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LETTERS TO THE EDITOR

CHANGE IN THE PATTERN OF PAEDIATRIC MAXILLOFACIAL FRACTURES SEEN IN KADUNA, NORTHERN NIGERIA

Trauma is the leading cause of morbidity and mortality among children worldwide.[1] However, in comparison with adults, maxillofacial fractures in children are relatively uncommon due to physiological and environmental factors. Between 4% and 12% of all maxillofacial fractures occur in children.[2-5] The reasons for the wide disparity in incidence rates include differences in age limit of the pediatric population studied, types of injuries classified and the socio-economic status of the population which influences access to health care facilities.

Nigeria, like many developing countries in Africa has witnessed tremendous socio-economic and demographic changes in the past 20years. This has altered the pattern of some health conditions. Since the last published series on pediatric maxillofacial fractures from our center in 1980,[2] to our knowledge no recent review from our center has been presented. This is important as our center was the first oral/maxillofacial care center in northern Nigeria and remains an important tertiary care facility in the region. The aim of this report was to evaluate the current pattern of maxillofacial fractures in the urban Northern Nigerian pediatric population as seen at the Maxillofacial Unit, Ahmadu Bello University Teaching Hospital, Kaduna, Nigeria for comparison with other Nigerian and international records.

A retrospective survey of cases of maxillofacial fractures seen between 1991 and 2000 at the Maxillofacial Unit, Ahmadu Bello University Teaching Hospital, and Kaduna, Nigeria was undertaken. Children aged 15 years and below were selected out for further study. Materials reviewed include case notes, radiological reports and theater records. Information retrieved for analyses were age, sex, cause of fracture, site (s) of facial fracture, associated injuries and treatment. Poor return for follow-up made it impossible to review complications. Mandibular fractures were classified as anterior that is, between the canine teeth, posterior – from canine to end of occlusion, angle, ramus, condyle, dentoalveolar and coronoid types. Middle third fractures were classified as Le Fort type, Zygomatic complex and nasal complex types.

Out of 443 cases of maxillofacial fractures seen within the study period, 21 (4.7%) were children aged 15years and below. There was a bimodal peak age of incidence at ages 9-10years old (n = 7, 33.3%) and 13-15years old (n = 8, 38%). The mean age of the children was 11.5 ± 2.6years (median age 11years) with no child below 6years of age. There were 15 males and 6 females. The main etiological factors were falls 12, road crashes 6, and fights 2. A total of 29 maxillofacial fractures were seen in the 21 patients giving a fracture to patient ratio of 1.4:1. Five patients (24%) had fractures of the middle-third and mandible but fractures most were mandibular only (14, 66%) while the rest were in the middle-third of the face alone (10). Treatment of cases is shown in Table 1. No associated injuries were recorded in the 21 children with maxillofacial fractures seen in this study.

Table 1. Treatment methods of maxillofacial fractures seen among Northern Nigerian children 15 y old and below

Treatment modality	No.
Arch bar only	11
Eyelets and arc bar	4
Eyelets only	3
Arch bar + nasal POP	1
Arch bar + transosseous wiring + frontomandibular suspension	1
Gillies` temporal approach	1
Total	21

POP: Plaster of Paris

According to Nørholt et al.,[6] fractures of the pediatric maxillofacial skeleton are rare due to the resiliency of their bones, the relatively small size of their body in proportion to the head and the comparatively protected lifestyle of children. As they grow older, and get involved in more social activities, their incidence of fracture increases. Unlike in the 1980 report from our center,[2] where 85 cases were seen in children aged 11 years old and below within 5years, we saw 21 cases aged 15years and below within 10years reflecting an apparent decrease in cases seen. Two reasons are postulated.

As at 1980, our center was the only one managing maxillofacial fractures in the entire Northern Nigeria, hence the center "harvested" all the cases seen in the region while twenty years later several tertiary and secondary care facilities are in existence providing health care services to the population. Also, the economic downturn in Nigeria which started in the mid-1980`s has decreased the level of car ownership of the Nigerian population. Previous Nigerian reports have shown that road crashes are responsible for most maxillofacial fracture cases.[7-10]

Generally, between 4%-12% of maxillofacial fracture occur in children.[2-4] The incidence rate of 4.7% in this study is within the range of these earlier reports. The low incidence of facial fractures among children is due to physiological and environmental factors enumerated as; greater resilience of the pediatric skeleton, higher bone to tooth ratio direct parental supervision of the activities of young children and limited outdoor activity,. As they grow older, the incidence of facial fractures rises.[6,11-15]

