INTRODUCTION:

The eruption cyst is a form of soft tissue benign cyst accompanying an erupting primary or permanent teeth and appears shortly before appearance of these teeth in the oral cavity [1]. Eruption cyst is the soft tissue analogue of the dentigerous cyst, but recognized as a separate clinical entity [1]. Literature shows small number of reported cases of eruption cysts and they appear to be more prevalent in the Caucasian race [2].

The cyst results from a separation of the dental follicle from the crown of an erupting tooth and fluid accumulation occurs within this created follicular space [3,4]. A case report of eruption cyst in the maxillary arch in an 11 year old boy is presented.

CASE REPORT:

An 11 years old boy reported to the dental outpatient department with a complaint of swelling on the upper right back teeth region since 2 days. Clinical examination revealed a 1 x1 cm dome shaped raised swelling in the region of maxillary left first premolar, which was bluish-brown in color, asymptomatic, except the appearance (Figure 1). Intra oral periapical radiograph showed a pericoronal shadow of soft tissue covering the erupting tooth (Figure 2). Based on the history and clinical findings a diagnosis of eruption cyst was made. Surgical exposure was carried out to expose the erupting tooth (figure 3).

The specimen send for histopathological examination which showed surface oral epithelium on the superior aspect, underlying lamina propria showed variable inflammatory cell infiltrate the deep portion of the specimen which represents the roof of the cyst showed thin layer of non keratinizing squamous epithelium. Thus diagnosis of eruption cyst was confirmed.

DISCUSSION:

The prevalence of eruption cysts (EC) may be low due to the fact that many authors classify them among the dentigerous cysts. In addition, since they are benign, there are a few studies in which the authors have done a definitive diagnosis using biopsy [1]. Most often the dentist sees only symptomatic eruption cysts and the majority resolve unnoticed. EC most commonly are found in the mandibular molar region [5]. The color of these lesions can range from normal to blue-black or brown, depending on the amount of blood in the cystic fluid [6]. The blood is seen secondary to trauma. If trauma is intense, these blood-filled lesions sometimes are referred to as eruption hematomas [7].

Most EC occur in the age group of 6-9 years, with the eruption of permanent first molars and incisors [1]. In the present case study the eruption cyst was found associated with permanent premolar. Clinically, it appears as a

dome shaped raised swelling in the mucosa of the alveolar ridge, which is soft to touch and the color ranges from transparent, bluish, purple to blue-black [1]. Eruption cyst occurs most frequently on the right side than left and among males than in females [1].

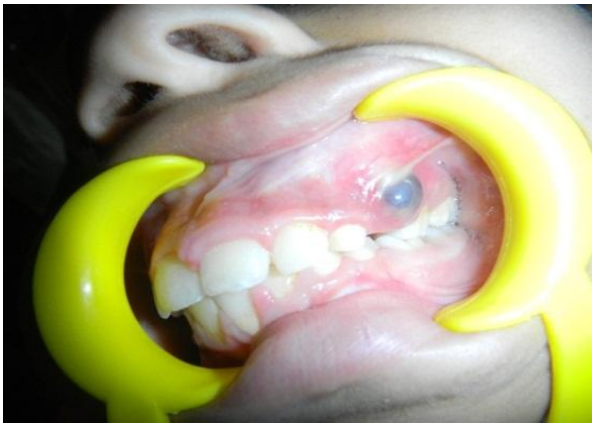

Fig. 1: Bluish brown dome shaped raised swelling in the region of maxillary left first premolar of 1x1cm size

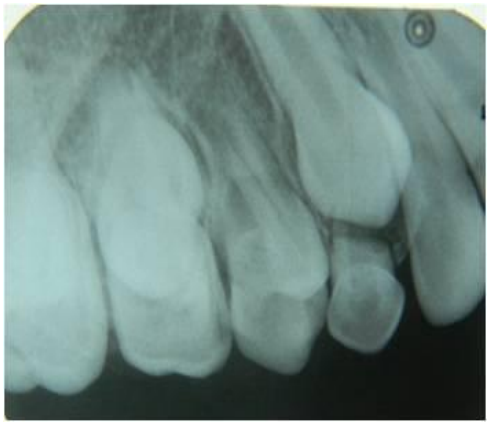

Fig. 2: Intra oral periapical radiograph showed a pericoronal shadow of soft tissue covering the erupting tooth

