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Low Birthweight in Hausa Infants

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Summary

Rehan NE and Tafida DS. Low Birthweight in Hausa Infants. *Nigerian Journal of Paediatrics* **1981;8:35.** The incidence of low birthweight among 3,890 live-born Hausa infants was 21.3%, being significantly higher in females (23.6%) than in males (19.3%). The incidence appeared to be influenced by both maternal age as well as parity. Possible aetiological factors included multiple pregnancies, prematurity and toxaemia of pregnancy.

Introduction

THE World Health Organisation (WHO)[1] has recommended that in countries where national surveys have not been carried out to evaluate various health problems, data collected at regional levels or in various hospitals should be used to formulate local standards. This recommendation holds good for Nigeria because very little work has been done to study the various health problems at the national level, and also because significant regional variations may exist due to different ethnic compositions. Majority of previous studies of birthweight and its allied problems in Nigeria have been carried out in the southern parts of the country particularly in Ibadan and Lagos.[2-6]

There are only two previous reports of birthweights from northern Nigeria.[7,8] The former deals with a multi-ethnic group, while the latter deals only with the predominant ethnic group, the Hausas. Both reports have tried to establish the standard birthweights for the area and have made only casual references to low birthweight (LBW). Since infants with low birthweights constitute a group requiring specialised care, it was thought worthwhile to evaluate in detail, the problems of low birthweight among the Hausas.

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Materials and Methods

From the records of the deliveries conducted at the Maternity Hospital, Katsina, between 1st of January, 1974 and 31st of December, 1977, the case records of all live-born infants of Hausa parents were selected for the present study. The low birthweight infants, i.e., those weighing 2,500g or below[9] were separated, their characteristics studied and possible aetiological factors identified. A comparision of this group was then made with those neonates who weighed more than 2,500g. The duration of gestation was determined from the menstrual history and by appropriate clinical examination before delivery. The data so obtained were however not considered to be very reliable, therefore no attempt was made

to correlate the birthweights with duration of pregnancy. Infants born before the 37th week of pregnancy were labelled as "premature."[9] The birthweight in all these cases was recorded by using a metric scale before the first feed.

Results

During the study period, 3,890 live Hausa infants (2,111 males and 1,779 females) were born to 3,780 mothers. Of these 3,890 infants, 408 males and 420 females weighed 2,500g or less. These 828 infants were born to 774 mothers and included 91 sets of twins and five sets of triplets though the outcome of all multiple pregnancies were not live births.

The maternal ages ranged from 13 to 45 years. The mean age was 21.7 ± 6.5 years. The parity ranged from 0 to 14 but there were more primigravidae compared to other parities. All the mothers belonged to lower and middle socio-economic classes. Seventy per cent were from the urban population while 30% were from rural areas. Thirty-one per cent had attended antenatal clinic, and the rest were unbooked cases.

The incidence of low birthweight (LBW) was 213/1,000 live births or 21.3%. The incidences according to maternal age and parity are summarised in Tables I and II respectively.

LBW pattern among male and female neonates

Table III gives the distribution of the 828 cases of LBW according to their sex, into various weight groups as recommended by WHO Expert Committee on Maternal and Child Health.[9] The majority of these babies (71.1%) weighed between 2,000g and 2,500g while only 1.6% weighed less than 1,000g. Although males exceeded females among the total live births in a ratio of 54.3 to 46.7, the percentage of females among LBW infants was higher (50.7%) as compared to that of males (49.3%). The incidence of LBW was 19.3% among males and 23.6% among females ($P < 0.001$).

TABLE I

Incidence of Low Birthweight According to Maternal Age

Age (Years)	Total Deliveries		Low Birthweight		% of Low Birthweight
	No. of Mothers	No. of Live Births	No. of Mothers	No. of Live Births	
< 15	70	59	19	16	27.1
15–19	1,190	1,206	311	318	26.4
20–24	886	906	172	184	20.3
25–29	650	677	105	118	17.4
30–34	498	530	90	105	19.9
35–39	195	212	30	37	17.5
> 40	86	88	6	7	7.9
Not Recorded	205	212	41	43	20.3
Total	3,780	3,890	774	828	21.3

$X^2 = 21.57$ df6 $P < 0.01$

TABLE II

Incidence of Low Birthweight According to Parity

Parity	Total Deliveries		Low Birthweight		% of Low Birthweight
	No. of Mothers	No. of Live Births	No. of Mothers	No. of Live Births	
0	1,317	1,329	362	369	27.8
1	470	476	81	84	17.7
2	423	438	64	70	15.9
3	365	376	63	78	21.7
4	270	285	60	68	23.8
5	194	206	29	39	18.9
6	208	217	28	30	13.9
>6	533	563	87	90	15.9
Total	3,780	3,890	774	828	21.3

$X^2 = 36.49$ df7 $P < 0.001$

TABLE III

Low Birthweight Distribution among Male and Female Neonates

Sex	*No of Cases*	*< 1,000 g*	*1,001–1,500 g*		*1,501–2,000 g*		*2,001–2,500 g*		*Total No.of LBW*	*Total Live Births*	*% of LBW*
		%	*No*	*%*	*No*	*%*	*No*	*%*			
Male	8	1.9	41	10.1	80	19.6	279	68.4	408	2,111	19.3
Female	5	1.2	30	7.1	75	17.9	310	73.8	420	1,779	23.6
Total	13	1.6	71	8.6	155	18.7	589	71.1	828	3,890	21.3

Effects of Environmental Factors

The monthly and seasonal incidence of LBW was uniform and no seasonal variation could be found. The incidence of LBW was 18.4% among urban women and 23.4% among those living in rural areas, the difference being highly significant ($P < 0.001$).

Neonatal Deaths

As the mothers were usually discharged 24 hours after delivery and were often lost to follow up, it is very difficult to give an accurate incidence of neonatal deaths. However, 21 children died within 24 hours of birth in the hospital and their distribution according to birthweights is given in Table IV.

Aetiological Factors

The various factors associated with LBW in the present series are summarised in Table V, which also compares the frequency of these factors in the LBW group with their frequency among the neonates weighing over 2,500g.

Discussion

Compared with developed countries, a LBW incidence of 21.3% appears to be very high, but when we compared our findings with those from other parts of Nigeria, we find an almost comparable incidence. Ladipo and Adelusi [6] have reported an incidence of 24.9% for UCH, Ibadan, while the figures quoted by Effiong *et al*, [5] from the same institution, are 17.3% for males and 21.2% for females. The differences between these figures and ours are not statistically significant. However, there is an inconsistency in the figures reported by Effiong and his colleagues.[5] Their original article [5] indicated that 2,079 LBW infants were born at UCH, Ibadan, during 1968–72 and the possible aetiological factors were studied among those 688 LBW infants who were born during 1968–70. On the other hand, in a recent article, Effiong [10] has mentioned that these 688 LBW infants were delivered during 1968–72. If this was not a misprint, the incidence of LBW at UCH, Ibadan, would be only 6.6%, which contradicts their

TABLE IV

Immediate Neonatal Deaths According to Birthweights

	Birthweight (g)				*Total*
	< 1,000	*1,001–1,500*	*1,501–2,000*	*2,001–2,500*	
Number of Deaths	9	7	4	1	21
Total Deliveries	13	71	155	589	828
%	69.2	9.9	2.6	0.2	2.5

TABLE V

Comparison of various factors in Low Birthweight and Normal Sized Neonates

Factor	**Frequency		Statistical Significance
	Low Birth-weight	Over 2,500g	
Multiple Pregnancy	22.2	1.8	0.001
Prematurity	12.9	1.2	0.001
Toxaemias	3.6	1.7	0.01
Malpresentations	3.4	2.0	0.05
A.P.H.	2.8	0.9	0.001
Maternal illness	1.8	1.0	* NS
Anaemia	1.4	0.8	* NS
Hypertension	0.7	0.2	* NS
U.T.I.	0.5	0.8	* NS
Cervical Incompetence	0.5	–	* NS
Hydramnios	0.5	0.1	* NS
Induced Labour	0.4	0.1	* NS
Unknown Causes	49.1	–	–

**Percentage of cases in each group.
*Not Significant.
A.P.H. = Antepartum haemorrhage
U.T.I. = Urinary tract infection

former statement. The figures of 9.4% and 9.8% quoted by Oduntan and Ayeni [4] for two MCH centers near Ibadan are significantly lower not only compared to our figures but also to the findings of other investigators from the same area.[6] This marked difference is probably due to the type of population studied by them. Oduntan and Ayeni[4] selected only full-term singleton infants, whose mothers had regularly attended ante-natal clinics, and predictably, their figure was close to the incidence reported in our previous study where we had analysed the birthweights of full-term singleton infants.[8] Osuhor[7] who carried out a study of 1199 infants of different ethnic groups in Malumfashi, which is very close to our unit, also found an incidence of 20%.