Among Nigerian children, maxillofacial fractures occur twice as often in boys as in girls.[2,3] In a report from Ile-Ife, a semi-urban town in southwestern Nigeria, the male to female ratio was 3:2 possibly due to the predominance of road crash-related fractures in their environment.[4] The present male to female ratio of 2.5:1 is similar to the earlier finding from our center.[2] In the 1980 study, 15.3% of the children seen with facial fractures were 5years old or below.[2] The current study had no case in this age group. The bimodal peak seen in the earlier report (age 8-9years 23.5%, age 12-13years 32.9%) has shifted to age 9-12 years (33.3%) and age13-15years (35.2%) in the present study. While the risk of fractures generally increases with age,[1-4,16] it is speculated that the age-related variations in injuries sustained are attributable to head-body relationship changes and development status of facial structures especially teeth and sinus.[1,3,17]

The main etiological factors in our report are falls (57%) and road crashes (29%) unlike in the 1980 report (falls 30.5%, road crashes 54.1%). This reversal reflects the decreased car ownership among Nigerians due to the economic downturn of the last 20years. An etiological pattern similar to our current finding was also in the report from Enugu, another urban center in Eastern Nigeria,[3] but unlike that from the semi-urban Ile-Ife.[4] The etiological pattern in Nigerian urban centers is similar to that found among Austrian children.[5] This is unlike in the adult population were the etiological pattern in both the Nigerian urban and rural population are alike but dissimilar to that of Western Europe.[2, 9, 10, 18,19] A probable reason is that urbanization and changes in the lifestyle of Nigerian urban population is increasingly tending toward that of the European population hence parents may be more careful in supervising their children leading to reduction in rate of road crashes in children. Also, it is possible that severe cases of trauma do not make it to hospital.

Despite differences in etiological pattern between the pediatric and adult population, the jaw distribution of fractures is similar. More mandibular fractures are recorded in children than in the middle-third of the face.[2-4] The reasons for this distribution have been previously reported.[10] Adekeye[2] found that mandibular fractures were in the symphysis (24%), body (21%) and dentoalveolar (21%) and condylar (12%) regions. While symphyseal mandible is within our definition of anterior, more of our cases were in the anterior (n = 7, 30% of mandibular sites) with the rest as dentoalveolar (n = 6, 26%) and posterior (26% of mandibular sites). This absence of condylar fractures in our study as compared to the 1980 report is surprising as they are more related to cases of fracture due to falls as the predominant etiological agent.[17,20] However, while falls in our series were often in the home ie, associated with daily living, that in European studies are usually from bicycle and motorbikes resulting in greater impact on the chin.

Associated injuries with maxillofacial fractures could be life-threatening if not detected quickly and managed expertly. We found none in our review of 21 cases of maxillofacial fractures while Adekeye,[2] had a 50% rate. While absence of associated injuries could be due to missed diagnosis; it is observed that high impact injuries such as road crashes and gun-shot injuries are more associated with trauma to other body parts than falls.[9,16]

The methods of treatment of maxillofacial fractures in Nigerian children have been documented previously.[2-4] In our report, similar methods of treatment were utilized. While poor follow-up visits limit our ability to evaluate the implications of treatment on the growth of these children, we believe the results are satisfactory. Using mandibular fractures, Moreno et al.,[21] found that post-operative complications rates are fundamentally related to the severity of the fracture rather than to the treatment modality utilized.

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SPONTANEOUS VAGINAL DELIVERY OF UNDIAGNOSED BIPAGOUS CONJOINT TWINS

Conjoint twins are usually rare events. The incidence in our environment has not really been documented but there have been previous reports.[1,2] Conjoint twins are classified in different ways. If they are fully formed except for the parts of con-junction, they are called bipagous conjoint twins (or duplicata completa).

Vaginal delivery of conjoint twins was documented in 1950[3] and 1981[4] in Western countries. None, to our knowledge, has been reported in Nigeria. We report a case of undiagnosed bipagous conjoint twins that were delivered by Spontaneous vaginal delivery, unassisted and in a primary level health facility.

A 29-year old woman on the 18th of September 2004 with history of delivery of a set of conjoint twins at a Rural Health Centre.

She was gravida 4 para 3 with 2 living children. Though she registered for antenatal care in a private hospital, she did not carry out any investigation including ultrasound or plain x-rays. She had nothing suggestive of “big –for- date” pregnancy or polyhydramnios during the pregnancy. At about 39weeks, she went into spontaneous labor and was rushed to the nearest rural health center where she was assisted by the attendant midwife to have a spontaneous vaginal delivery of a set of conjoint twins. Both twins cried immediately after birth, passed meconium from their respective ani and also passed urine normally. They were promptly referred to our Teaching Hospital.