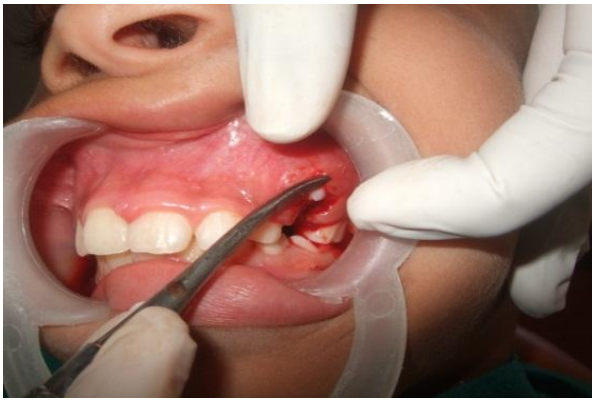

Fig. 3: Surgical exposure was carried out to expose the erupting tooth

In the present case it occurred on the left side. Most often, eruption cysts are found to be asymptomatic but there can be pain on palpation due to secondary factors such as trauma or infection [8]. Pain was reported as a secondary factor. Differential diagnosis should

be considered before delivering any treatment and varies from granuloma, amalgam tattoo and eruption hematoma [8]. The eruption hematoma occurs because of bleeding from the gum tissue during eruption and the accumulation of blood is external to the epithelium of the enamel [9]. While in the eruption cyst, it is the cystic fluid that mixes with the blood. The exact difference between the two is still unknown. The eruption cyst glows under trans illumination but the hematoma does not glow [8]. Other authors reported that if bleeding occurs within the cyst, due to trauma or local infection, the eruption cyst becomes bluish in color and is then known as an eruption hematoma, or a blue stain, which may be the first sign of a follicular cyst [1]. The eruption cysts do not require treatment and majority of them disappear on their own [10]. Surgical intervention is required when they hurt, bleed, are infected, or create esthetic problems [1]. If the cyst does not rupture spontaneously or the lesion becomes infected, the roof of the cyst may be opened surgically [4]. Interventional treatment may not be necessary because the cyst ruptures spontaneously, thus permitting the tooth to erupt [9]. If this does not occur, simple excision of the roof of the cyst generally permits speedy eruption of the tooth [9]. Simple incision or partial excision of the overlying tissue to expose the crown and drain the fluid is indicated when the underlying tooth is not erupting or the cyst is enlarging. Use of Er, Cr-YSGG laser for treatment of eruption cysts is suggested by Boj *et al.*, [10,11,12]. It has certain advantages over conventional exposure with scalpel. They can be listed as non-requirement of anesthesia, no excessive operative bleeding, does not produce heat or friction and patient will be comfortable. It is bactericidal and has coagulative effects, tissue healing is better and faster, and it is not associated with postoperative pain [11,12]

CONCLUSION:

EC is clinically asymptomatic but when it gets secondary infected causing pain. Patient or the parents usually bothered about the appearance. Since the tooth erupts through the lesion, no treatment may be necessary.

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UNUSUAL PRESENTATION OF KERATOCYSTIC ODONTOGENIC TUMOR IN THE MANDIBLE WITH IMAGING FEATURES – A CASE REPORT

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(**Running title**: Keratocystic Odontogenic Tumor)

ABSTRACT:

Keratocysytic Odontogenic tumor is a lesion occurring in the oral cavity that has a high recurrence rate. This lesion has an ability to mimic other lesions affecting the jaw. The tumor has a varied clinical and radiographic presentation. This article presents a case report of a 45 year old male patient presenting with an asymptomatic swelling on the right lower jaw associated with an impacted tooth and depicts an unusual radiographic picture.

Key words: odontogenic tumor, keratocyst, recurrence,

(Submitted December 2012; Accepted February 2013)

INTRODUCTION:

Keratocystic odontogenic tumor (KOT) defined by the World Health Organization (WHO), is a benign, intraosseous neoplasm of dental origin, with a characteristic lining of parakeratinized stratified squamous epithelium [1]. It was previously known as Odontogenic keratocyst and was first described by Phillipsen in 1956 [2]. The term Keratocystic odontogenic tumor was recommended by WHO which describes its neoplastic nature. It has a slight male predilection, usually between the second and third decades of life, but can occur at any age group. It can be located anywhere in the jaws,

most commonly occurring in the mandibular posterior region [3].

It can be considered as a benign odontogenic tumor with many distinguishing clinical and radiological features. The radiographic appearance of the lesion varies widely, which makes the diagnosis difficult. Herewith in this article, we report a case of Keratocystic odontogenic tumor in a 45 year old male patient who presented with an asymptomatic swelling in the right lower jaw that has varied distinguishing clinical and radiological features.