There was no obvious associated factor with LBW in about half (49.1%) of our cases, and a majority of these were born after 37 weeks of gestation. After excluding such cases, the most prominent aetiological factors were multiple pregnancies, prematurity and pre-eclamptic toxaemia, contributing 22.2%, 12.9% and 3.6% respectively. Compared with infants who weighed more than 2,500g, the incidences of prematurity, multiple pregnancies and toxaemias were higher in the LBW group and the differences were statistically significant. Jellife,[11] Forfar *et al*,[12] Neutra and Nefe,[13] also observed these factors to be of paramount aetiological value. Among the previous Nigerian studies, Efflong *et al*[5] and Adelusi and Ladipo[14] have enumerated the aetiological factors. The factors observed by these investigators were the same as noted in our study, but the prevalence of various factors was numerically different.

A tendency to produce LBW babies was evident among young mothers. Women aged 19 years and below constituted 42.6% of mothers in the LBW group, whereas only 30.9% women were of that same age range when the baby weighed more than 2,500g. The difference was statistically significant ($P < 0.001$). Simultaneously, there were more primiparae in LBW group (46.8%) as compared to the over 2,500g group (31.8%) and 84% of these primiparae were aged 19years and below.

The maternal age and parity are so closely interrelated that it is difficult to determine the relative effects on the incidence of LBW. Some investigators consider maternal age to be more important [4 15 16] while others have laid more emphasis on parity.[8 17 18] The present study shows that both age and parity significantly influence the incidence of LBW. However, when the incidence of LBW is studied according to various age groups while keeping the parity constant (Fig), it is observed that in almost each parity group, the younger mothers have a higher incidence of LBW as compared to their older counterparts. When judged against the social background of this area, the maternal age, to some extent, can be blamed for a higher incidence of low birth weight. Majority of girls in this area marry at a

young age, at times, even before puberty, so that some of them have their first menstrual period in their marital homes. Consequently, child bearing starts at an age when their reproductive faculties are not fully developed.

The higher incidence of LBW in rural as compared to urban population may be due to accessibility of urban population to better medical care and ante-natal facilities. This is evident from the fact that, as compared to 13% of the rural cases, 38.4% of the urban cases were booked ($P < 0.001$).

The highest percentage of deaths in the present study occurred among infants weighing less than 1,000g and the immediate neonatal death rate declined in each successive higher weight group, exhibiting a strong relationship between LBW, and immediate neonatal mortality ($P < 0.001$). Only one child died out of 589, who weighed between 2,001 and 2,500g. This lends support to Effiong's assumption[10] that African LBW infants are at no risk when their weight is 2,000g or more.

Unfortunately, due to the lack of reliable data regarding maternal height and smoking habits, we could not study the effects of these two important variables in the present series.

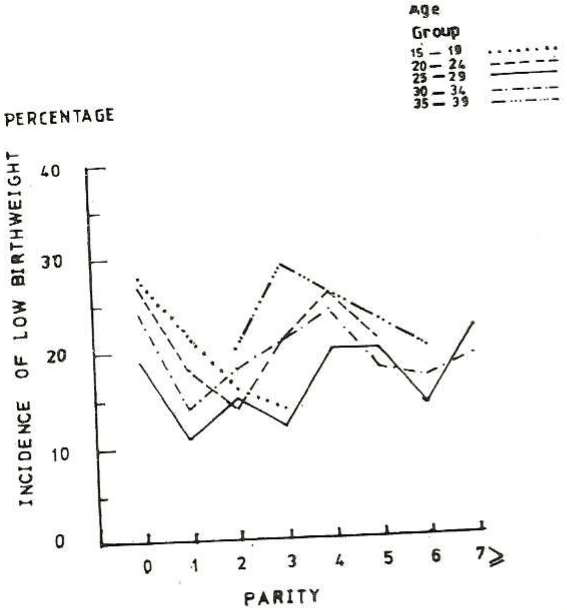

Fig. Effect of age and parity on the incidence of low birthweight

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Congenital Anorectal Anomalies in Western Nigeria

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Summary

Adekunle OO and Johnson AOK. Congenital Anorectal Anomalies in Western Nigeria. *Nigerian Journal of Paediatrics*, **1981; 8:40.** Forty-five patients with congenital anorectal anomalies are presented. The sex ratio is 1:1. The high (supralevator) type anomalies are slightly more common than the low (translevator) anomalies. Associated structural anomalies occurred in only 13% of all the cases. Results of treatment were good in low anomalies, satisfactory in intermediate, but very poor in high anomalies. Late presentation, high default rate after colostomy, and paucity of specialised paediatric units, appear to contribute to poor prognosis. It is suggested that avoidance of colostomy in low anomalies, and definitive treatment during the first admission in intermediate and high anomalies will improve prognosis.

Introduction

After congenital inguinal hernias, congenital anorectal anomalies appear to be the commonest congenital gastrointestinal anomaly in Nigerian children. Anorectal anomalies pose peculiar problems of management in our environment partly because of the socio-cultural beliefs and practices of the population, and partly because of the paucity of specialised centres where corrective surgery for these anomalies can be offered. A previous review of these anomalies at the University College Hospital (UCH), Ibadan, concerned twenty patients.[1] The present communication presents the study of a larger number of patients and highlights the problems of management of the condition. It also suggests a treatment policy which we believe is best suited to the realities of an unsophisticated society.

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Materials and Methods

The case records of all infants with congenital anorectal anomalies admitted into the paediatric wards of the UCH, Ibadan, between 1970 and 1977, were studied. Information collected included age at presentation, sex, nature of the anomalies, associated congenital anomalies, treatment and outcome.

Results

Sex, Age and Incidence

Forty-five patients with congenital anorectal anomalies were seen during the period covered by the study. They consisted of 22 males and 23 females giving a sex ratio of approximately, 1:1. In the same period as the study, there were 9,577 admissions into the paediatric wards and 389 of these were cases of congenital anomalies of the gastrointestinal tract. Thus the 45 cases of congenital anorectal anomalies represent 0.47% of all paediatric admissions and 11.8% of congenital anomalies of the gastrointestinal tract.

Table I shows the age at presentation. Eight patients (18%) were admitted on the day of birth and 26 (60%) presented in hospital by the

TABLE I

Age at Presentation of 45 Children with Congenital Anorectal Anomalies

Number of Patients	26	3	4	2	–	1	–	1	–	–	–	3	1	–	–	1	3
Age at presentation (Weeks)	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	>16

end of the first week. Eight (18%) were aged three months or above before presenting in hospital.

Types of Anomalies

These are summarised in Table II and have been classified according to the International Classification proposed by Stephens and Smith.[2]

The 19 patients with the translevator type of anomaly consisted of 8 males and 11 females. Among the males, there were 2 cases of anal stenosis, 5 of completely covered anus and 1 of anocutaneous fistula. In contrast, there were among the females, 3 cases of anal stenosis, 4 of anocutaneous fistula and 4 of anovestibula fistula. The 7 children with the intermediate type anomaly consisted of 4 females and 3 males, while the supralevator type of anomaly occurred in 11 males and 7 females. The intermediate and supralevator anomalies were associated with recto-urethral and rectovesical fistulae in males and recto-vaginal fistulae in females.

TABLE II

Types of Defects in 45 Children with Congenital Anorectal Anomalies

Type of Anomalies	*No. of Patients*	*% of Total*
Translevator (Low)	19	42
Supralevator (High)	18	40
Intermediate	7	16
*Not Known	1	2
Total	45	100

*Type of defect could not be determined because of inadequate description in the case records.

Associated Anomalies

Six patients (13%) had associated non-anorectal anomalies, and the nature of these anomalies are listed in Table III. It is noteworthy that the two associated anomalies requiring urgent surgical intervention occurred in patients with supralevator anomalies. In addition, three children with supralevator anomaly had associated neonatal jaundice; one of these had erythrocyte glucose-6-phosphate dehydrogenase (G-6-PD) deficiency and Christmas disease, while another had G-6-PD deficiency only.