At presentation the babies were about four hours old, cold to touch and wrapped in their mother’s delivery cloths. Both were females with a

combined weight of 2.7kg. They were both conscious, satisfactorily active with good intermittent cries. They were neither pale nor jaundiced. Their skins were joined from the sternum down to the hypogastrium. Twin 1 looked smaller and dehydrated. There was cyanosis on the left leg and right foot. There was also edema on the left foot. Her respiratory rate was 34 cycles per minute, the heart rate was 120bpm, and the femoral pulse on that left leg was not palpable. First and second heart sounds were heard; there were no murmurs. The breath sounds were vesicular in both lungs fields. The Bowel sounds were present and normal. A rectal examination confirmed that she had patent anal orifices with good sphincteric tone. All other systems were grossly normal. Twin 2 was bigger and looked generally healthier than twin 1. All her vital signs and clinical findings were essentially normal. The diagnosis of Thoraco-omphalopagous conjoint twins was then made.

Size 6 feeding tube was successfully passed into the stomach of each of the babies and gastric juices with bile were obtained from each of them. 10% Dextrose in 1/5 normal saline was set up on each of them to run as calculated by their combined weight. Vitamin K injection was given to each intramuscularly. The babies were kept warm by wrapping with warm packs and blanket. It was decided to refer them to center that is conversant with management of conjoint twins. After three days at the referral hospital, the babies suddenly died one after the other. Aspiration of feeds was highly suspected, because there were no ante-mortem signs of distress.

Postmortem examination revealed that all the joining from the sternum to the hypogastrium was predominantly at the skin level. No organs were shared. The sternae, the ribs, the thoracic cavities and viscera were all separate. However, the heart of twin 1 had a single chamber from which emanated the aorta. The heart of twin 2 had the usual 4 chambers but they were enlarged. Other systems were essentially normal. Cause of death was then ascribed to Heart failure.

The exact incidence of conjoint twinning is unknown and figures are unreliable because not all cases reach the hospital; some are aborted while some are thrown away as monsters. The figure however is in the range of 1 in 14 000 births in the non-caucasian.[5]

Although vaginal delivery for conjoint twins has been reported, [1]when diagnosed in utero, the recommended mode of delivery is cesarean section not vaginal delivery, because of the attendant risks to both mother and twins.[2,4] Undiagnosed conjoint twins may cause dystocia in labor leading to emergency operative delivery thereby endangering the survival of the twins. Surprisingly, there were none of such in this case. When diagnosis is made after birth, prompt transfer to a more ideal institution is a wise decision, as was done for these twins.

There is great need to improve the health care delivery system to make it available and accessible to all our pregnant women. Education of our women on the need for antenatal registration, regular antenatal clinic attendance and hospital delivery should be emphasized. This will make for early detection of such anomalies so that proper arrangement could be made for safe delivery and management of the conjoint babies.

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PERCEPTION AND MANAGEMENT OF GUINEA WORM DISEASE IN INFECTED AND AT RISK NON- INFECTED COMMUNITIES IN OYO STATE, NIGERIA

Dracunculiasis or guinea worm disease caused by worm infestation continues to be a public health problem among rural communities in Africa, which depend upon unprotected water sources for drinking.

Its health, social, educational and economic cost to the individual, the household and the community which is considerable and it's transmission cycle are well documented.[1,2] Key intervention strategies to eradicate guinea worm are safe water supply, vector control using abate, health education and case management. Besides the worm itself and the stagnant water/copepods combination, the third element in the cycle of the disease is the human being. This is probably the key element, for without the people's cooperation and understanding none of the control strategies or intervention efforts will have much chance to succeed.

The aim of this study therefore is to find out knowledge, attitude and practises (KAP) in the management of guinea worm disease in infected and at-risk non-infected villages.

This study was carried out in12 villages in Akinyele Local Government Area (LGA) of Oyo State, Nigeria which lies between latitude 7°26/ 23// N to7° 40/ 30// N and between longitude 3° 47/ 4// E to 4° 05/ 00// E.

Infected villages include these ones with their population at the time of study, Alagba (297),Oyeteju (45) and Olomitutu (215) while at-risk non-infected villages include Apapa (50houses), Alase (45houses), Ketepe (77), Deinlokun (25 houses), Aba Oso (56), Olorisaoko (55 houses), Alore (16 houses), Aroro (10 houses) and Ajibade (over 100 houses). Pretested and structured questionnaires were administered to all heads of households in infected and small non-infected villages while it was administered to every other house in bigger non-infected villages. The questions were translated into the language of the community, Yoruba. The questionnaire was used to record the demographic information of respondents. Source and treatment of drinking water, time of first infection, season, anatomical location, duration and severity of the infection were recorded. Effect of the disease on farming activities, information on the medication used and the knowledge of the disease were also recorded.