CASE REPORT:

A 45 year old male patient visited the department of Oral Medicine and Radiology, with a complaint of asymptomatic swelling in the left lower jaw since one month. He noticed the swelling one month back which gradually increased to the present size around 3×3 cm in diameter. Swelling began following a tooth ache and it was associated with throbbing type of pain which aggravates upon touch and during mastication. His medical and family history was noncontributory. Extra oral examination revealed the presence of diffuse swelling over left lower jaw measuring approximately 4×3 cm in size, extending inferiorly 0.5 cm below the inferior border of mandible. Swelling was non fluctuant, non-compressible and hard in consistency.

Intraoral examination revealed diffuse swelling involving left buccal vestibule extending posteriorly towards the retromolar region leading to obliteration of the left buccal vestibule Swelling was non tender and hard in consistency. Left mandibular canine was found to be missing and there was erosion of bone in relation to left mandibular lateral incisor and first premolar through which purulent discharge was visualized. Buccal cortical expansion was evident and there is thinning of bone lingually which made mylohyoid ridge more prominent. Severe cervical abrasion was noted in relation to left mandibular first premolar, second premolar and first molar. Aspiration revealed yellow coloured fluid. On vitality test, all mandibular teeth except mandibular right second premolar, first molar and second molar showed no response. Considering the history and clinical examination, provisional diagnosis of radicular cyst was made.

Panoramic radiograph revealed well defined hazy radiolucent lesion with a sclerotic margin involving the lower jaw (Fig. 1). Margins appear to be smooth, regular and non-corticated. Superior aspect of the lesion has a scalloped margin with respect to the teeth. The lesion extends from the distal aspect of left mandibular second molar, grows anteroposteriorly, and crosses the midline and reach till the distal aspect of right mandibular first premolar. Displacement of the teeth and resorption of roots of the teeth are evident. Presence of multiple septae with the radiolucent lesion gives a multilocular

appearance to the lesion. There was presence of tooth like radiopacity suggestive of an impacted tooth within the radiolucent lesion, located near the inferior border of the mandible. However the inferior border of the mandible was intact. Computed tomographic (CT) axial view showed a well-defined hyperdense area involving the mandible with its buccolingual and anteroposterior extension (Fig 2). The lesion crosses the midline to involve the other half of the jaw. Buccal cortical plates appear to be intact but there was perforation of lingual cortical plate on the left side of the jaw. There was also an evidence of hyperdense structure in the middle of the lesion suggestive of an impacted tooth.

The 3D reconstructed computed tomographic view showed the exact extension of the lesion with an evidence of through and through perforation of the mandible with respect to the lesion (Fig 3). On the basis of history, clinical and radiographic examination a final diagnosis of Keratocystic odontogenic tumor was made. In the present case seeing the extent of the lesion a surgical excision was planned. Surgical removal of the tumor was performed along with partial mandibulectomy and the specimen was sent for histopathological examination. Histopathologic picture showed parakeratinized stratified squamous epithelium largely of a uniform thickness overlying a fibrous connective tissue showing focal areas of severe inflammation. Epithelial connective tissue interface was flat with basal cells of epithelium showing palisading appearance. Areas also showed proliferation and rete ridge formation with loss of surface keratinization. Histopathologic picture of the given section of the lesion was suggestive of infected odontogenic keratocyst (Fig 4).

Treatment carried out was enucleation of the lesion with partial resection of the mandible following which surgical plating was done. Patient was reviewed after 6 months, reported with no recurrence.

DISCUSSION:

Keratocystic odontogenic tumor (KOT) is a benign unicystic or multicystic, intraosseous tumour of odontogenic origin. KOT form approximately 11% of all jaw cysts and they have a very high recurrence rate. The reason for the high recurrence rate may be due to the proliferation of islands of odontogenic epithelium that may be present in the wall giving rise to satellite microcysts. It has been reported that KOT most commonly occur between second and third decade of life but may be diagnosed at any age [3].

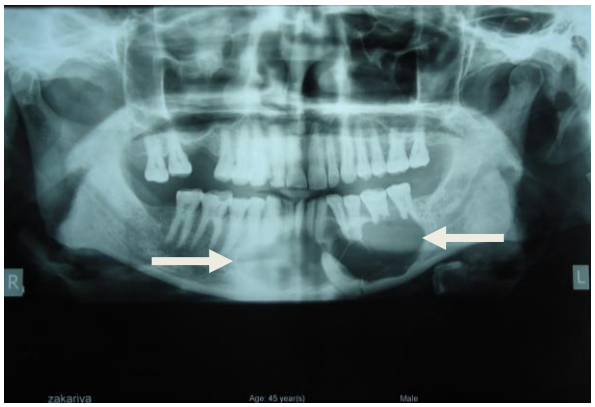

Fig: 1: Panoramic radiograph reveals a well defined hazy radioluscent lesion with superior scalloped margin with an evidence of resorption and displacement of teeth and an impacted tooth