Treatment and Outcome

The treatment employed in the present series of anorectal anomalies are shown in Table IV. Among the 19 patients with translevator defects, the procedures carried out consisted of anal dilatation in 2 patients, perforation of anal membrane in 3, and anoplasty in 5. The remaining 9 patients had initial colostomy and this was

TABLE III

Associated Abnormalities in 45 Patients with Anorectal Anomalies

Type of Anorectal Anomaly	*Type of Associated Anomaly*	*No. of Patients*
Supralevator (High)	Intestinal malrotation	1
	Talipes with Omphalocele	1
Translevator (Low)	Hypospadias	1
	Polydactyly	1
	Hemivertebrae, Rib defects and Undescended Testis	1
Intermediate	Atrophy of radial border of left hand	1
Total		6

followed by anoplasty in 3, abdomino-anal pull-through in 2 and perforation of anal membrane in one. All these 16 patients also had subsequent regular anal dilatation post-operatively and achieved faecal continence. Of the remaining 3 patients, one died soon after colostomy and 2 were lost to follow-up before corrective surgery could be performed. Thus 84% of children with translevator anomalies had good results after correction of the anomalies.

All the 7 patients with intermediate type anomaly had initial colostomy. This was followed by abdominoanal pull-through in 4 patients, and anoplasty in one. The remaining 2 patients defaulted after colostomy. One of the 4 patients who underwent abdominoanal pull-through died, while one developed faecal incontinence. The remaining 2 and the child who underwent anoplasty had good results giving a success rate of 43%.

TABLE IV

Treatment in 45 Patients with Congenital Anorectal Anomalies

Treatment	*Type† of Anomaly*			
	T	*I*	*S*	*U*
Dilatation only	2	–	–	–
Perforation of Anal Membrane and Dilatation	3	–	–	–
Anoplasty	5	–	–	–
Colostomy only	3	2	13	1
Colostomy and Anoplasty	3	1	–	–
Colostomy and Abdomino-Anal Pull Through	2	4	3	–
Colostomy and Perforation of Anal Membrane	1	–	–	–
No Treatment	–	–	2	–
Total	19	7	18	1

† *Types:*
T — Translevator
I — Intermediate
S — Supralevator
U — Unknown

Of the 18 patients with supralevator type of anomaly, 2 died before any form of surgical treatment could be offered, while 16 had colostomies initially. Following the colostomies, 2 more patients died while 11 defaulted from the clinic, and only 3 returned to have abdominoanal pull-through operations. Of these 3, one had good anal sphincteric action but the remaining two developed incontinence of faeces. Thus, the success rate for treatment of the supralevator anomalies was only 6%.

Discussion

The reported incidence of congenital anorectal anomalies is one in every 5,000 births and rarely is there any family history.[3] Because of the diverse nature of these anomalies, there has been some confusion in the classification, but the one proposed by Stephens and Smith[2] is now widely used. This classification, which recognises three major subdivisions of anomalies, is based on anatomical rather than embryological considerations. It consists of supralevator (high), translevator (low) and intermediate anomalies. The translevator anomalies are usually easy to treat and have good prognosis because the sphincteric mechanism is intact. On the other hand, the intermediate and supralevator anomalies are difficult to treat and almost always require initial colostomies, a social stigma in the Nigerian society. In addition, because the sphincteric mechanism is commonly deficient in these types of anorectal anomalies, the outcome after definitive surgery is often unsatisfactory.

The diagnosis of an anorectal anomaly is usually easy because of the absence of an external anal opening or the presence of an ectopic one, and hence most patients present at birth or soon after. Some patients with wide ectopic external openings which were initially adequate for bowel evacuation may present late because the opening may subsequently become inadequate as the stools become firmer. Constipation with recurrent straining prompts the mothers to seek medical attention in such cases.

The determination of the variety of anomaly is not always straightforward. Examination of the perineum will reveal most cases of translevator anomalies either as a bulge or as an ectopic anus in the perineum or as a pinhole anal opening. In the supralevator variety, the perineum is blind but meconium may be seen coming from an opening high on the vaginal wall in females, while gas bubbles or greenish coloured urine may be passed in the males. The classical Wangesteen and Rice[4] method of plain radiography of the child in the inverted position measures the distance between the position of the anal dimple and the termination of rectal/anal air translucency. However, the presence of some tenacious meconium plug at the distal end of the rectum undisplaced by air may increase this distance and give a false impression of the level and vareity of the anorectal anomaly. Lateral invertogram is therefore often more helpful.[2] In this view, the pubococcygeal line is just above the level of the puborectalis sling which is where supralevator anomalies end. More precise assessment is also possible by means of a fistulogram through the ectopic external opening or barium examination through the distal colostomy loop.

Supralevator and translevator anomalies were equally common in the present series which is in keeping with the observation by Bankole.[1] In contrast, McPherson[5] reported that supralevator varieties were more common while Partridge and Gough[6] found that the translevator anomalies occurred more commonly.

The International Classification of Stephens and Smith[2] recognises two major types of supralevator anomalies. These are anorectal agenesis and rectal atresia. The former is the commoner and usually forms a fistula with the urogenital tract in both sexes. Rectal atresia is more difficult to diagnose because the anus appears normal and there is no associated fistula. In the present series there were 16 cases of anorectal agenesis of which 9 were females and 7 were males. All the females had high recto-vaginal fistulae while 5 males had rectovesical and 2 recto-urethral fistulae. Rectal atresia occurred in 2 patients, one male and one female.

Two groups of patients with translevator anomaly are readily identifiable: those with an abnormal opening at the normal site, for example, anal stenosis and covered anus (complete), and those with an ectopic opening. In males, the ectopic opening will be found on the perineum while in the females, the opening may be in the perineum or at the vestibule. While anal stenosis and covered anus formed the bulk of the translevator varieties in males, vestibular ectopic anus was commoner in the females. The intermediate type of anorectal anomalies is the least common in this series and most of the cases are females with low rectovaginal fistulae.

The reported incidence of associated congenital defects in anorectal anomalies varies between 25% and 80%.[7–9] Only 13% of our patients had associated structural anomalies, and this figure rises to 18% if the 2 patients with erythrocyte G-6-PD deficiency are included. However, it seems likely that the true figure would have been higher if the default rate after colostomy had been less and if all the children had been more extensively investigated.

Most infants with anorectal anomalies require some form of immediate treatment. Anal stenosis needs only regular dilatation, while covered anus requires perforation of the membrane followed by regular dilatation. Other translevator anomalies with ectopic opening will require some form of anoplasty. Most cases with intermediate and all those with supralevator anomalies usually require a temporary transverse colostomy to allow time for detailed analysis of the situation, and in some patients, for immediate relief of acute intestinal obstruction. One stage definitive procedure in supralevator and intermediate anomalies have been recommended by some authors.[5] However, the view is widely held that definitive treatment should be delayed to between 6 and 12 months as earlier treatment may damage the puborectalis sling.[3] The effect of the age at definitive surgery on the outcome of management could not be

assessed in our patients because of the high default rate after colostomy.

In the present series as well as in others, the result of treatment of translevator anomalies was excellent. Sixteen of the 19 patients were continent of faeces after operation. There was one death, 5 days after colostomy. In the intermediate variety, the results were satisfactory if the two cases who defaulted after initial colostomy are excluded, leaving 3 of 5 patients completely cured. The results of the supralevator type of anomaly were the poorest. Anal continence was satisfactory in only one of the 3 patients who had pull-through operations and there were 4 hospital deaths. The poor results of treatment in supralevator anomalies in this series is in keeping with the experience from other centres.[5 6 10]

There was a high default rate after colostomy. There were 33 colostomies of which 3 died immediately post colostomy, 15 defaulted (default rate, 45%) while the rest had definitive treatment. Default after colostomy is perhaps not unexpected because of the social stigmata generally attached to this procedure in our environment. For example, a recent review of carcinoma of the rectum in Ibadan revealed that 30% of patients refused abdomino-perineal resection because colostomy was socially unacceptable (unpublished data). Majority of cases who failed to return after initial colostomy may have died either from natural causes, from associated anomalies, from intercurrent diseases or from neglect by embarrassed and superstitious parents. In this context therefore, the role of the Medical Social Worker and the Health Visitor in giving the parents of children with colostomies much needed support and guidance is of paramount importance. It may also be worthwhile to consider one-stage definitive operations in which the colostomy if any, would be closed before the child is discharged from the hospital. Major constraints against this include late presentation in many instances resulting in an acutely ill child, limited anaesthetic facilities and specialised paediatric nursing care. All these make a major operation hazardous. Besides, the great demand for the few paediatric beds available for other acutely ill children makes a long hospital stay sometimes impracticable.