Two hundred and sixty(260) households were sampled. 35.4% households in infected villages and 64.6% households in non-infected villages. 63.1% males and 36.9% females were sampled.80.8% were farmers. The number of respondents (75) that practice boiling and filtering of drinking water in infected villages is significantly higher than the number of respondents (32) that practice same treatment in non-infected villages(X^2 = 95.5 ; P<.05) .Some respondents use alum as a preventive measure against guinea worm disease. 46.7% of the respondents from infected areas and 48.8% from non-infected areas had been infected before, 29.6% had their first infection as a child and had been re-infected many times. Most (68.8%) of the respondents were infected in the dry season. Most (91.2%) had the guinea worm coming out of their legs (Figure 1). Almost all the respondents (96%) had only one worm emerging at any one time. The period of disability in treated and untreated cases lasted from 1- 10 days to over a year. Table 1 shows symptoms perceived before bleb formation. 73.6% had their guinea worm ulcer healed within 1- 30 days. Most (81.6%) use palm oil for treatment. Herbs used include the traditional drug (agbo) made from boiled medicinal leaves (Olugambe leaves – *Ipomea* spp). Most of the respondents infected before (76%) could not farm at the time of infection and only 12% had alternative labor. Table 1 also shows that majority of the respondents from the infected villages (65.2%) and 66.1% from non-infected villages were ignorant of the fact that the guinea worm infection is caused by drinking contaminated water. When respondents were further probed,70.7% in infected villages and 76.8% in non – infected villages did not know guinea worm is transmitted in drinking water.

81.2% of the respondents from all areas believed that all ages are equally susceptible to infection. There is no significant difference between the number of people that know susceptibility is due to the use of bad water in infected and non-infected areas (X^2 = 0.26;P>.05). 84.2% from all villages did not know if the disease is preventable or not. 64.4% said it could be treated using palm-oil. Majority of the respondents from all areas (77.3%) claimed to be doing nothing to control the disease in their houses. Of significance are the 10.4% that claimed to treat their drinking water for control.

It is necessary to educate the villagers on the role of ponds as transmission sites, on the need to boil water and filter drinking water and the inability of the alum to kill the parasites intermediate hosts.. Educating villagers in all areas will help curb the menace of importation of the disease to at – risk non-infected areas where little or nothing is done about disease prevention.

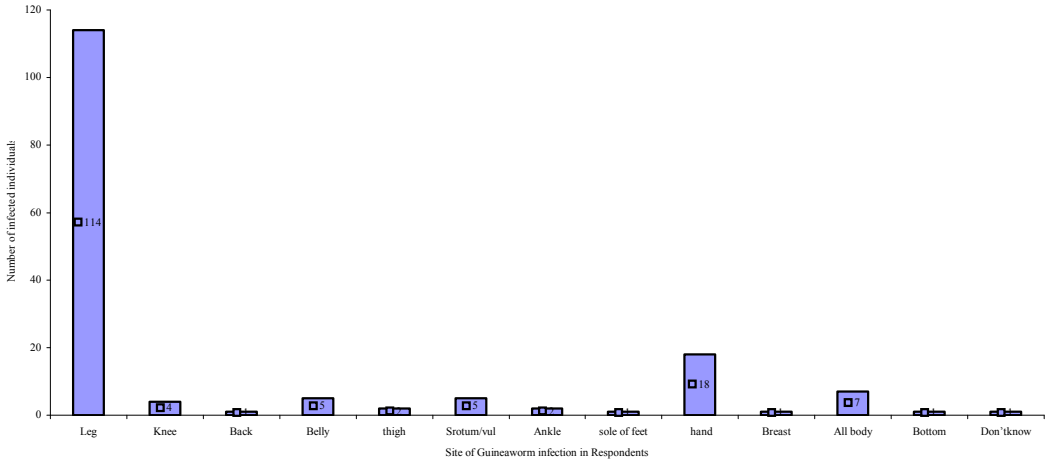
This study shows that guinea worm infection does not confer any tangible protective immunity on the patients. In this LGA where rainfall rises to a peak of 1000mm in the wet season, transmission is restricted to the tail end of the dry season. Patent guinea worm disease therefore reaches its peak in the following late dry season and early rainy season. Guinea worm came out mostly from the legs of those infected and most had only one worm emerging at any given time as found in studies by Kale.[2] The older respondents recognized the prodromal signs of guinea worm infection. Use of palmoil, shea-butter and herbs for treatment of the disease in this study conforms to earlier studies.[1]

The long incubation period of the disease (9-12 months) makes it hard for people to visualize the direct connection between the water they drink today and the disease they suffer the following year.