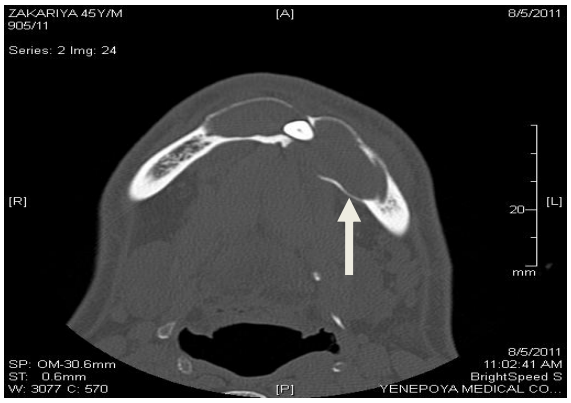

Fig. 2: CT axial view showing the extent of the lesion with the evidence of an impacted tooth and perforation of lingual cortical plate

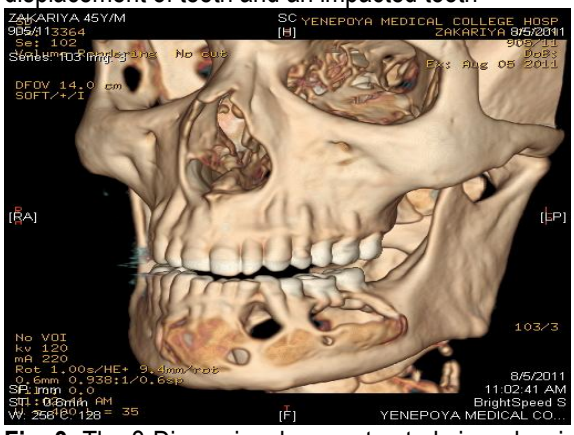

Fig. 3: The 3-Dimensional reconstructed view showing the extensive lesion with buccal & lingual cortical plate perforations. Shows the presence of bony septa within the lesion

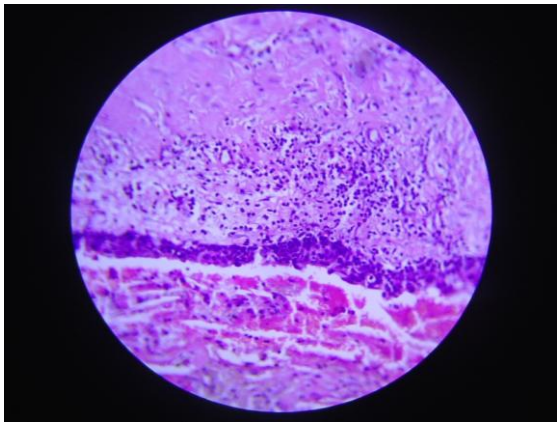

Fig 4: Histopathologic picture showing parakeratinized stratified squamous epithelium and basal cells of epithelium showing palisading appearance (10X zoom)

In another study, it was reported that the incidence was highest in the older age groups, and a decade younger in woman than in men [4]. The mandible is involved more frequently than the maxilla, the percentage of KOT occurring in the mandible ranges from 65-83% of cases [5]. The tumor can occur at any site in the mandible but most of the lesions arise in the posterior body, angle and ascending ramus of the mandible [4]. The lesion may be symptomless unless infected. It sometimes forms around an unerupted tooth; sometimes adjascent teeth may be displaced [1]. The present case was a 45 year old male patient with an asymptomatic swelling in the mandible with evidence of an unerupted tooth.

Unlike other lesion, the epithelium of KOT appears to have an innate growth potential.

This difference in growth mechanism gives it a different radiographic condition. Conventional radiographs such as panoramic radiographs may be adequate in most of the cases to determine the location and estimate the size of KOT. Advanced imaging modalities such as computerized tomography may be required to assess the full extension of the lesion [5]. In the present case, both the panoramic and computed tomographic imaging was taken.

KOTs occur as a well defined lesion which may be unilocular or as multilocular [6]. Most of the lesions of KOT are unilocular and often appears with smooth and regular borders. In the present case, panoramic view showed a well-defined multilocular hazy radioluscent lesion with superior scalloped border crossing the midline in the mandible with the presence of multiple septae making the lesion multilocular. The presence of septae within the bony cavity is the most striking feature of ameloblastoma, which serve to produce partial loculation of the cavity [7]. Scalloping of the sclerotic margin of the lesion that extends between the roots of the teeth is a characteristic feature of traumatic bone cyst. In the present case, scalloping of the margins was seen over the anterior part of the lesion which is a distinguishing feature. Since the present lesion was associated with an impacted tooth, dentigerous cyst should also be considered in the differential diagnoses [7]. Here the lesion emerges from the middle third root portion of the impacted tooth, and not from the cemento-enamel junction of the tooth, which is most frequently seen in case of a dentigerous cyst. The lesion in the present case extends from the posterior aspect of left mandibular second molar and was not localized surrounding the impacted tooth as in case of dentigerous cyst.