Despite these reservations and in view of the immense social stigma attached to a colostomy, it is suggested that colostomy should be avoided in cases of low anomalies and definitive treatment carried out at the time of first presentation. Definitive surgery on children with high anomalies and without severe associated anomalies should not be delayed beyond the first admission. It is hoped that these measures coupled with the support and encouragment of Medical Social Workers and Health Visitors will considerably reduce the default rate and improve morbidity and mortality associated with the treatment of congenital anorectal anomalies in a developing country like Nigeria.

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Experience with the Management of Exomphalos and Gastroschisis in Ibadan

AA ADEYOKUNNU* AND NA AKINGBEHIN**

Summary

Adeyokunnu AA and Akingbehin NA. Experience with the Management of Exomphalos and Gastroschisis in Ibadan. *Nigerian Journal of Paediatrics* **1981; 8: 45.** Ninety-eight cases of exomphalos and gastroschisis were managed in the Department of Paediatrics, University College Hospital, Ibadan, over a period of eight years (1970–1978). Fifty-two (53%) of the cases were given non-operative while forty-six (47%) had surgical management. Mortality was 13% in those treated non-operatively and 59% in those treated surgically. The advantages and disadvantages of both forms of management are discussed and factors contributing to prognosis analysed. Operative management seems ideal when exomphalos occurs in an infant who is full term and of good weight, when the defect is minor and the sac is healthy, and when the scout films of the chest and abdomen reveal no significant abnormality, provided a good team of anaesthetists and surgeons is available for expedient surgical correction. For the fragile premature infant or any infant with major defect and infected defects, the use of non-operative technique under antibiotic cover as in-patients seems to offer the best course of action.

Introduction

EXOMPHALOS is herniation of some intra-abdominal contents through an open umbilical ring. The resulting protrusion is covered by a translucent, avascular membrane which consists of a fusion of peritoneum, amniotic membrane, and Wharton's jelly. Exomphalos differs from umbilical hernia in that it has no skin covering except at its base. The sac of an exomphalos may rupture before or soon after birth and he condition may therefore be confused with gasttroschisis. In gastroschisis, evisceration of the abdominal contents occurs through a full thickness defect of the abdominal wall at a point other than the umbilicus, and the insertion of the umbilical cord into the abdomen remains in the normal position.[1]

Exomphalos is not a common anomaly. Various reports[2 6] put the incidence at between 1 in 5,000 and 1 in 10,000 births. In Nigeria, an incidence of about 1 in 1,400 births has been reported from one hospital.[7]

The embryologic basis of exomphalos has been the subject of intense research and reviews.[8 12] The most widely accepted view is that a small exomphalos is due to failure of the umbilical ring to contract thereby permitting a herniation into

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the umbilical cord while large exomphaloses are regarded as morphogenic failures of the four folds of the body of the embryo to converge properly.[12 13] The reasons for the failures that produce exomphaloses are unknown. There have been isolated reports of occurrence in siblings and twins, and chromosomal abnormalities have also been found in some cases[14 16] but on the whole, hereditary factors do not seem to play any key role in the causation of the condition. On the other hand, the frequent association of exomphalos with a wide range of other malformations including those of the jaws, tongue and gastrointestinal tract,[5] and genitourinary tract,[4] suggest a general interference with embryonic development at an early stage of intrauterine life.

The best method for the management of exomphalos remains controversial. In the past, the mode of treatment for all cases was surgical correction, but this was accompanied by a high mortality rate.[18 19] More recently, it has been shown that a significant reduction in the mortality rate could be achieved with non-operative method in cases of exomphalos that have not ruptured.[5 20 21] The present communication reports our experience with the management of 98 cases (96 exomphalos and 2 gastroschisis) seen over an 8-year period in the Children's Department, University College Hospital (UCH), Ibadan.

Patients and Methods

All the children in whom the diagnosis of exomphalos or gastroschisis were made on clinical grounds between 1970 and 1978 were studied. The age, sex, weight and maturity of each patient were recorded, and the interval between birth and the time of reporting in the hospital noted. The size of the defect, associated malformations as well as the results of radiological, bacteriological and biochemical investigations carried out on any of the patients, were noted. The mode and the outcome of management in each case were also studied.

The anomalies were divided on the basis of their sizes into three categories namely: minor, major and gastroschisis. Irrespective of whether the sac was intact, infected or ruptured, an exomphalos was classified as minor if the diameter of the fascial defect was less than 5cm (Fig 1).

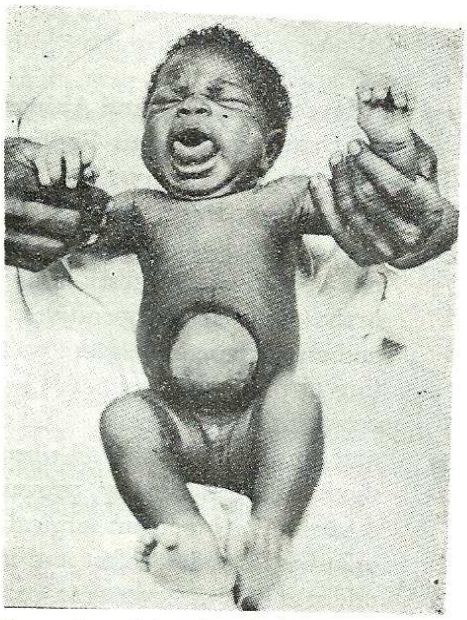

Fig. 1. Exomphalos minor, the diameter of the fascial defect is less than 5cm

Where the diameter of the fascial defect was greater than 5cm, the exomphalos was classified as major (Fig. 2), and cases in which a large portion of the gut had eviscerated were classified as gastroschisis.

Management was either surgical or conservative (non-surgical). The non-surgical management involved painting of the defect with aqueous solution of 2% mercurochrome. Our choice of the method of management was determined by the size and state of the defect, the state and maturity of the patient, and the presence and severity of associated malformations. Fifty-two of the 98 cases were treated conservatively while 46 were treated surgically.

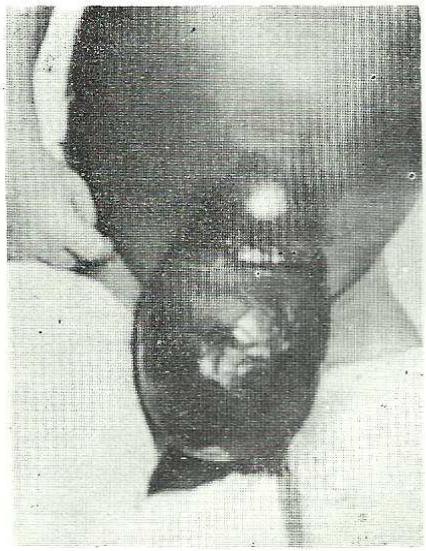

Fig. 2 Exomphalos major with diameter of fascial defect greater than 5cm. Both legs in plaster slabs to restrain the thighs rubbing on the defect.

Results

Sex, Birthweight and Age at Presentation

The 98 patients consisted of 63 males and 35 females (male to female ratio of 1.8:1). The birthweights of the infants are summarized in Table I. Thirty-six (37%) were premature by weight, their birthweights varying between 1.0 and 2.5 kg. Forty-eight (49%) weighed between 2.6 and 3.5 kg; and 14 others (14%) weighed 3.6kg and above.In comparison with the average birthweight of Nigerian babies[22 23] this last group of babies were significantly big.

TABLE I

Birthweights and Sex Distribution of Patients

Wt. (kg.)	*No. of Cases*		*Total*	*Percentage of Total*
	Male	*Female*		
1.0–2.5	23	13	36	37
2.6–3.5	31	17	48	49
3.6 and above	9	5	14	14
Total	63	35	98	100

The interval between birth and presentation was within 24 hours of delivery in 37 cases, between one and three days in 39 cases, between three and seven days in 18 cases and over one week in 4 cases.

Types of Defect and Associated Anomalies

A minor intact exomphalos was present in 24 (24%) and a major defect in 72 (73.5%) cases. The sac had ruptured in 15 of the major cases. Six infants were intially diagnosed clinically as gastroschises but only 2 were confirmed to be so at operation, the remaining 4 being examples of ruptured exomphalos major. Fifty-seven patients (58%) had associated anomalies (Table II); these were present in all organ systems but were commonest in the gastrointestinal tract.