Table 1. Guinea worm infection in Heads of Households in Akinyele Local Government Area

Symptoms perceived before bleb formation	Infected areas	Non infected	Total
Rashes	21 (48.8%)	51 (62.2%)	72 (57.6%)
Burning sensation at site of blister formation	17 (39.5%)	28 (34.1%)	45 (36%)
Cold	9 (20.9%)	13 (15.9%)	22 (17.6%)
Blurred vision	1 (2.3%)	-	1 (0.8%)
Fever	2 (4.7%)	2 (2.4%)	4 (3.2%)
Appearance of worm under skin	2 (4.7%)	-	2 (1.6%)
Dk/none	7 (16.3%)	13 (15.9%)	20 (16%)
Cough	-	1 (1.2%)	1 (0.8%)
Headache	-	1 (1.2%)	1 (0.8%)
Time it took guinea worm ulcer to heal in respondents			
1 – 30 d	33 (76.7%)	59 (72.0%)	92 (73.6%)
Months	4 (9.3%)	7 (8.5%)	11 (8.8%)
Dk	3 (7.0%)	11 (13.4%)	14 (11.2%)
No ulcer	3 (7.0%)	5 (6.1%)	8 (6.4%)
Treatment applied to the guinea worm infection by respondents			
Palm oil	35 (81.4%)	67 (81.7%)	102 (81.6%)
Shea butter	17 (39.5%)	6 (7.3%)	23 (18.4%)
Herbs	6 (14.0%)	8 (9.8%)	14 (11.2%)
Hospital	3 (7.0%)	8 (9.8%)	14 (11.2%)
Perception of the cause of the guinea worm disease by respondents.			
Bad drinking water	13(14.1%)	18(10.7%)	31(11.9%)
Inherited	12(13.0%)	12(7.1%)	24(9.2%)
Others	7(7.6%)	30(17.9%)	37(14.2%)
Don't know	60(65.2%)	111(66.1%)	171(65.8%)
"do you know that guinea worm disease is transmitted through drinking water?"			
Yes	26(28.3%)	34(20.2%)	60(23.1%)
No	1 (1.1%)	5(3.0%)	6(2.3%)
Don't know	65(70.7%)	129(76.8%)	194(74.6%)
"which people in the community are usually susceptible to infection?"			
Any age	77 (83.6%)	134 (79.8%)	211 (81.2%)
Both 15+ male& female	-	1 (0.6%)	1 (0.4%)
Dk	15(16.3%)	33 (19.6%)	48 (18.5%)
Reasons for susceptibility			
Bad water	13(14.1%)	20(11.9%)	33(12.7%)
Inherited susceptibility	40(43.5%)	29(17.3%)	69(26.5%)
Don't know	42(45.7%)	120(71.4%)	162(62.3%)
"can guinea worm disease be prevented?"			
Yes	14(15.2%)	24(14.3%)	38(14.6%)
No	1 (1.1%)	2 (1.2%)	3(1.2%)
Don't know	77(83.7%)	142(84.5%	219(84.2%)
Ways of preventing the disease (out of those that said yes)			
Government action	3(21.4%)	1(4.2%)	4(10.5%)
Avoiding flood	-	1(4.2%)	1 (2.6%)
Good drinking water	11(78.6%)	15(62.5%)	26(68.4%)
Good food	3(21.4%)	-	3(7.9%)
Cleanliness	1(7.1%)	6(25%)	7(18.4%)
Traditional medicine	-	1(4.2%)	1(2.6%)

Figure 1. Site of Guinea worm infection in Heads of Households in Akinyele Local Government Area



Most (74.6%) of respondents interviewed did not know if the guinea worm is transmitted in drinking water or not which shows that water treatment was not done out of conviction.

Some of the respondents never infected before claimed to have movement of worms all over their bodies. Respondents believe infection is not from drinking water because some people in the infected villages had never been infected before although born and lived all their lives there and because they feel it is either in the body, blood or a family disease of those infected. Those that said it is from drinking water stated it must be from a common source.

When villagers' minds are not set on getting rid of the disease, it might be difficult to eradicate the disease completely in the country. The location of boreholes and wells is not possible in all areas because of small population and topography of some areas. It will therefore be advantageous if health education is made to cover infected and at-risk non-infected areas in order to speed up the eradication of the disease.

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