On clinical intraoral examination of the reported case, thinning of lingual cortical plate with increased fluctuation was felt, suggestive of perforation of the cortical plate. The perforation of the cortex was not well appreciated with two-dimensional image on the panoramic radiograph. However, the exact dimension of the lesion and the perforation of buccal and lingual cortical plates were visualized by CT scan sections and its three dimensional representation. Thus computed tomography plays an important role in imaging of an extensive lesion, thereby aids in the diagnostic process as well as treatment planning.

Histopathologic examination is usually required to arrive at a definitive diagnosis of KOT. Histologically KOTs can be classified into three categories: orthokeratinised, parakeratinised, or a combination of both [8]. The orthokeratinized subtype produces normal skin keratin with the presence of keratohyaline granules whereas the parakeratinized subtype involves disordered production of keratin without keratohyaline granules. The parakeratotic type has a more aggressive clinical presentation and is more frequent (80%) than the orthokeratotic variants [8]. The

present case was a parakeratinized variety with areas of proliferation and rete ridge formation, which has a more chance for recurrence.

Many treatment options for KOT have been described in literature. The main goal of treatment is to reduce the risk of recurrence and morbidity of extensive resection [9]. Treatment modalities such as decompression, simple enucleation with or without curettage and resection have been employed in the treatment of KOT [10]. In the present case enucleation of the lesion along with partial resection of the mandible were carried out.

CONCLUSION:

The clinical and radiographic features of KOT's are not pathognomonic. It usually becomes difficult to distinguish with other lesions especially when the lesion is destructive with varied features. Advanced imaging is necessary for the visualization of its full extent following which definitive diagnosis is made histopathologically. The tumor requires a long term follow up since it has an aggressive and recurrent nature.

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LETTER TO THE EDITOR

IMPROPER USE OF MY RESEARCH DATA

Aravind R. Kudva

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Dear Editor-in-Chief

IMPROPER USE OF MY RESEARCH DATA

I would like to bring to your notice that the Manuscript PJMS MS 169 IG 2012, titled "Efficiency of Sodium Hypochlorite and Four other Intra Canal Medicaments in Eliminating the Candida albicans in the Root Canal System – An Ex-vivo Evaluation," which was published in your esteemed journal, *Pacific Journal of Medical Sciences* (PJMS), Volume 10, No. 1, June 2012, pp. 28–34, is not the original work of the purported authors, Gary Ignatius and K. Pradeep. They acquired the data wrongfully.

The research was done by me during the 2008 academic year in the Department of Conservative Dentistry and Endodontics, Yenepoya Dental College, Mangalore. During my Post Graduate studies, I completed the research project under the supervision of my Professor and Head of the Department Dr. K Harish Kumar Shetty. The details of my work, including the photographs and research data, were in a Compact Disc submitted to the Department.

One of my Post Graduate classmates, Dr. Gary Ignatius, who is currently my colleague in the Department, is the "Corresponding Author," as indicated in the published article and confirmed by the information received from the Office of the Managing Editor of PJMS. Without my knowledge and consent, and also without the consent and approval of my supervisor, Dr. Gary Ignatius took the data from the CD, wrote it up and submitted the manuscript to the PJMS for publication.

The Ethics Committee of Yenepoya Dental College, Mangalore, objects to giving credit to the alleged authors for the work they did not do. As such, the “authors” have been asked to ensure that the paper is withdrawn from publication and that it is not cited in any scientific forum. They have been requested to fulfil all the requirements of your journal for withdrawing published papers from circulation.

I am kindly requesting that this letter be published in one of the issues of PJMS and that appropriate action be taken, if possible, to withdraw the paper from further circulation.

Yours Sincerely,

Signed ***Signed***

Dr. Aravind R Kudva

Dr K Harish Kumar Shetty
Head of Department, Conservative Dentistry and Endodontics, Yenepoya Dental College, Mangalore

(Submitted: February 2013, Accepted March 2013)

INSTRUCTIONS FOR AUTHORS

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Book:

Gillett JE. The health of women in Papua New Guinea. PNGIMR: Kristen Press, 1991

Chapter in a Book:

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Published proceedings paper:

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