Laboratory and Radiological Findings

Bacteriological cultures of exomphalos swabs and of blood were performed in 41 patients. These included all the 22 cases reporting after 72 hours of delivery, and 19 of the 39 cases who sought medical attention between 24 and 72 hours post-delivery. Positive cultures were obtained in 24 cases and the isolated organisms included *E. Coli* (9 cases; 7 from swabs and 2 from blood), *Pseudomonas* (7 cases; all from swabs), *Klebsiella* (5 cases; 3 from swabs and 2 from blood), *Proteus* (2 cases; both from swabs) and *Staph. aureus* (1 case; from blood).

Blood sugar was estimated in 27 cases including the 14 babies who weighed more than 3.6kg on admission. Blood sugar levels of less than 1.9 mmol/l (35 mg%) were obtained in 13 cases, all of whom also had somatic gigantism and macroglossia which are classic features of the Beckwith-Wiedemann syndrome, (Fig. 3). In 6 cases, the blood sugar level was less than 1.1mmol/l (20mg%) and this necessitated vigorous treatment for hypoglycaemia.

Fifty-six cases had radiographs of the abdomen and chest, and 32 of these showed evidence of either pneumonitis or consolidation of one or

both lungs. Fluid levels indicating intestinal obstruction occurred in 8 cases, while situs inversus (Fig. 4), absence of sternum, and hemivertebrae occurred in one case each.

TABLE II

Associated Malformations in 57 Cases of Exomphalos/Gastroschisis

Malformation	*No. of Cases*
FACE and TONGUE	
Macroglossia	13
Cleft lip and palate	5
GASTROINTESTINAL TRACT	
Malrotation of the gut	16
Meckel's diverticulum	6
Ileal atresia	3
Jejunal atresia	2
Persistent omphalo-mesenteric duct	1
Imperforate anus	7
Biliary atresia	2
GENITOURINARY SYSTEM	
Ectopic kidney	3
Duplex kidney	1
Bifid ureter	1
Ectopia vesicae	3
LIMBS	
Talipes equinovarus	8
CARDIOVASCULAR	8
CENTRAL NERVOUS SYSTEM	
Cataract	1
Microcephaly	1
Hydrocephalus	3
Meningomyelocele	1
VERTEBRAL	1
ABSENT STERNUM	1
CHROMOSOMAL DEFECTS	
Down's Syndrome	3
Trisomy 'E'	2
Total	92

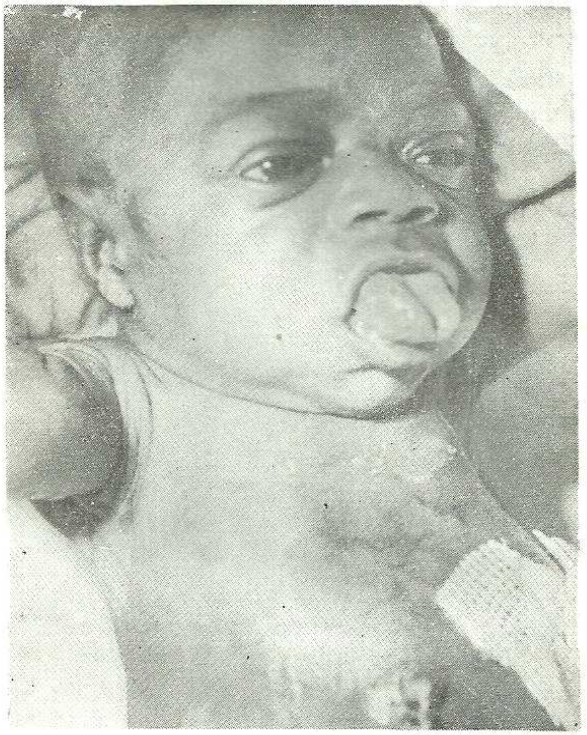

Fig. 3 Female infant with repaired exomphalos, macroglossia somatic gigantism and hypoglycaemia (example of Weidemnann-Beckwith Syndrome)

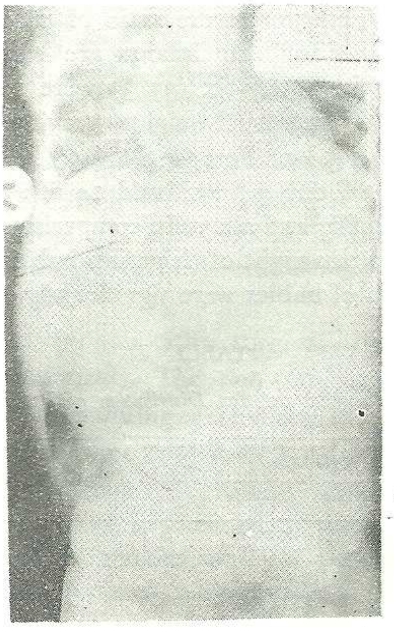

Fig 4 X-ray of chest and abdomen of a patient with exomphalos showing situs inversus and right-sided pneumonitis

Management and Outcome

Thirty-three out of the 57 cases with intact exomphalos major received non-operative treatment, and five of these died, (a mortality rate of 15%). The remaining 24 cases underwent surgery and 11 of these survived while 13 died (mortality rate of 54%). Among the 15 cases with ruptured sacs, two were treated conservatively because of gross infection and one of these survived and the other died. The remaining 13 cases underwent surgery and 11 of these died (a mortality of 84.6%). Seventeen of the 24 cases of exomphalos minor were operated upon and 2 died, a mortality of 28.5%. The two cases of gastroschisis were managed surgically, one of them died while the other survived.

The mortality in relation to the age and weight of the patients at presentation are summarized in Tables III and IV.

TABLE III

Mortality in Relation to Birthweights in 98 Cases of Exomphalos/ Gastroschisis

Wt. (kg.)	*No. of Cases*	*No. Alive*	*% of Cases*	*No. Dead*	*% of Cases*
1.0–2.5	36	19	52.8	17	47.2
2.6–3.5	48	33	68.8	15	31.2
3.6 and above	14	12	85.7	2	14.3
Total	98	64	65.3	34	34.7

TABLE IV

Mortality in Relation to Length of Delay before Hospitalization

	Length of Delay	*No. of Cases*	*No. Dead*	*% Mortality*
(*a*)	Less than 1 day	37	9	24.3
(*b*)	1 – 3 days	39	15	38.5
(*c*)	3 – 7 days	18	8	44.4
(*d*)	Over 1 week	4	2	50.0

Discussion

The present study showing a preponderance of males over females, a low birth weight in 37%, and exomphalos major in 73.5% of the cases, compares favourably with the findings of Jacho,[2] Aitken,[15] and Firor.[25] The presence of associated anomalies in 58% of our cases also compares with the incidence of 38% recorded by Hutchin,[1] and 44% reported by Smith and Leix.[26] This high incidence of associated anomalies is not surprising because all the organ systems involved, namely: the heart, the central nervous system, the renal tract and the gastrointestinal tract share the same embryogenesis.

The best method of management of exomphalos is controversial. Operative management enables the surgeon to explore the abdominal contents and if need be, correct any associated gastrointestinal tract anomalies. Compared with non-operative management, the period of hospitalization for surgical management is also short; thus one to four weeks was the average length of hospitalization for our surgically managed patients whereas the conservatively managed cases required eight to twelve weeks of hospitalization. Surgical correction was therefore practised in all cases until it was realized that non-operative management of exomphalos major was associated with reduced mortality.[4 19]

The conservative method of management of exomphalos was reportedly introduced in 1957 by Grob.[27] The method involves the painting of the exomphalos sac with 2% aqueous solution of mercurochrome. This dries the sac to a thick eschar under which granulation tissues form, followed by gradual epithelization and cicatrix formation. Good results have followed this regimen[4 19 20 28] but the disadvantages include, prolonged hospitalization and the attendant demand on the nursing services, the occasional delays in diagnosing intestinal anomalies, the risk of peritonitis during the early phase of resolution of the sac, and the uncommon but distinct possibility of mercury poisoning from absorption of mercurochrome.

The mortality rates from operative and non-operative methods of management in the present study were fifty-nine and thirteen % respectively. The disadvantages of operative management were most glaring in exomphalos major. Sixty-five % of patients with exomphalos major who underwent surgery died; whereas only 17 % of those managed conservatively, died. Surgical management of exomphalos major presents considerable technical problems. The small abdominal cavity may not be large enough to contain all the eviscerated structures, and therefore, reduction is often only accomplished under tension. This causes increased intra-abdominal pressure and respiratory embarrassment and predisposes to other complications such as intestinal kinking and obstruction, pressure necrosis of the intestine, and compression the inferior vena cava with the attendant reduction in the venous return to the heart. Surgeons of are aware of these complications and therefore do not always attempt a primary closure of big defects but sometimes resort to manipulative techniques such as multi-stage repair of the defect, and the use of Silastic Teflon Sheath. Massive adhesion formations however constitute a potential hazard of these alternative operative procedures.

Survival following surgery appears also to be partly related to the quality of available surgical services. For example, among the last 29 cases in the present series, only one death occurred out of the 13 cases surgically managed by a paediatric surgeon whereas 4 out of 5 babies in the same group who had their surgical correction performed by general surgeons, died.

The overall mortality rate of 34·7% in the present series compares with an average mortality rate of 50% in eleven centres reviewed by Hutchin.[1] Contributory factors to mortality in all series have included prematurity, associated congenital malformations, the size of the sac, and rupture of the exomphalos sac. Bacterial infections and delay in the institution of management constitute additional adverse factors in the present series. These two problems did not feature prominently in the series from developed countries, and are causally related to poor environmental sanitation and scarcity of the appropriate medical facilities prevailing in underdeveloped countries.

Encouraging as the non-operative management is, the length and cost of hospitalization make it unsuitable for poor countries. In a recent paper, Olowe[21] suggested that this major disadvantage of conservative management might be overcome if patients are treated on outpatient basis. In the light of this suggestion, we ventured outpatient management in some of our patients after 3 weeks of hospitalization, but were soon obliged to readmit them for further periods of 8 to 12 weeks each, because of infections of the exomphaloses at home. Outpatient management can therefore not be regarded as the best way for reducing cost and alleviating the disadvantages of prolonged hospital stay in these patients.

From the foregoing, it would seem that operative management of exomphalos is ideal in an infant who is full term and of good weight, whose defect is minor and the sac is healthy, and in whom the scout films of the chest and abdomen reveal no significant abnormality, provided a good team of anaesthetist and surgeon is available for expedient surgical correction. For the fragile premature infant or any infant with a major defect and those whose defects are infected, the use of non-operative technique under antibiotic cover as inpatients seem to offer the best course of action.

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Levels of Calcium, Phosphorus, Alkaline Phosphatase, Bilirubin and Glucose in Newborn Infants and their Mothers

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Summary

Abdurrahman MB, Raza MK, Ajayi VA, Zakolshi W and Ame J. Levels of Calcium, Phosphorus, Alkaline Phosphatase, Bilirubin and Glucose in Newborn Infants and their Mothers. *Nigerian Journal of Paediatrics* **1981; 8:52.** Calcium, phosphorus, alkaline phosphatase and bilirubin levels were measured in cord blood from full-term neonates and in venous blood from their respective mothers. Capillary blood glucose was measured in the neonates. The mean values in the neonates were calcium 2.56 mmol/l (10.24 mg/dl), phosphorus 1.64 mmol/l (5.13 mg/dl), alkaline phosphatase 127iu/l, and glucose 33.4 mg/dl; the median for phosphorus was 1.68 mmol/l (5.16 mg/dl). Levels of calcium and phosphorus were significantly higher in the neonates than in the mothers, but there was no difference in the mean level of alkaline phosphatase. There was significant correlation between cord serum levels of calcium, phosphorus and alkaline phosphatase and the respective maternal serum levels. There was no correlation between cord serum calcium and phosphorus. The levels of blood glucose in the babies were low, with a range of 20–50 mg/dl and a mean of 33.4 mg/dl. 29 out of 86 babies had blood glucose less than 30 mg/dl, but none of these babies was symptomatic.

Introduction

BIOCHEMICAL disorders commonly seen in a newborn nursery include hypocalcaemia,[1 2] hypoglycaemia[3 4] and hyperbilirubinaemia.[5 6] These disorders can give rise to immediate and long term complications, the most serious of which is brain damage.[7 8] Knowledge of the normal range of values of these biochemicals is therefore a prerequisite in the prevention and proper management of their disorders.

The purpose of the present study was to determine the normal serum levels of calcium, phosphorus, alkaline phosphatase, bilirubin and glucose in full-term babies, and to define the relationships, if any, between the serum levels of these substances in the babies and their mothers.

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Materials and Methods

Healthy, full-term, normally delivered babies with normal birthweights and their respective mothers were selected for the study. An additional criterion was that the mothers had no problems during pregnancy, and showed no overt signs of malnutrition. Cord and maternal blood were collected at the time of delivery, and serum was extracted from each blood sample by centrifugation. If estimation could not be done on the sera on the day they were collected, the samples were stored in a deep freeze and the estimation done within a week.

Blood for glucose was separately obtained from the babies by heel-pricks, and the glucose level measured by 'Dextrostix'* and 'Eyetone'* Dextrose meter. Accuracy of the Dextrose meter was assessed earlier by comparing the values of glucose obtained by the standard glucose oxidase method[9] with those obtained with the Dextrose meter. Using blood of varying glucose concentrations, a linear relationship with a correlation coefficient of 0.95 was obtained.

Serum calcium was determined by the cresolphthalein complexone method of Baginski, *et al.*[10] Serum phosphorus was determined colorimetrically.[11] Alkaline phosphatase was measured by the method of Kind and King,[12] and serum bilirubin by the photoelectric method of Malloy and Evelyn.[13]

Results

There were 125 babies (68 males and 57 females), with mean birthweight of 3.2 kg, and 123 mothers, with a mean age of 23.2 years and mean parity of 2.9. Blood was not collected from two mothers and these were therefore not included in the analysis. Blood glucose was determined in only 86 babies.

*Ames Company

The distribution of blood glucose levels in the 86 babies is shown in Figure I. The blood glucose levels in the babies were generally low, ranging between 20 and 50 mg/dl (mean = 33.4 mg/dl). Twenty-nine (34%) babies had blood glucose less than 30 mg/dl. The mean glucose level of 35.9 mg/dl in female babies was significantly higher than that of 31.6 mg/dl in male babies ($t = 2.329$; $P < 005$). The mean cord calcium concentration of 2.56 mmol/l (10.24 mg/dl) was significantly higher than that of 2.39 mmol/l (9.56 mg/dl) in the mother ($P < 0.001$) (Table I). Sixty-three (50%) of the 125 babies had serum calcium level of between 2.5 and 2.9 mmol/l (Fig. 2). Although the mean cord serum calcium was high, 12 babies had levels less than 2 mmol/l, but none of them had signs of hypocalcaemia. The mean serum phosphorus level in the newborn babies was 1.64 mmol/l compared with 1.24 mmol/l in their mothers. The difference is statistically significant ($P < 0.001$). The medium cord serum phosphorus was 1.68 mmol/l. The distribution of the serum levels in the 125 babies is presented in Fig. 3. There was a wide scatter in the serum levels of alkaline phosphatase in both the babies (Fig. 4) and their mothers, but there was no difference in the mean values. The mean concentration of alkaline phosphatase in cord and maternal sera was higher than the local normal range of 35–70 iu/l in non-pregnant adults.

There was no significant sex difference in the mean levels of cord serum calcium, phosphorus and alkaline phosphatase.

In our laboratory, the normal serum bilirubin in adults is 3.4–20 umol/l, while 50 umol/l is arbitrarily used as the upper limit of normal total serum bilirubin in the newborn. Nineteen mothers had total serum bilirubin level greater than 20 umol/l, and 7 babies had levels greater than 50 (Fig. 5). Four out of the 7 babies had unconjugated bilirubin greater than 50 umol/l. The median cord serum unconjugated bilirubin was 20.5 umol/l. None of the babies or mothers developed jaundice during their stay in the hospital.

TABLE I

Biochemical Values in Newborn babies and their mothers

Substance	*Source*	*Range*	*Mean*	*SD*	*Confidence Interval*	*P*
Calcium (mmol/l)	Cord (n= 125)	1.5–4.0	2.56	0.50	1.58–3.54	<0.001
	Mothers (n= 123)	1.4–3.7	2.36	0.47		
Phosphorus (mmol/l)	Cord (n= 125)	1.0–3.0	1.64	0.42	0.82–2.46	<0.001
	Mothers (n= 123)	0.7–2.1	1.24	0.29		
Alkaline Phosphatase (iu/l)	Cord (n= 125)	25–420	127	86	42–296	<0.5
	Mothers (n= 123)	25–333	126	71		
Glucose (mg/dl)	Baby (n= 86)	20–50	33.4	9.0	15.8–51	

n = number of subjects
SD = Standard deviation
p = level of statistical significance between mean cord and maternal levels.

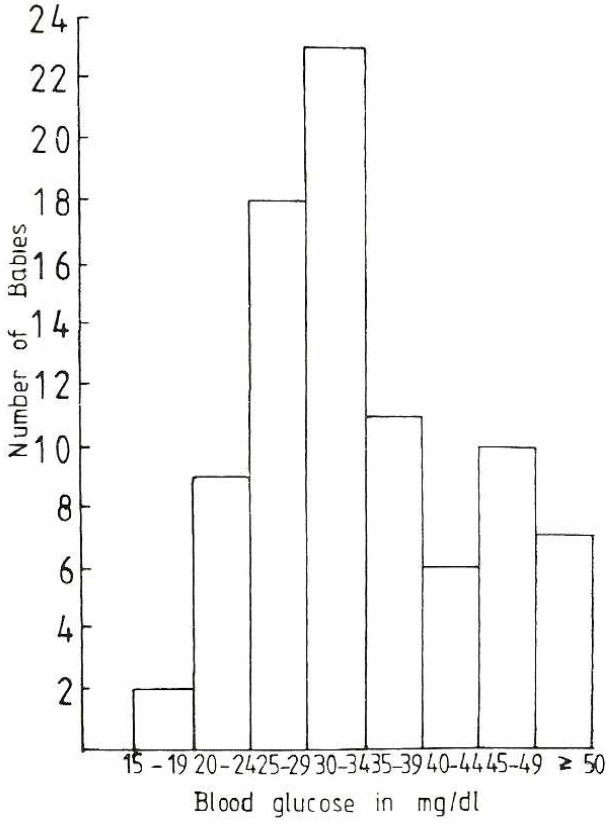

Fig. 1 Distribution of blood glucose in 86 newborn babies

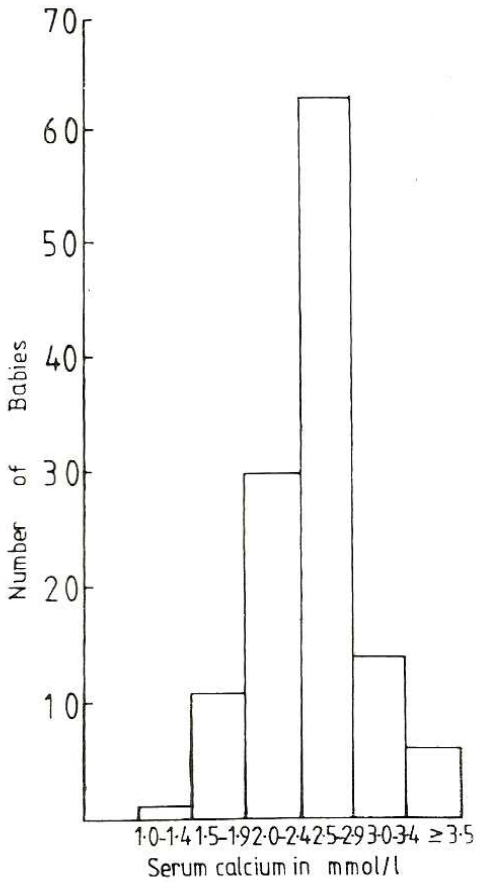

Fig. 2 Distribution of cord serum calcium in 125 newborn infants

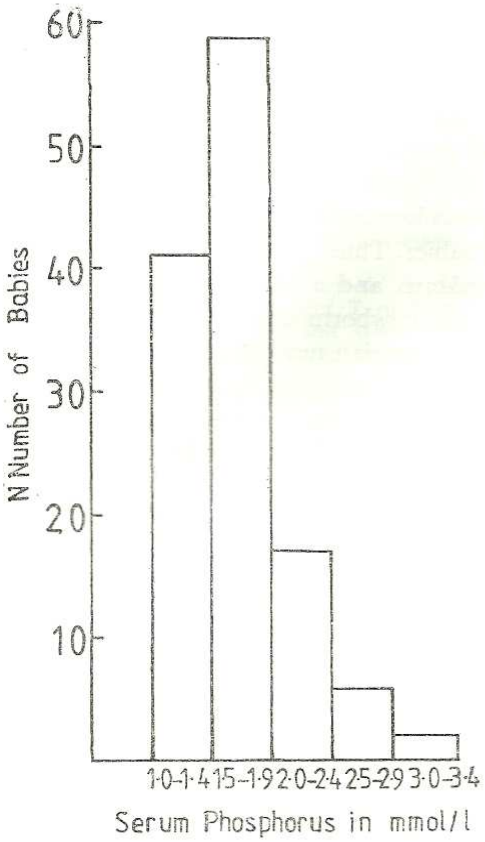

Fig. 3 Distribution of cord serum phosphorus in 125 newborn babies.

The distribution of glucose, calcium and alkaline phosphatase fell into the so-called 'normal" (or Gaussian) pattern (Figs. 1,2,4), whereas the distributions of phosphorus and bilirubin were skewed to the left (Figs. 3,5). There was a significant correlation between cord serum levels of calcium, phosphorus and alkaline phosphatase and the respective maternal serum values (Table II). There was no correlation between cord serum calcium and phosphorus levels ($r = 0.94, P > 0.1$).

TABLE II

Relationship between maternal and cord biochemical values

Substances	*Coefficent of correlation*	*t*	*p*
Calcium	0.499	6.332	<0.001
Phosphorus	0.277	3.168	<0.01
Alkaline Phosphatase	0.375	4.447	<0.001

Discussion

The mean serum calcium level of 2.56 mmol/I (10.24 mg/dl) in babies in the present study is on the high side of the normal range of 9–11 mg/dl in healthy adult Nigerians.[14] This relative hypercalcaemia in the newborn has also been reported by Todd, Chuinard and Wood[15] and by David and Anast.[16] The high serum calcium in the neonates is unlikely to be due to hyperactivity of foetal parathyroid glands because of the low or undetectable immunoreactive parathyroid hormone in cord sera,[16] but is likely to be due to a high foetal-to-maternal calcium gradient by means of a specific calcium transport system.[17]

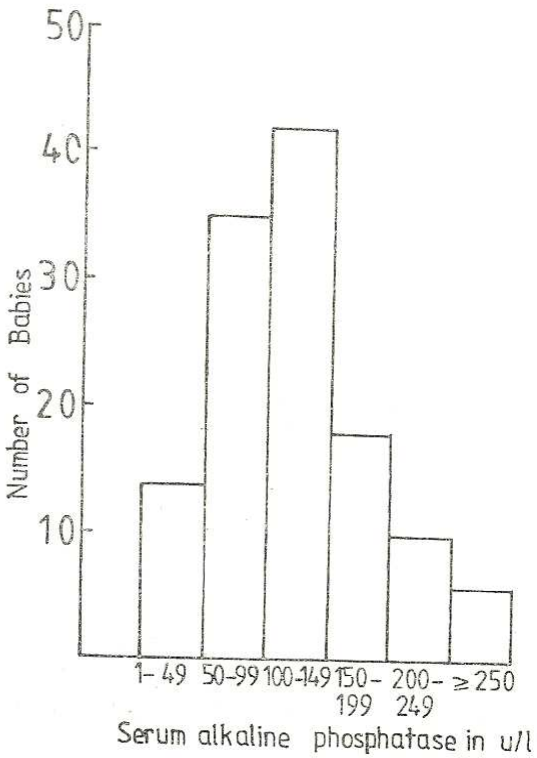

Fig. 4 Distribution of cord serum alkaline phosphatase in 125 babies.

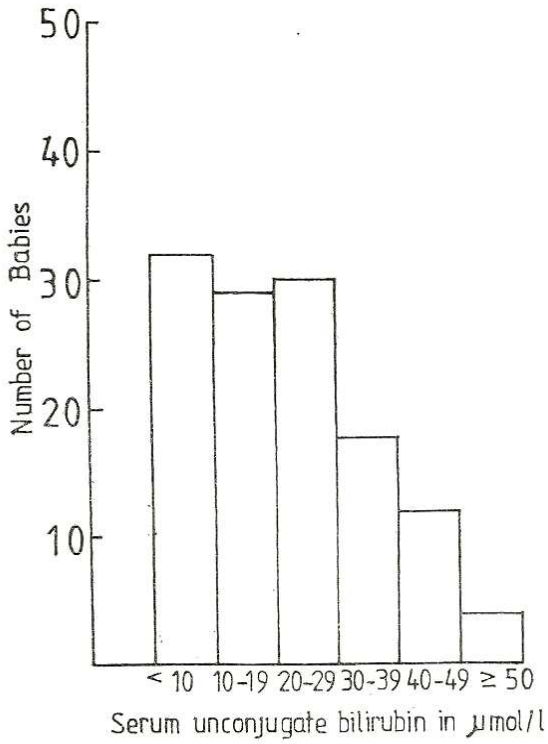

Fig. 5 Distribution of unconjugated bilirubin in cord serum from 125 babies.

The mean cord serum phosphorus of 1.64 mmol/l (5.13 mg/dl) is higher than the normal adult level of 2–4 mg/dl.[14] The high cord serum phosphorus and the lack of correlation between serum calcium and phosphorus found in this study are similar to the findings of Todd, Chuinard and Wood[15] and of David and Anast.[16] Without isoenzyme studies, it was not possible to determine how much of the alkaline phosphatase in the cord serum was of placental origin.

If blood glucose of less than 30 mg/dl is used to define hypoglycaemia,[3] one third of the babies in the present study would be considered to have asymptomatic hypoglycaemia. Using the same criterion, 20 (16.6%) out of 120 term babies reported by Effiong and Ojo[18] would also be considered hypoglycaemic. It seems unlikely that such a high percentage of full-term and adequate for gestational age babies could have abnormal blood glucose levels. Rather, it seems more likely that the normal blood glucose level in Nigerian babies is lower than the level reported in caucasian babies. Thus, whereas the values of calcium, phosphorus and alkaline phosphatase found in Nigerian newborns are similar to those reported in the caucasian newborns, the blood glucose in the Nigerian neonate seems lower than in the caucasian neonate.

The present study has also revealed significant differences between newborns and adults in respect of the biochemical contents of their sera. Therefore, adult biochemical values should not be used as the criteria for diagnosing biochemical disturbances in the newborn, in order to avoid unnecessary or delayed intervention.

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The Danger of Using Methylated Spirit to Treat the Umbilical Cord in Newborn Infants

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Summary

Olowe SA, Ahmed I and Egri-Okwaji MTC. The danger of using Methylated Spirit to treat the Umbilical Cord in Newborn Infants. *Nigerian Journal of Paediatrics* **1981; 8:58.** Four neonates sustained severe burns from accidental ignition of the methylated spirit which was being used to dress their umbilical cords. One of them died. The continued use of alcohol for cord dressing is questioned since it has not been proven to sterilize the cord or prevent bacterial colonization of the baby. Triple dye is suggested as a cheap, effective and safe alternative.

Introduction

Despite all the advances in the knowledge of immunization and public health, neonatal tetanus continues to be a major cause of perinatal mortality and morbidity in developing countries.[1] In the vast majority of cases, the infection is acquired through direct contamination of the umbilical cord, either at the time of cutting it at birth or through infected dressing before it separates and falls off. Prevention of umbilical sepsis therefore remains a major concern of paediatricians and nurses working in these areas. One of the preventive methods often practised in hospitals and maternity homes (and usually recommended to mothers caring for their babies at home) is dressing of the cord with methylated spirit.[2] In this article, we report four cases of severe burns suffered by neonates at the time of treating their cords with methylated spirit.

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Case Reports

Case 1

This 7-day old baby girl was born at home after a full-term uncomplicated pregnancy. She was not weighed at birth. She had a mild jaundice which cleared before the age of 5 days. On the day of the accident, the mother had given her the morning bath and was cleaning the umbilical cord with methylated spirit. Some of the spirit spilled on the bath towel and the baby's body. Both caught fire from a nearby naked flame and the bottle of spirit exploded in the mother's face.

The baby arrived in the Children's Emergency Room about 3 hours after the accident. On examination, she had largely first degree superficial burns of the head, face, abdomen, back, upper and lower limbs. The total area involved was estimated to be about 55%of the body

surface. She responded well to treatment with antibiotics, blood transfusion, antitetanus serum, open dressing with "Flamazine" cream (Smith & Nephew Pharmaceuticals Ltd.), analgesics and physiotherapy. At the time of discharge, 25 days after admission, she had contractures of the terminal interphalangeal joints of the left hand, and physiotherapy was continued as an outpatient.

Case 2

Baby S, a female, was born at full term weighing 3.6kg. She was delivered in a health centre and discharged home within 24 hours. The umbilical cord separated in the first week of life. The baby was in good health until the age of 14 days. While receiving her morning bath, there was public electric power failure, and a hurricane lantern had to be used to provide light. While the umbilical cord (which had in fact healed) was being treated with methylated spirit, the spirit caught fire and spilled on the baby. The baby was seen in the Children's Emergency Room within 2 hours of the accident. She had first degree burns of the lower limbs and genitalia, involving about 23% of the body surface. Treatment consisted of analgesics, antibiotics, antitetanus serum, intravenous fluids and "Flamazine" cream. The lesions healed well and she was discharged home after 22 days.

Case 3

Baby Y was a full-term male whose cord was being cleaned with methylated spirit in the presence of an open candle flame for light. The local public electric power supply had been interrupted and the mother was not aware of the inflammability of methylated spirit. The bottle of spirit was accidentally knocked over and spilt, then caught fire, spreading all over the baby, mother and elder sister nearby. He had full thickness burns covering 25% of the body surface. He was brought to the emergency room within 5 hours and admitted to the Ward two hours later. Management consisted of antitetanus serum, antibiotics, intravenous fluids and whole blood transfusion, analgesic as well as open dressing with "Flamazine" cream and incubator care. The wound got secondarily infected with *Pseudomonas pyocyanea*. He died after one week in the ward, presumably from overwhelming septicaemia.

Case 4

A full-term baby girl who had third degree burns of the perineum, the lower limbs and part of upper limbs, covering about 15% of the body surface. The circumstances of the burn were akin to that of Case 1. She was on admission for 10 weeks. At the time of discharge, she had fibrous contractures of the knee and ankle joints for which she continued to receive treatment in the plastic surgery and physiotherapy departments.

Discussion

Infection of the umbilical cord has been shown to be an important cause of neonatal sepsis. Jellard[3] found that the umbilicus was an important reservoir of staphylococcal infections in a maternity hospital. Similarly, Barrett, Mason and Fleming[4] had shown that the umbilicus is often the focus of infection with *E. coli* and other gram-negative bacilli in the newborn period. In addition to these potentially serious infections, a baby born in the developing countries is also at risk of developing neonatal tetanus through contamination of the umbilical cord. In an effort to reduce these infections to a minimum, routine care of the cord with alcohol or methylated spirit (either solely or in conjunction with other agents) has often been recommended. Unfortunately, alcohol is highly inflammable and our four cases have shown that the use of methylated spirit for the care of umbilical cord in the presence of a naked flame also constitutes a fire hazard.

According to the Committee on Fetus and Newborn of the American Academy of Pediatrics, there is no single method of cord care which has been proven to limit colonization and disease. In a controlled study, Neumann *et al*[6] found that

with cord care, alcohol did not alter the staphylococcal colonization rate of babies in their nursery. Recently, Barrett, Manson and Fleming[4] have recommended the use of silver sulfadiazine. These authors found it very effective against gram-negative bacilli and group B *Streptococci*, but less effective against *S. aureus* than the triple dye (Triple dye is composed of 2.29 gm of brilliant green, 1.14 gm of pro-flavine hemisulfate, 2.29gm of crystal violet and enough of water to make 1,000 ml). However, silver sulfadiazine or antibiotic aerosol sprays[7] are very costly and not readily available in many developing countries. It is therefore unrealistic to recommend these for general use. Routine application of 0.33% hexachlorophene dusting powder as recommended by Morley[8] can also no longer be supported in view of the known neurotoxic effects of that chemical.[5]

For routine care of the umbilical cord in rural areas, triple dye seems to be ideal. It is cheap, non-toxic, un-inflammable and can be readily prepared by any dispensary or pharmacy. Several investigators[9 10] have found it impressive in preventing *S. aureus* colonization of the newborn and in controlling streptococcal infection in a nursery. Its weak effect on gram negative enteric bacteria can probably be improved by multiple applications of the dye on the cord rather than the single application recommended by Barrett, Mason and Fleming.[4]

Triple dye can stain linen and clothings but this is not a serious problem as mothers and nurses can, with little care, learn to avoid this snag.

Our cases highlight the danger inherent in the continued use of alcohol or methylated spirit for cord care. Multiple applications of triple dye are suggested as a practical, cheap and safe alternative.

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